



proceedings

conference on women
and the workplace

SOEH

SOCIETY FOR OCCUPATIONAL AND ENVIRONMENTAL HEALTH

PROCEEDINGS
CONFERENCE ON WOMEN AND THE WORKPLACE

June 17-19, 1976

Washington, D.C.

Editor: Eula Bingham, Ph.D.

Sponsors

Society for Occupational and Environmental Health
National Institute for Occupational Safety and Health
The National Foundation—March of Dimes

Co-sponsors

Coalition of Labor Union Women
D. C. Lung Association
United Church Board for Homeland Ministries
Industrial Union Department—AFL-CIO

Conference Chairpersons

Dr. Eula Bingham
Ms. Clara Schiffer



Publisher:
Society for Occupational and Environmental Health
1714 Massachusetts Avenue, NW
Washington, D.C. 20036
(202) 785-8177

Copyright © 1977 by the
Society for Occupational and Environmental Health

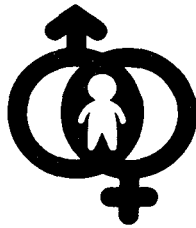
Printed in the United States of America
Library of Congress Catalog Number: 77-76490
April 1977

Conference and editing of the proceedings supported by Contract
No. 210-76-0154 with the National Institute for Occupational
Safety and Health, Department of Health, Education and Welfare.

Sponsorship of this Symposium and publication of these pro-
ceedings does not constitute NIOSH endorsement of the views
expressed or the recommendation of any commercial product,
commodity, or service mentioned.



C.3



**THE NATIONAL FOUNDATION
MARCH OF DIMES**

*Publication of the proceedings made possible by a grant from
The National Foundation—March of Dimes.*

Publisher's Note

In 1972 a series of informal discussions was held by concerned physicians, laboratory scientists, epidemiologists, hygienists, economists, planners, administrators, and political scientists representing a wide range of disciplines and interests. Government, academia, management, labor, and public interest groups—all were involved. These discussions led to the realization that a neutral forum was needed for the analysis of the scientific, social, and administrative implications of environmental health research and regulation, without the limitations of specialization, and unhampered by organizational constraints.

At the New York Academy of Sciences on November 12, 1972, 100 founding members addressed this need. The day's discussion resulted in the establishment of the Society as a neutral, not-for-profit, international organization for scientific and technical exchange among professionals and concerned lay people.

The organization that has emerged is fulfilling the hopes of its founders.

At its first annual meeting on December 4, 1973, now Vice President Walter Mondale had this to say:

"You represent—more than anyone else—the basic skills and experience necessary to the accomplishment of the national objective declared by the Occupational Safety and Health Act of 1970."

The Society for Occupational and Environmental Health is unique in a number of ways:

- It is problem-oriented, achieving its goals through a multi-disciplinary attack on key issues on neutral ground.
- Its broad-spectrum approach brings together professionals and lay people from organizations with often differing commitments, but with a mutual concern for the solution of common problems.
- The wide range of interests enables the Society to advance neglected areas of occupational and environmental health, including regulation, education, administration, and research.
- The diversity of financial support—members, industries, unions, government, and private organizations—minimizes the possibility of compromising neutrality.

By the summer of 1976, the Society had established an aggressive program of conferences in selected areas of critical concern, highlighted by the Conference on Women and

the Workplace in Washington, D.C., June 17-19, 1976. That conference has resulted in these proceedings.

These proceedings represent the first commercial monograph to be published by the Society. Six prior monographs were published privately or through the government. Under the leadership of the Society's Committee on Publications and Awards, chaired by Mr. Wayne Brooks and Mr. Frank Wallick, SOEH has experimented with a newspaper, HAZARD, and now through a generous grant from The National Foundation, an attempt is being made to utilize the marketplace for dissemination of informative books to serve the thinking public. Monies gained from the sale of this publication are committed to a publishing fund especially set up by the Society for this purpose.

A special note must be made of the kind patience of Mr. Gabriel Stickle of The National Foundation and of the technical guidance of Doctors Jack Butler and Kenneth Bridbord of the National Institute for Occupational Safety and Health. The immense problem of logistics and the details of technical editing were competently dealt with by Ms. Sandy Zimmerman of our staff and Mr. Paul Brodeur, our editorial consultant.

Sheldon W. Samuels
Secretary-Treasurer

Washington, D.C.
April 1977

CONTENTS

OPENING REMARKS	1
Dr. Joseph K. Wagoner President-Elect Society for Occupational and Environmental Health	
CONFERENCE PREVIEW	2
Dr. Eula Bingham Associate Director Department of Environmental Health University of Cincinnati School of Medicine	
SESSION I - RISKS OF TOXIC SUBSTANCES TO FUTURE GENERATIONS	
<u>Moderator:</u> Dr. Eula Bingham	
LABORATORY APPROACHES	
The Chemical Environment and Mutagenesis	5
Dr. Marvin S. Legator Division of Environmental Toxicology and Epidemiology University of Texas Medical Branch	
Toxic Substances and Congenital Malformations	28
Dr. Josef Warkany Children's Hospital Research Foundation University of Cincinnati, College of Medicine	
Male as Well As Female Mice are Affected by <i>In Utero</i> Exposure to Diethylstilbestrol	32
Dr. John A. McLachlan Environmental Toxicology Branch National Institute of Environmental Health Sciences	
OBSERVATIONS AMONG HUMAN POPULATIONS	
Transplacental Effects of Diethylstilbestrol on the Human Male Fetus: Abnormal Semen and Anatomical Lesions of the Male Genital Tract	39
Dr. William Gill Department of Surgery and Urology University of Chicago	

Transplacental Carcinogenesis: Prenatal Diethylstilbestrol (DES) Exposure, Clear Cell Carcinoma and Related Anomalies of the Genital Tract in Young Females 47
Dr. William R. Welch
Department of Pathology
Harvard Medical School

Blood Dyscrasias and Childhood Tumors and Exposure to Chlorinated Hydrocarbon Pesticides 51
Dr. Peter F. Infante
National Institute for Occupational Safety and Health

Congenital Minamata Disease: Methyl-Mercury Poisoning and Birth Defects in Japan 75
Aileen M. Smith
New York City

DISCUSSION

Dr. Charles Shaw 84
University of Texas Medical Department
M.D. Anderson Hospital and Tumor Institute

Dr. Umberto Saffiotti 85
Experimental Pathology Branch
National Cancer Institute

Dr. Eldon Sutton 87
Vice President for Research
University of Texas at Austin

QUESTIONS, ANSWERS, COMMENTS - SESSION I 89

SESSION II - BIRTH DEFECTS, CANCER, AND MISCARRIAGE ASSOCIATED WITH ANESTHETIC GASES, VINYL CHLORIDE, AND OTHER INDUSTRIAL CHEMICALS

Moderator: Dr. John Finklea 92
Director
National Institute for Occupational Safety and Health

BIOLOGICAL MANIFESTATIONS

Cancer, Miscarriages, and Birth Defects Associated With Operating Room Exposure 94
Dr. Thomas H. Corbett
Research Service
U.S. Veterans Administration Hospital
Ann Arbor, Michigan

Genetic Effects Associated with Industrial Chemicals	100
Dr. Joseph K. Wagoner	
Division of Surveillance, Hazard Evaluation and	
Field Studies	
National Institute for Occupational Safety and Health	

Birth Defects and Vinyl Chloride	114
Larry Edmonds	
Birth Defects Branch	
Center for Disease Control	

PLACEMENT OF WORKERS IN HIGH-RISK AREAS

An Industry Viewpoint	120
Dr. Harold L. Gordon	
Medical Director	
Dow Chemical Corporation	

A Workers' Viewpoint	124
Sylvia Krekel	
Occupational Health and Safety Specialist	
Oil, Chemical & Atomic Workers Union	

A Legislative Viewpoint	127
Peggy Taylor	
Professional Staff	
U.S. Senate Subcommittee on Labor and Welfare	

DISCUSSION

Dr. Samuel Milham, Jr.	133
Non-Communicable Diseases Unit	
Washington State Department of Social and Health Services	

Dr. Bertram Carnow	134
Occupational and Environmental Medicine	
University of Illinois School of Public Medicine	

QUESTIONS, ANSWERS, COMMENTS - SESSION II	136
---	-----

SESSION III - OPEN FORUM FOR WORKING MEN AND WOMEN - WHAT ARE YOUR CONCERNS:

<u>Moderators:</u> Dr. Gloria C. Gordon	149
Department of Psychology	
St. Louis University	

Andrea Hricko	150
Labor Occupational Health Program	
University of California at Berkeley	

SESSION IV - HEALTH RISKS ASSOCIATED WITH JOB PLACEMENT PATTERNS
RELATED TO SEX

<u>Moderator:</u> Dr. David Wegman Harvard School of Public Health	167
Women as a High-Risk Population Dr. Shirley Conibear University of Illinois School of Public Health	
Hazards Related to Personal Physical Strengths Dr. Don Chaffin Occupational Health and Safety Engineering Program University of Michigan	174
Health Problems in the Airline Industries Sunny K. Wofford Health and Safety Project Coordinator Association of Flight Attendants	179
Respiratory Disease Prevalence in Beauticians and Its Relationship to Aerosol Sprays Dr. Alan Palmer Support Services Branch National Institute for Occupational Safety and Health	184
Women in the Textile Industry Dr. Kaye Kilburn Department of Medicine University of Missouri Medical Center	189
Radiation Exposure and Protection Vilma Hunt Associate Professor of Environmental Health Pennsylvania State University	196
Mercury: A Health Hazard Associated with Employment of Women as Dental Assistants Meier Schneider Medical Services Division City of Los Angeles Personnel Department	202

DISCUSSION

Dr. Marcus Bond Corporate Medical Director American Telephone & Telegraph Company	214
Dr. Janette Sherman Detroit, Michigan	216

SESSION V - LEAD AND WOMEN, A UNIQUE PROBLEM?

Moderator: Dr. Warren Muir
Council on Environmental Quality

BIOLOGICAL MANIFESTATIONS

Review of Lead Toxicity 227
Dr. Kenneth Bridbord
National Institute for Occupational Safety and Health

The Effect of Lead on Reproduction 232
Dr. Peter F. Infante
National Institute for Occupational Safety and Health

JOB PLACEMENT OF WOMEN IN THE LEAD TRADES

An Industry Position 243
Dr. Sidney Lerner
University of Cincinnati School of Medicine

A Workers' Position 251
Claudia Prieve
Industrial Hygienist
United Steelworkers of America

The Department of Labor's Position 259
Dr. Morton Corn
Assistant Secretary for Occupational Safety and Health
Department of Labor

DISCUSSION

Sue Nelson 263
Legislative Associate
House Subcommittee on Manpower, Compensation, and
Health and Safety

Wayne Brooks 265
Director, Occupational Safety and Health
Organization Resources Counselors, Inc.

QUESTIONS, ANSWERS, COMMENTS - SESSION V

SESSION VI - A SAFE WORKPLACE: CURRENT PERSPECTIVES AND
FUTURE NEEDS

Moderator: Jane A. Lee 292
National Institute for Occupational
Safety and Health

Workers' Compensation: The Long-Term Consequences of 292
Work-Related Injury for Women
Dr. Julia Loughlin Makarushka
Health Studies Program
Syracuse University

Guidelines for Evaluating the "Disability" of Pregnancy 302
Dr. Leon J. Warshaw
Vice President and Corporate Medical Director
Equitable Life Assurance Society of the United States

PANEL DISCUSSION OF SOCIETAL RESPONSIBILITIES AS SEEN BY WOMEN

Andrea Hricko 310
Labor Occupational Health Program
University of California at Berkeley

Jeanne Culler 313
Staff Assistant for Occupational Safety and Health
Amalgamated Clothing Workers

Carolyn Bell 316
Industrial Hygienist
United Rubber Workers

QUESTIONS, ANSWERS, COMMENTS 321

PANEL DISCUSSION OF LEGAL REQUIREMENTS AND IMPLICATIONS

Peter Robertson 327
Director, Office of Federal Liaison
Equal Employment Opportunity Commission

Portia Y.T. Hamlar 330
Attorney, Legal Department
Chrysler Corporation

Anne Trebilcock 335
Assistant General Counsel
United Auto Workers International Union

QUESTIONS, ANSWERS, COMMENTS - SESSION VI 341

PRESENTATION OF THE SECOND AWARD OF THE SOCIETY FOR OCCUPATIONAL AND ENVIRONMENTAL HEALTH TO DR. HARRIET L. HARDY	351
ACCEPTANCE BY DR. HARDY	353
A STATEMENT BY THE COALITION OF LABOR UNION WOMEN'S TASK FORCE ON OCCUPATIONAL HEALTH AND SAFETY	358
A STATEMENT BY THE WOMEN'S POLITICAL ACTION - OCCUPATIONAL HEALTH CAUCUS	359
A STATEMENT BY THE LABOR SAFETY AND HEALTH INSTITUTE	361

OPENING REMARKS

Dr. Joseph K. Wagoner, President-elect
Society for Occupational and Environmental Health

It is my pleasure and privilege as President-elect of the Society for Occupational and Environmental Health to greet you and to open the Conference on Women and the Workplace. I should also like to extend the greetings of the other sponsoring agencies of this conference, the National Institute for Occupational Safety and Health and the National Foundation March of Dimes, as well as those of the co-sponsors, the Industrial Union Department of the AFL-CIO, the Coalition of Labor Union Women, the D.C. Lung Association, and the United Church Board for Homeland Ministries.

I would like to congratulate the two conference Chairpersons, Dr. Eula Bingham and Clara Schiffer, for organizing this conference around a social issue, which is clearly of great and increasing importance, not only to women and men of this current generation, but also to the well-being of future generations.

This year we mark the 200th anniversary of the birth of this nation, and I think today is really an occasion for sober reflection, for on this bicentennial we are holding the first conference in the history of this country on women and the workplace. It was back in 1775 that Percival Potts identified the first cancer of occupational origin -- scrotal cancer in chimney sweeps exposed to coal soot. Yet, in spite of the fact that women represent a sizable portion of our labor force, it was not until 1946 that vulval cancer was identified in cotton spinners exposed to oil.

I think there is an increasing concern with regard to two major rights. The first is the right to work regardless of sex. The second is the right to life whether one is male, female or of a generation yet unborn. And now I would like to turn the conference over to Dr. Bingham, who has done a tremendous job in organizing it.

CONFERENCE PREVIEW

Dr. Eula Bingham
Associate Director
Department of Environmental Health
University of Cincinnati
School of Medicine

Good morning. The necessity for a conference such as this arises as Dr. Wagoner indicated because of health issues and social issues. Currently we believe that the health issues center around two areas: First of all, the occupations where women traditionally have been employed in large numbers; secondly, the occupations that are associated with a potential for genetic damage or a potential for transfer of chemicals across the placenta to the fetus. This may result in cancer for the offspring, or in a birth defect, or it may produce some other toxicological effect.

We shall have speakers who are currently involved in investigations of the effects of environmental or occupational contaminants, and who have paid particular attention to the female. We have, for example, Dr. Josef Warkany, an investigator who has actually been a pioneer in trying to determine the cause of birth defects. We have Dr. Marvin Legator who will provide comments about genetic risks in women and, I think, in men also. We have speakers who have paid a great deal of attention to women in particular occupations, such as Dr. Kaye Kilburn, who has studied women in the textile industry.

Now, these are the major health areas we shall cover here in the next two and a half days, but I would like to raise with you certain issues and questions at the very beginning so you may better understand our planning of the program.

For example, does the National Institute for Occupational Safety and Health or other NIH institutes automatically include females and males in their investigation of occupational health problems?

Secondly, have female workers ever been considered when a health and safety standard has been promulgated, or is the prototype of an American worker always the white American male?

If female workers are to be considered in the development of standards, and it is found that in a particular operation or occupation they represent a susceptible segment of the population, what are the solutions to be proposed? Should the solution be not to hire them, to fire them, to ignore them, or should it be to set an occupational standard that will protect them?

When males are the most susceptible in a particular operation, what has been the solution? Don't hire them, fire them, ignore them -- and I will admit sometimes they are ignored -- or set a standard to protect them.

There is little doubt that part of the need for a conference such as this arises because there has been a failure in the past to recognize women as part of the work force. Would you believe that I have had women come up to me when we were organizing this conference, and say, "I am really afraid to have a conference on women and the workplace because if health risks are discussed, then women will be excluded from certain occupations. Or they have reminded me that it has only recently been mandated that women shall be offered equal opportunity, and that there has been slow and gradual improvement in the occupational opportunities for women, and that a conference such as this may result in repressive hiring practices.

And my answer is, it is here. We already have repressive hiring practices and we have to face up to what is going on. Let's talk about it in public. Some of us think that one of the roles for the Society for Occupational and Environmental Health is to provide a public forum for discussing occupational, environmental and social issues. So that is why we are having a conference at this time.

Now, I would like to come face to face with the fact that this is a very emotional issue. I would expect tempers and emotions to flare in the next few days. In fact, I will be surprised if they don't.

We have in our audience women, or representatives of women, who are fearful of job transfer to a lesser paying job because they are female and carry offspring. We have women who are angry and who demand their right to seek a job in a lead battery plant.

We have women who need to work and who are concerned for their unborn children. We also have in our audience industrial representatives, who are paralyzed with fear over possible liability and lawsuits, and some who counsel their companies not to hire women in certain areas.

I see representatives from governmental agencies, who know that they have to deal with one of the most difficult health issues that has emerged.

Dr. Finklea recently pointed out to us in a seminar at the University of Cincinnati the variability of governmental responses and policy positions on how to deal with the health issues of women in the occupational environment.

While it is not new for the U. S. Government to be in conflict

with itself on policy matters, I believe we have seen expressed by NIOSH and OSHA a desire for the scientific and medical community to help decide a unified policy in this area.

We have representatives in the audience from the Equal Employment Opportunity Commission, and I hope these individuals are going to keep reminding us as we discuss our approaches to health problems, what the law actually is.

We may even have men in the audience who, when reminded of the fact that genetic damage can occur in males, as well as in females, will help us come up with solutions to so-called women's problems. I am not sure that we are going to arrive at solutions here this week, but I hope we can have a dialogue that will help us understand the points of view and the positions of industry and labor, and the dilemma of government in coming up with standards to solve this health problem.

We will now begin our conference by discussing laboratory approaches and actually what is known about the genetic risks, malformations, and cancer that may be caused by certain substances. There may be some of you in the audience who will not understand all the scientific jargon that some of our speakers will use. If you do not understand, come up to the speakers after the presentations, or later on, and talk with them. I am sure that I can speak for our speakers this morning in saying that they are willing to discuss these issues with you, and to explain the meaning of some of the terms that they will use.

Our first speaker is Dr. Marvin Legator, who will talk with us about toxic substances and genetic risks.

SESSION I

RISKS OF TOXIC SUBSTANCES TO FUTURE GENERATIONS

MODERATOR: Dr. Eula Bingham

THE CHEMICAL ENVIRONMENT AND MUTAGENESIS

Marvin S. Legator, Professor
and

Stephen J. Rinkus, Research Associate
Division of Environmental Toxicology and Epidemiology
University of Texas Medical Branch
Galveston, Texas

SUMMARY

In the past fifty years, man has developed an unprecedented reliance on synthetic chemicals, the overwhelming majority of which are uncharacterized with respect to carcinogenicity and mutagenicity. Prominent categories of these substances include prescription and non-prescription drugs, pesticides, food additives, and industrial chemicals. In retrospect, some of the substances have caused chronic adverse effects such as cancer and genetic diseases. The Toxic Substance Controls Act marks the first step in resolving the problem by requiring testing of substances of questionable benefit-versus-risk to health and the environment. It must be clear from the outset that adoption of preliminary screens based on microbial studies, the so-called tier approach, is toxicologically naive and indefensible. The logical approach employs combined testing in intact mammals and should consist of a battery of tests whose precisions are such that a doubling of the control level of mutation is statistically significant at the 5% level. A positive response in any of the animal systems characterizes the substance as a mutagen, although a substance should not be considered non-mutagenic if the B-error's magnitude precludes detection of mutagenic activity. Priority of testing should reflect concern for the population in the childbearing age, the length of exposure, the persistence of the substance in the environment, and a structure-activity relationship. The unique possibilities of industrial monitoring and a benefit-risk analysis are also discussed.

INTRODUCTION*

In this century, man has sought to use chemicals to raise the quality of life. Among these chemicals are those that by their usage alone touch many aspects of life in this country: drugs, pesticides, food additives, industrial chemicals, etc. With hindsight, it is now not too surprising that some of these substances have had unintentional results. Some widely used chemicals have been shown to induce adverse, chronic effects such as carcinogenic and mutagenic responses. Let us place the situation into some sort of technico-social perspective.

The use of medicinals in this country has been steadily increasing since the 1930's. Figure I shows the U.S. quantities of sales of medicinals co-graphed with the population growth for the years 1930 through 1974. Extrapolation of both curves on the graph suggests that by 1983 the average citizen will take one pound of medicinals a year; as of 1970, for which the latest comparison can be made, we consumed .76 pounds a year. The market itself consists of about 290 prescription formulations (2) and an estimated 100,000 to 500,000 non-prescription drug products (3). The value of sales of bulk medicinal chemicals, excluding finished preparations and dosage-form products, totaled \$814.8 million in 1974 (4). The total number of prescriptions filled in retail pharmacies in 1975 amounted to nearly 1.5 billion, valued at \$5.20 per prescription (2), or a total value of sales of \$7.7 billion for the year. Furthermore, the present trend is to use drugs over an increasing period of time for chronic diseases, as opposed to short-term therapy for acute conditions (see Table I: 1970). Figure I's insert indicates a logarithmic increase in the quantity-of-sales per person index. Clearly, we are a drug culture -- a growing drug culture.

A similar situation of increased use of chemicals is seen with pesticides. Figure II and insert are indicative of the growth in the use of pesticides in the United States; the quantity of sales of pesticidal active ingredients has skyrocketed from 287,150 pounds in 1940 (6) to 1.365 billion pounds in 1974 -- a resounding 4754-fold increase in the span of only thirty-odd years. Fungicides, herbicides, and insecticides constitute 11%, 43%, and 46% of the production volume, respectively, in 1974 (7). Fifteen

**In the following section, the discussion makes references to U.S. Tariff Commission data on the sales of synthetic, organic chemicals. Their figures do not reflect exports and imports, consequently, they are not strict, usage figures for this country; in fact, no such detailed accounting exists. However, since these statistics are cited repeatedly in various government reports (1,6,7), they are considered reliable indications of the disappearance of the chemicals at the domestic level.*

hundred chemical pesticides have been developed, 275 of which have achieved any significant commercial importance; but these significant chemicals may appear in as many as 8,000 individually formulated pesticide products (6). The value at the manufacturer's level of sales of pesticidal active ingredients totaled \$1.8 billion in 1974 (4). Of the billion pounds of pesticides now sold annually, half are used in agriculture: 50% of all insecticides are applied to non-foods, tobacco and cotton; while corn, fruit, and vegetables receive the greatest amounts applied to food crops; 41% of the herbicides are used on corn, the rest being applied to various other crops; fruits, vegetables, and field crops are the main targets of the fungicides (8). Sixty-five percent of those pesticides used for agriculture and forest lands are sprayed by aircraft (7). Another half billion pounds of pesticides are used by government agencies, industries, and homeowners; EPA estimates that homeowners deposit on an average between 3,392 and 6,784 pounds of pesticidal active ingredients per square mile per year, probably making their lawns and gardens the heaviest pesticide-treated land areas in the country (9).

In this same vein of mass exposure to chemicals is the ever growing reliance on food additives to enhance the desirability and longevity of foods. Some 1,830 direct additives (not including such widely used ingredients as sugar, salt, starch, etc.) can appear in food products, and the yearly per capita consumption of these additives is about ten pounds (10). The value at the manufacturer's level of food additives has been estimated to exceed one half billion dollars (11). While half of these additives are consumed at less than one half milligram per person per year (10), among those consumed in greater amounts are some synthetic flavors and food dyes. Figure III illustrates the quantity of sales of Food, Drug, and Cosmestic Colors during the past three decades. The value of sales of F. D. & C. Colors was about \$33 million in 1974 (4). In the past decade, there has been a stronger increase in the annual per capita amount of F. D. & C. Colors, as illustrated in the insert to Figure III.

With the intention of upgrading the standard of living, science and industry are developing and introducing into the market as many as 1000 new compounds a year in commercial quantities (12). That these chemicals have proved effective in combatting sickness and increasing the food supply is without question. Table I shows a formidable shift in the leading causes of death in this country this century. The disappearance of bacterial and parasitic diseases as the great killers they once were in 1900 is directly attributable to modern medicine which readily employs drugs. Equally indicative would be the decline of infant mortalities from 51.9 deaths per 1,000 to 16.3 deaths per 1,000 between the years 1935 through 1972 (13); or the twenty-two year rise in the average American life expectancy since the turn of the century (14). Comparable benefits have been reaped in the rate of production of staple foods. Figure IV demonstrates that the dramatic increase

in crop production needed by our growing population corresponds in time to the onset of wide-scale employment of pesticides in the 1940's. As a particular example, corn production in this country was tripled from 1,418 kg/ha in 1933 to 4,243 kg/ha in 1963 (15).

However, the fact that these chemicals have exerted other effects on the population is also without doubt. It has become increasingly clear that as the incidence and severity of infectious diseases has declined, impairments in man owing to gene and chromosomal changes are having an increasingly serious impact on the health and economy of the human community. The finding that some man-made chemicals presently in wide-spread use in the population are mutagenic in sub-human experimental systems suggests that such chemicals constitute a potential genetic hazard for man. The risk poses itself to both present and future generations: to the former, as the production of genetic lesions in somatic cells, possibly leading to cancer; while to the latter, as the production of transmissible gene mutations and alterations in chromosome structure and number. Consider again Table I. In 1970, 17.2% of all deaths were attributable to cancer, as opposed to 15.6% in 1960, and only 3.7% in 1900. The situation is not simply that Americans are living longer to develop a natural, life-terminating disease, as illustrated in Figure V. In its most recent report to the President, the Council on Environmental Quality advised that 60% to 90% of all cancers are related to various environmental factors, including exposure to some chemicals (17). In socio-economic terms, the estimated 1.3 million cancer patients require \$1.8 billion per year in hospital care alone, or tens of billions of dollars if all direct expenditures incurred are considered (18); as much as 1.7 million work years are lost to the national economy and to family income as a result of cancer (19).

Unfortunately, comprehensive data on the incidence, variety, distribution, and etiology of actual gene mutations and chromosomal aberrations has not been compiled. Estimates on the occurrence of true genetic disease in the population would indicate that 12 million people carry defective genes or chromosomes that manifested themselves with birth defects (20). Based on chromosome examinations of 31,801 newborn children in seven major cities in four industrialized nations, indications are that from one in 117 to one in 285 newborns exhibit major cytogenetic abnormalities (21). Concerning the prenatal period, the occurrence of chromosomal aberrations in spontaneous abortions has been reported at one in 33 aborted fetuses for the last five months of gestation, one in four for the fourth month, and one in three for the first trimester (22). The overall incidence on conceptuses that are spontaneously lost could be as high as 45% (23). As was expressed at a recent seminar on genetic toxicology, it is hard to believe that the human race has endured successfully such rates over its 800 generations (24). It must be kept in mind that discussion only of

those genetic lesions whose phenotypic expression allows detection during gestation and birth would be a gross understatement of the magnitude of mutations and genetic disease. It would not account for the other dominant, recessive, and sex-linked disorders that manifest themselves later in the lifetime of the individual; likewise, it would overlook the multifactorial, genetic interplay in some forms of heart disease, arthritis, diabetes, cancer, hypertension and schizophrenia. Furthermore, Drosophila studies would suggest that mere heterozygosity for a lethal, or mildly deleterious, recessive mutation involves an incurred disadvantage on viability, despite the phenotypic dominance of the other, unmutated gene (25).

Again, the socio-economic impact of genetic disease is massive. Estimates have placed the monetary burden of genetic disease at 25% of the national expenditure for health services (26). The loss in life-years as a result of birth defects, 80% of which are thought to be genetic in origin (27), dwarfs the other major diseases (see Figure VI).

This is not to say that all, nor necessarily the majority of, chemicals cause cancer or induce mutations. The fact is that not enough information on the entire health consequences of the chemicals is known. Some are definitely detrimental, and the overwhelming majority have been released onto the market with inadequate testing for mutagenesis. For example, our survey of the top 200 prescription drugs for the U.S. in 1974 (69% of the market) reveals that, of the 190 separate entities comprising these top 200 drug products, only 53% (101 chemicals) have at least one mutagenicity-related study reported in the literature; the vast majority of the testing that was performed was in systems of little or no toxicological relevance (28).

The scope of the problem goes beyond intentional, mass exposure to uncharacterized chemicals. EPA has identified 253 possible organic chemicals in drinking water sampled across the country, including halogenated hydrocarbons such as vinyl chloride, carbon tetrachloride, and chloroform; and pesticides such as DDT, aldrin, dieldrin, heptachlor, and chlordane. All of these compounds are either confirmed or suspected carcinogens (29-35). Industrial and municipal discharges, urban and rural runoff, natural sources, and water and sewage treatment practices are among the confirmed sources of these pollutants. Again, the potential carcinogenicity of many of the other chemicals in the water supply is simply not known. However, the fact that these chemicals do appear in the blood (36) and tissues (37), and are implicated in increased cancer rates has been reported (38).

The set of circumstances that led to this chemical crisis is not as elusive as its resolution. In a 20th century continuation of the Industrial Revolution, we embarked on an era of unprecedented use of synthetic chemicals. It is an age much in step with

the Nuclear and Space Ages, and it is part of the same technico-social phenomena. Mass exposure to these substances began in a time that lacked an appreciation of carcinogenesis and mutagenesis. Since then, chemicals have become as enwebbed in and essential to our life style as computers, and their production has grown into a multi-million dollar affair -- all this in less than the span of one human lifetime. On the basis of the great number of chemicals, their ubiquitousness in our culture, what is already known about the adverse effects of some of the chemicals, and what remains undetermined about the others, it would appear that the miracles of the chemical age have an unforeseen price, the extent of which we are only beginning to experience. The latency period following the induction of a cancer has been estimated at 15 to 40 years (39), and without national surveys on the incidence of pregnancies spontaneously aborted due to chromosome anomalies and the incidence of genetic diseases in general, we can only speculate on the detrimental effects on the genetic pool from injurious chemical exposure. In terms of ourselves as a population of living organisms, we are suffering chemical shock.

CHEMICAL MUTAGENESIS TESTING

A. *Combined Testing*

With such views in mind, the 94th Congress has sent to the President the Toxic Substance Control Act. Representative Eckhardt, who was an architect of the bill in Congress, has warned that the law's passage will mark the theoretical advent of an improved national policy on chemical use, but that its actual ability to prevent the further release onto the market of chemicals that unjustifiably jeopardize our health and environment depends critically on the commitment of the scientific community (40). In brief, the law would require that a chemical suspected by EPA to pose a probable and "unreasonable risk of injury to health or the environment" (41) be submitted to tests that develop appropriate data. Hence, the merit of the law to stem this chemical shock hinges on what tests are to be considered appropriate.

The growing awareness that it is no longer possible to introduce new chemicals into the environment without toxicology testing in the area of mutagenesis makes it imperative that we examine the relevance of these existing procedures as compared to those techniques that are commonly used in other areas of toxicology. Mutagenicity studies should not be considered simply as a means to identify a potential carcinogen, but also as a means of preventing genetic abnormalities whose importance to man may well eclipse all other areas of combined testing. The so-called tier approach (42), or preliminary screens, based on microbial studies are inadequate and toxicologically naive for evaluating potential mutagens. The use of in vitro microsomal preparations combined with microorganisms or other indicators as an exclusive primary screen is an approach whose deficiencies must be recognized. An in vitro

microsomal activating systems cannot reflect the complex dynamic processes that are carried out in the intact animal. Indeed, it is impossible to devise a standard in vitro activation system that can be used generally to screen potential mutagens and carcinogens. Even if such an in vitro system could activate all compounds that are metabolized by microsomal enzyme systems, the fact that many materials are either potentiated or detoxified by other routes, e.g. intestinal flora, would argue against the use of the system as an initial screen test. Thirdly, an important class of chemicals that induce nondisjunction by affecting spindle mechanism, one of the most important categories of cytogenetic abnormalities, would be missed by bacterial studies.

In every category of chemicals in our environment, there are examples of carcinogens and mutagens that would not be detected by such microbial procedures, with and without activation. Urethane, the insecticidal group represented by Dieldrin, Cycasin (the naturally occurring toxins), and halogenated purines are a few of the many examples of compounds that would not be detected by this simplistic approach (43). In the literature, one can find data suggesting that the correlation between microsomal activation procedures and known mutagens and carcinogens is between 70 and 90 percent (44). These correlations, which in themselves are not adequate to justify in vitro procedures as a primary screen for detecting potential carcinogens and mutagens, are questionable. For instance, quantitative data and levels of significance have not been stated; and in most cases, selected compounds, rather than randomly chosen materials, were evaluated. In government-sponsored research projects, a failure to code the materials, which has allowed investigators to know beforehand if the compound was a suspected or confirmed carcinogen or mutagen, has led to an overestimate of this correlation (43). In fact, a recently completed study indicates that only 65% of known carcinogens are active in Salmonella testing strains, with or without activation (45).

All responsible scientists share the goal of identifying mutagens and eliminating them from our environment. It is essential, however, that we do not adopt indefensible toxicological procedures in attempts to achieve our goal. The crux of the matter is not the identification of active compounds by microbial procedures, with or without activation, but the fact that we may miss potent carcinogens and mutations by screens that cannot be considered as preliminary screens for detecting active materials. Industrial toxicologists and other interested individuals should not be lulled into a false sense of security and assume that the chemicals are not mutagenic when the compound is found not to be active by existing microbial procedures (46). In an in-depth study of chemical mutagens, one would first select those test systems that have the capability of indicating the various types of DNA alternations produced by chemicals that are active per se, or those which are activated by enzymes of the tissues or intesti-

nal flora of the host. In principle, with an unknown chemical, one would start with the best available animal systems, including those tests that evaluate metabolic products of the intact host. Since there is no single test for detecting chemical mutagens, a complete testing protocol would utilize a battery of tests carried out in the intact animal. The integration of the results from these systems should offer the optimum opportunity of identifying mutagens that are potential hazards to man. The subsequent studies of an active compound would rely on refining procedures to isolate and identify the active compound and, subsequently, to characterize the genetic lesions induced by the chemicals under study. This approach is contrary to tier approach which uses in vitro systems and then advances to animal tests; such an approach should be reserved. In the field of chronic toxicology, available methods are used to evaluate new compounds before and after they are introduced into the market place, as well as to evaluate currently used materials. Although, in many instances, these procedures are time-consuming and expensive, no one would suggest that they be abandoned until more suitable methods are developed. In like manner, to postpone the evaluation of chemicals for mutagenic activity, or to settle for short-term procedures that may indeed yield misleading information, is to diminish the importance of this area of toxicology. Furthermore, it represents a failure to recognize the fact that currently available procedures for mutagenic evaluation are less time-consuming, less expensive, and probably more meaningful than many tests that are available and presently used in the field of toxicology. Indeed, if one employs a battery of tests that would detect compounds which cause point mutations and chromosomal aberrations, including nondisjunction, the total cost would be only a fraction (approximately one-third) of the \$100,000 that is presently allotted for a single carcinogenic evaluation.

B. Testing Procedures (47)

Procedures for detecting and characterizing various types of genetic lesions include the following:

- (1) Detection of "premutational lesions": repair studies in experimental animals.
- (2) Detection of "point mutations": Host mediated assay, and body fluid analysis in experimental animals, using various indicator organisms as well as in vitro studies, with and without microsomal activation.
- (3) Detection of chromosomal change in experimental animals:
 - a. Dominant lethal test.
 - b. Translocation studies.
 - c. Micronuclei tests.
 - d. Direct cytogenetic analysis with both meiotic and mitotic cells.
 - e. Sister chromatid exchange studies.

While collaborative studies have rarely been conducted in the field

of toxicology, it is noteworthy that already in the field of genetic toxicology the dominant lethal test and in vivo cytogenetic analysis have been subjected to collaborative studies (48). The utilization of all or most of the above procedures should characterize the majority of mutagenic agents. Additionally, these studies combined with in vitro procedures will in many instances classify the induced genetic lesion. Existing procedures are as good as, if not better than, existing methods in the field of chronic toxicology; furthermore, we have the ability to gain insight into the molecular basis for the genetic alteration.

C. Industrial Monitoring

The industrial environment provides a unique set of circumstances for detecting and characterizing chemicals that induce chronic effects such as the induction of mutations. In many instances, we can identify workers who are exposed to a variety of chemicals. Exposure to some of these chemicals may be limited to workers in various industries, while exposure to a variety though heaviest among workers, may also be occurring in the general population. It is encouraging to note that some of our more progressive corporations have embarked on a comprehensive program to detect chemical mutagens (48).

In the context of an industrial program to characterize mutagenic agents, there are three aspects that one can consider. First, we can deal with experimental compounds that have yet to be introduced into the market place, or those agents that have not been adequately tested for mutagenic activity. These chemicals should be thoroughly investigated by a variety of available mammalian procedures, as previously discussed. Another aspect would be the evaluation of chemicals to which workers are exposed. Again, one can rely on cytogenetic analysis and the investigation of the blood, urine, and semen (when available) of these workers to look for potential mutagens; evaluation of these body fluids can employ the indicator organisms. A third aspect would involve classical epidemiological studies. Unfortunately, the epidemiological studies come rather late in the game, for if they are positive, significant adverse effects in the human population have already occurred.

D. Interpretation of Results

Toxicology as well as pharmacology is often criticized for not being a more quantitative science. (Facetiously, it has been said that conventional procedures for carrying out chronic toxicity studies include counting the dead, weighing everything that can be removed, slicing everything that can be sliced and feeding everything that could be fed for two years.) Actually, the importance of the quantitative data and the various factors that can influence a biological response in animals has long been appreciated by toxicologists and pharmacologists. The route of chemical administration, the chemical's distribution in the tissues, the nutritional state of the

animal, and, especially, the biotransformations of chemicals entering the circulation are some of the more obvious factors contributing to the variation of biological results in the same, as well as in different, animal species. A given chemical at different rates may be esterified in the rat, conjugated in the dog, and acetylated in man. All of these factors will influence assays for mutagenicity as well as measurements for any other toxicological effect.

The importance of a dose-response curve, the concept of a threshold effect, and the difficulties of extrapolating from animal data to man, especially in the area of carcinogenicity, have been the subject of many reports (49). As far as can be determined, the carcinogenic hazard never disappears with a diminishing dose but rather becomes infinitely small. A further complication of establishing a threshold concentration in carcinogenesis is the possibility of co-carcinogens (50) being present in the environment, further obscuring even the possibility of obtaining a no-effect level. The concept of a threshold response in mutagenesis is hardly different from that in carcinogenesis. In the case of X-rays, a direct linear relationship between dosage and genetic effects in various biological systems gave rise initially to the "single hit" interpretation of X-ray effects (single hit and ionization of DNA). This single hit causes a permanent alteration of DNA (mutation), and the effects are usually additive when doses are applied intermittently. A number of substances (e.g. 1,2-dithioglycerol) are known to modify the lethal and mutagenic effects of X-rays. The existence of compounds that can modify the mutagenic effects of X-rays has cast some doubt on the target (single hit) theory and has led to the assumption that the genetic effects of radiation may be a more indirect result. As in carcinogenesis, a direct effect or an effect modified by environmental agents in mutagenesis is possible.

In mutagenic studies, a dose-response curve for interpreting animal results should be a prerequisite. Mathematical models that take into account the size of the animal population studies, as well as the dose-response in extrapolating to a large population exposure, are presently under investigation (51). The precision of the test used for mutagenicity should be such that a doubling of the control level of mutation would be statistically significant at the 5 percent level. A substance should be considered as a potential mutagen if one or more of the procedures carried out in the intact animal are positive. In a given experiment, failure to establish an existing effect with a chemical is generally referred to as a type II or beta (B) error. It is essential that in all mutagenic studies sufficient animals are used, enough slides are read, and sufficient implants are analyzed, etc., to minimize the B error. A casual examination of the literature in this field frequently reveals that the studies conducted would preclude assuming a negative response.

PRIORITY FOR TESTING

The assigning of priorities to environmental agents and their subsequent in-depth screening and characterization offers the possibility of eliminating the most important deleterious environmental agents. At first glance, the task of screening environmental agents for mutagenicity seems overwhelming. No data are available for most of the thousands of compounds introduced into our environment over the last three decades. The appreciation of this formidable task led to a search for a simple, economic screen to detect mutagens. As stated before, it is not logical to utilize simple systems, such as microbial procedures, in such a screening for potentially active compounds; in fact, the most meaningful screen would rely on a battery of tests carried out in intact animals. In view of these considerations, we must establish priorities for testing environmental agents and then proceed to screen the selected compounds in a meaningful manner. A comprehensive screen using a combination of available methods in animals, including testing of metabolites produced in the intact host, is still comparatively rapid and economical when compared to the conventional carcinogenicity screens.

A selection of compounds for testing should be based on the following criteria: (1) usage pattern, i.e. the exposure of a large segment of our population, especially people of child-bearing age, (2) length of exposure, (3) chemical's persistence in the environment, and (4) structure-activity relationship for mutagenesis*. Employing usage pattern as the criteria, what seems like an overwhelming task reduces to a reasonable number of chemicals whose testing does not present an enormous financial burden. For example, 200 prescription drugs control 69% of the market; this translates into 190 separate active ingredients (28). The non-prescription, or over-the-counter (OTC), drug market contains as many as 500,000 products (3). Although Congressmen were told that this plethora boils down to 216 active ingredients (53), our survey indicates that at least another 30 "real" active ingredients, not of the ilk of oatmeal, cottonseed oil, etc., should have been on that list of chemicals that can appear in OTC's. While a final number of active substances was not determined in our survey, it is felt that the number does not exceed 300 (28). On a positive note, a third of these are probably uninteresting as they are comprised of such presumably non-mutagenic items as vegetable oils, salts, and alcohols.

Once again, a variegated and voluminous market as that of the pesticides can be managed by concentrating on the market leaders: 19 fungicides, 37 herbicides, and 31 insecticides control 94%, 98%, and 93% of their respective markets (6).

*Such a vital relationship is in the process of development at the Environmental Mutagen Information Center, Oak Ridge, Tennessee (52).

With the banning of F. D. & C. Red No. 2 in February 12, 1976, only ten synthetic colorings remain approved for use in food. Regarding food additives in general, a priority list for them undoubtedly exists in the FDA since that body was ordered by the President in 1969 to review the safety of food additives; this was in response to the banning of former GRAS (Generally Recognized As Safe) additives, the cyclamates, due to their carcinogenic property. Disclosure of such a list is of paramount importance if the Toxic Substances Control Act is to work. In general, a lot more statistical information is needed of the government, including the kind that will allow the development of a meaningful priority list for industrial and miscellaneous chemicals.

BENEFIT-RISK ANALYSIS

In past cases, concern for the mutagenic activity of a compound surfaced prior to a comprehensive evaluation of the suspected product. In almost every instance, regulatory agencies and various expert committees were asked to make decisions on the continued use of a product with incomplete information. Polarization frequently occurred between consumer advocates and the manufacturer of the product. With a minimal amount of information, consumer groups advocated the restriction of the product, while the manufacturer usually questioned the reliability of the incriminating data and took the view that more definitive work was needed prior to taking any action. The Government usually vacillated between the two extremes. It would be the hallmark of the Toxic Substance Control Act if it reduces this situation. Though it is doubtful that we will ever have enough data to satisfy some manufacturers, we should determine minimal requirements before concluding that a chemical is mutagenic. Regardless of the intended use of the product, no decision on the restriction or the elimination of the chemical should be made solely on the basis of non-animal testing. A compound should not be considered non-mutagenic if the B error is of such magnitude to preclude detecting of a minimal mutagenic effect. The experiment should be constructed so that a doubling over background rate (cytogenetic response, increase in point mutations, etc.) is significant at the 5% level. A positive response in any of the animal systems should be sufficient to characterize the compound as a mutagen. This positive finding will be sufficient to eliminate a non-nutritive food additive or non-prescription drug from the market place. The elimination or prudent exposure to other positive compounds such as drugs, pesticides, and industrial chemicals will depend on the use-benefits outweighing the potential risks.

BIBLIOGRAPHY

1. Fulda, Thomas R., Prescription Drug Data Summary: 1974, DHEW Pub. No. (SSA) 76-11928, 1974, p. 25.
2. The Top 200 Drugs, Pharmacy Times 42(4): 37-44, 1976.
3. Hodes, B., Nonprescription Drugs: An Overview, Int. J. Health Serv. 4(1): 125-130, 1974.
4. U. S. Tariff Commission, Synthetic Organic Chemicals: U.S. Production and Sales, GPO, Washington, D.C., annual.
5. U. S. Bureau of the Census, Statistical Abstract of the United States, GPO, Washington, D.C., 1975, p. 5.
6. EPA, Pesticide Study Series 5: Pollution Potential in Pesticide Manufacturing, GPO, Washington, D.C., 1972.
7. USDA, The Pesticide Review, annual, Agricultural Stabilization and Conservation Service, 1975.
8. Pimentel, David, Extent of Pesticide Use, Food Supply, and Pollution, N.Y. Entomol. Soc. 81:13-13, 1973.
9. EPA, Pesticide Study Series 2: The Use of Pesticides in Suburban Homes and Gardens and Their Impact on the Aquatic Environment, GPO, Washington, D.C., 1972.
10. Hall, Richard L., Food Additives, Nutrition Today 8(4): 20-28, 1973.
11. Saunders, H. J., Food Additives, Chem. Eng. News 44(42): 200-120, 44(43): 108-128, 1966.
12. Train, Russell E., speech delivered by the Administrator of EPA before the National Press Club, February, 1976, as reported in The American City and Country, May, 1976, p. 34.
13. DHEW, Facts of Life and Death, DHEW Pub. No. (HRA) 74-1222, GPO, Washington, D.C., 1974, p. 31.
14. DHEW, Life Tables, Vital Statistics of the United States, 1973, DHEW Pub. No. (HRA) 75-1104, GPO, Washington, D.C., 1975, table 5-5.
15. Hayes, Wayland J., Toxicology of Pesticides, Wilkins and Wilkins, Baltimore, 1975, p. 17.
16. DHEW, Cancer Rates and Risks, DHEW Pub. No. 1148, GPO, Washington, D.C., 1964, p. 6.

17. Council on Environmental Quality, Environmental Quality -- Sixth Annual Report of the Council on Environmental Quality, GPO, Washington, D.C., 1975, p. 28.
18. DHEW, Cancer Rates and Risks, DHEW Pub. No. (NIH) 75-691, GPO, Washington, D.C., 1974, p. 3
19. Murray, James L., Impact of Cancer: Years of Life Lost Due to Cancer Mortality, J. Nat'l Cancer Inst. 52(1): 3-7, 1974.
20. DHEW, What Are the Facts About Genetic Disease, DHEW Pub. No. (NIH) 75-370, GPO, Washington, D.C., 1974, p. 6.
21. Friedrich, U. and J. Nielsen, Chromosome Studies in 5,049 Consecutive Newborn Children, Clin. Genet. 4:333-343, 1973.
22. Carr, D. H., Chromosomal Anomalies in Human Fetuses, Res. in Reprod. 4: 3-4, 1972.
23. _____, Chromosomes and Abortion, Advances in Human Genetics, 2: 201-257, 1971.
24. Jacobson, Cecil B., lecture delivered at the First Annual Course in Principles and Practices of Genetic Toxicology, The University of Texas Medical Branch, Galveston, Texas, Sept., 1976.
25. Temin, R. et al., The Influence of Epistasis on Homozygous Viability Depression in *Drosophila Melanogaster*, Genetics 61: 497-519, 1969; Stanley Zimmering, supra note 24.
26. Lederberg, J., in The Mutagenicity of Pesticides Concepts and Evaluation, eds. S. S. Epstein and M. S. Legator, M. I. T. Press, Boston, 1971.
27. supra note 20, at 8.
28. Legator, M. S. and S. J. Rinkus, unpublished results.
29. EPA, Preliminary Assessment of Suspected Carcinogens in Drinking Water: Report to Congress, 1975, p. 3.
30. Chem. Eng. News. 52(4): 6, 1974.
31. Della, P. et al., Induction with Carbon Tetrachloride of Liver-Cell Carcinomas in Hamsters, J. Nat'l. Cancer Inst. 26: 855-863, 1961.
32. WHO, Evaluation of Carcinogenic Risk of Chemicals to Man, International Agency for Research and Cancer Monographs 1: 61-65, 1972.

33. Tomatis, L. et al., The Effect of Long-Term Exposure to DDT on CF-1 Mice, Intern. J. Cancer 10: 489-506, 1972.
34. Walker, A. I. et al., The Toxicology of Dieldrin (HEOD), I. Long-Term Oral Toxicity Studies in Mice, Food Cosmet. Toxicol. 11:415-432, 1973.
35. Carcinogenicity of Heptachlor and Chlordane, Sci. Total Environ. 6(2): 103-154, 1976.
36. Dowty, B. et al., Halogenated Hydrocarbons in New Orleans Drinking Water and Blood Plasma, Sci. 187: 75-77, 1975.
37. supra note 17, at 375 and 379-381.
38. Sci. News 106: 311, 1974.
39. supra note 17, at 23.
40. Eckhardt, Bob, U.S. Representative, supra note 24.
41. House of Representatives, 94th Congress, 2nd Session, Toxic Substance Control Act, Report No. 94-1679, September 23, 1976, p. 3.
42. Bridges, B. A., Some General Principles of Mutagenicity Screening and a Possible Framework for Testing Procedures, Environ. Health Persp., Exp't Issue No. 6 (Dec., 1973): 221-227
43. A combined government agency hearing on short-term mutagenicity testing methods, Washington, D. C., July 15, 1975.
44. McCann, J. and B. N. Ames, Discussion Paper: The Detection of Mutagenic Metabolites of Carcinogens in Urine with the Salmonella/Microsome Test, Annals N.Y. Acad. Sci. 269: 21-25, 1975.
45. Heddle, John A., Comparison of Tests for Mutagenicity or Carcinogenicity Using Assays for Sperm Abnormalities, Formation of Micronuclei and Mutations in Salmonella, Symposium on Quantitative Biology, Vol. 40, 1977, in the press.
46. Kolata, G. B., Industry Adopts Controversial "Quick" Tests, Sci. 192: 1215-1217, 1976.
47. Legator, M. S., and S. Zimmering, Genetic Toxicology, Annual Review of Pharmacology, 15: 387-408, 1975.
48. Kilian, D. J. et al., Industrial Monitoring: A Cytogenetic Approach, Annals N. Y. Acad. Sci. 269: 4-11, 1975.

49. The Delaney Clause Controversy, Prev. Med. 2(1): 123-170, 1973.
50. Gaudin, David et al, DNA Repair Inhibition: A New Mechanism of Action of Steroids With Possible Implications for Tumor Therapy, Proc. Soc. Exp. Biol. Med. 146: 401-405, 1974.
51. Buffler, Pat, personal communication.
52. Wassom, John S., Director of the Environmental Mutagen Information Center, supra note 24.
53. Hearings Before the Subcommittee on Monopoly of the Select Committee on Small Business: U. S. Senate, 92nd Congress, Part 2, July 21-23; Sept. 23, 1971: Mood Drugs, 937-938.

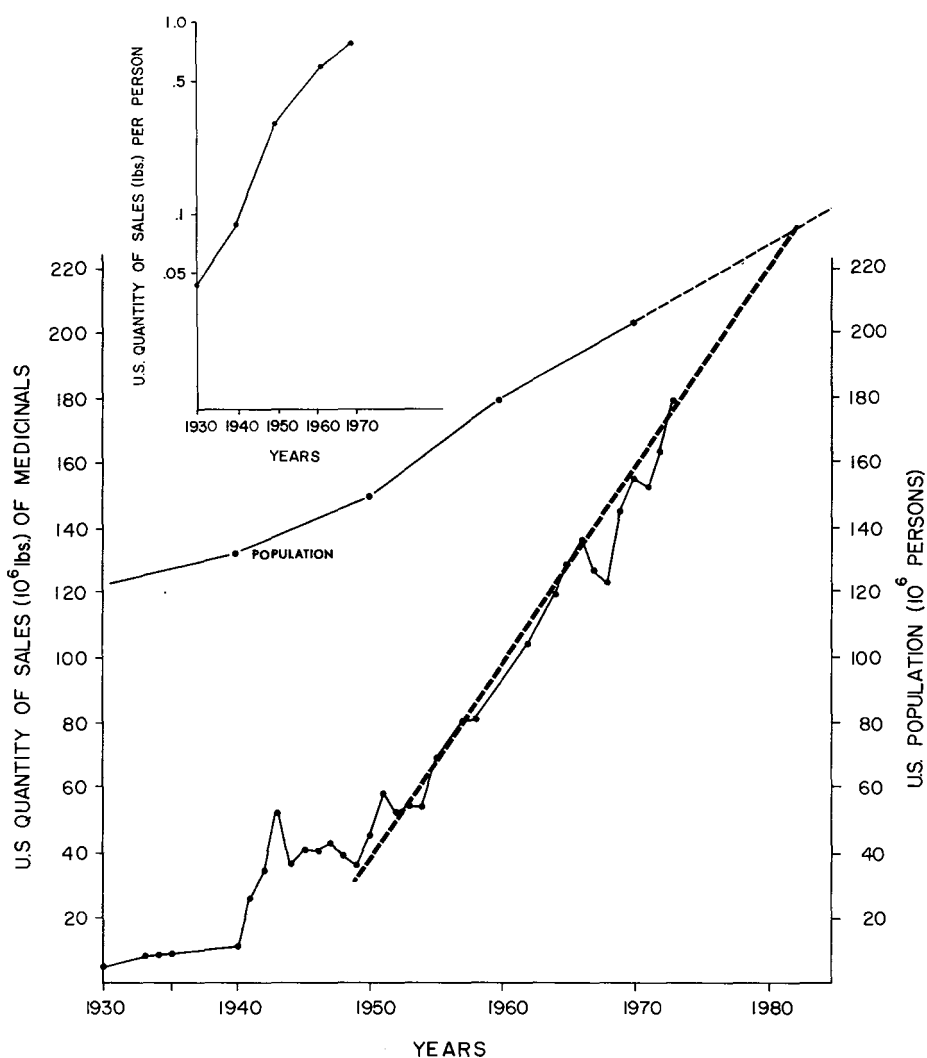


Figure I. U. S. Quantity of Sales of Medicinals and Population, 1930-1974. The data are for bulk medicinal chemicals having therapeutic value for human or veterinary use and for animal feed supplements (4,5).

Insert: U. S. Quantity of Sales of Medicinals Per Person, 1930-1970.

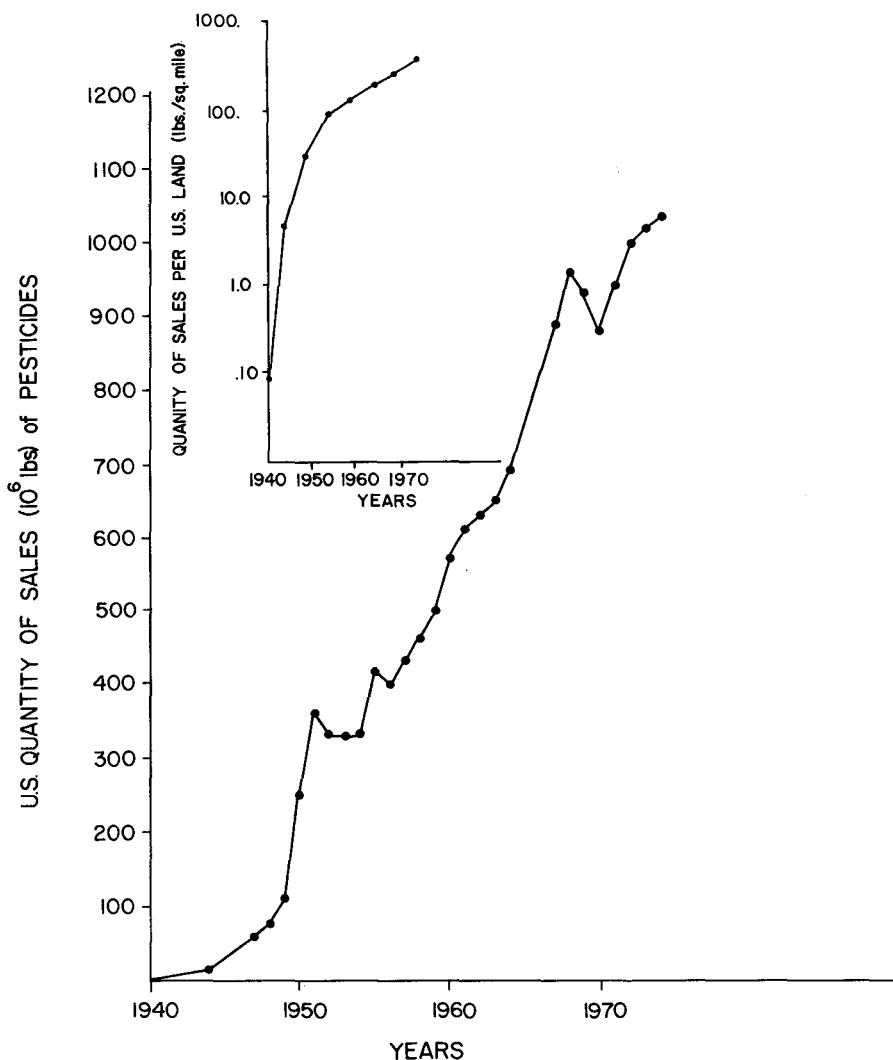


Figure II. U. S. Quantity of Sales of Pesticides, 1940-1974. The data are given in terms of 100% active ingredients for fungicides, herbicides, insecticides, rodenticides and related products such as plant hormones, seed disinfectants, soil conditions, soil fumigants and synergists (4).

Insert: Quantity of Sales Per U. S. Land (5), 1940-1974.

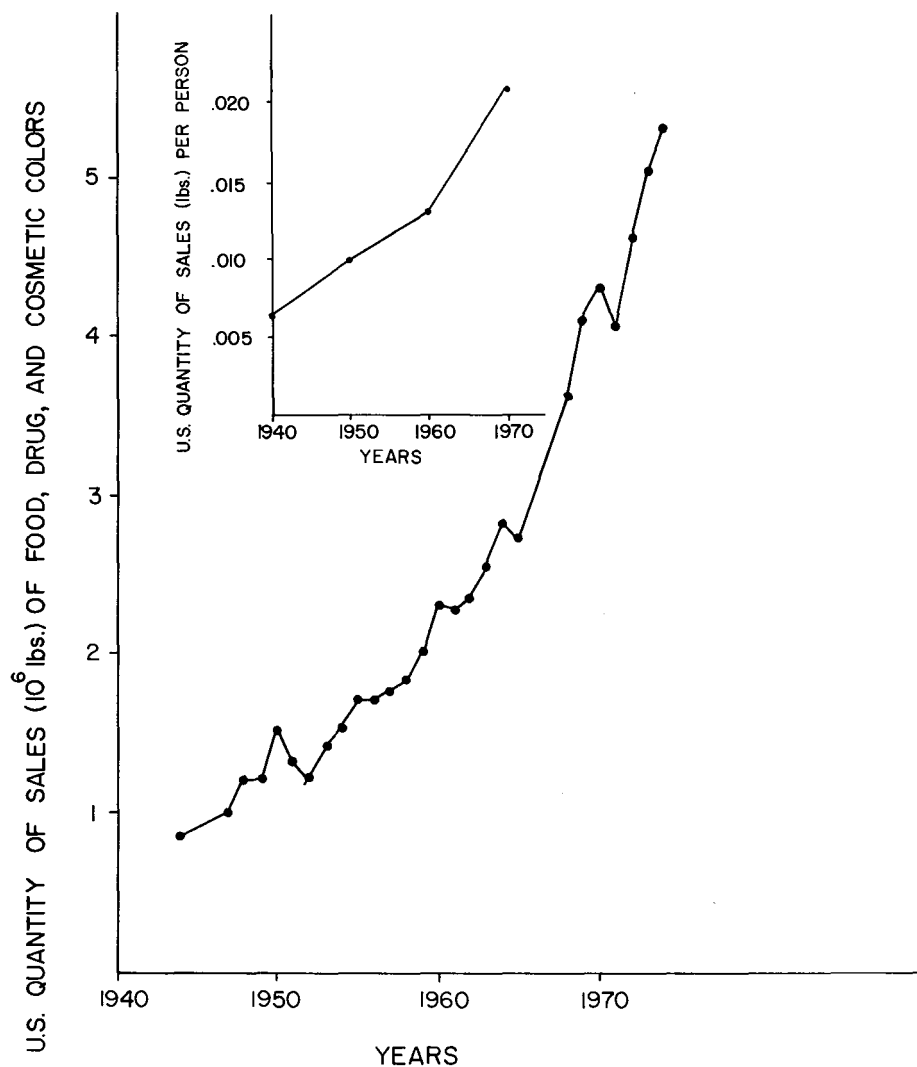


Figure III. U. S. Quantity of Sales of Food, Drug, and Cosmetic Colors, 1940-1970 (4).

Insert: U. S. Quantity of Sales of F. D. & C. Colors per Person, 1940-1970.

Leading Causes of Death, 1900, 1960, and 1970

Rank	Cause of death	Deaths per 100,000 population	Percent of all deaths
1900			
	(All causes)	(1,719)	(100)
1	Pneumonia and influenza	202.2	11.8
2	Tuberculosis (all forms)	194.4	11.3
3	Gastritis, etc	142.7	8.3
4	Diseases of the heart	137.4	8.0
5	Vascular lesions affecting the central nervous system	106.9	6.2
6	Chronic nephritis	81.0	4.7
7	All accidents ¹	72.3	4.2
8	Malignant neoplasms (cancer)	64.0	3.7
9	Certain diseases of early infancy	62.5	3.6
10	Diphtheria	40.3	2.3
	Total		64
1960			
	(All causes)	(955)	(100)
1	Diseases of the heart	366.4	38.7
2	Malignant neoplasms (cancer)	147.4	15.6
3	Vascular lesions affecting the central nervous system	107.3	11.3
4	All accidents ¹	51.9	5.5
5	Certain diseases of early infancy	37.0	3.9
6	Pneumonia and influenza	36.0	3.5
7	General arteriosclerosis	20.3	2.1
8	Diabetes mellitus	17.1	1.8
9	Congenital malformations	12.0	1.3
10	Cirrhosis of the liver	11.2	1.2
	Total		85
1970			
	(All causes)	(945.3)	(100)
1	Diseases of heart	362.0	38.3
2	Malignant neoplasms (cancer)	162.8	17.2
3	Cerebrovascular diseases	101.9	10.8
4	Accidents	56.4	6.0
5	Influenza and pneumonia	30.9	3.3
6	Certain causes of mortality in early infancy ¹	21.3	2.2
7	Diabetes mellitus	18.9	2.0
8	Arteriosclerosis	15.6	1.6
9	Cirrhosis of the liver	15.5	1.6
10	Bronchitis, emphysema, and asthma	15.2	1.6
	Total		85

¹ Violence would add 1.4 percent; horse, vehicle, and railroad accidents provide 0.8 percent.

² Violence would add 1.5 percent; motor vehicle accidents provide 2.3 percent; railroad accidents provide less than 0.1 percent.

³ Birth injuries, asphyxia, infections of newborn, ill-defined diseases, immaturity, etc.

Source: President's Science Advisory Committee Panel on Chemicals, *Chemicals and Health* (Washington, D.C.: Government Printing Office, 1973), p. 152; U.S. Department of Health, Education, and Welfare, Public Health Service, *Facts of Life and Death*, DHEW Pub. No. (HRA) 74-1222 (Washington, D.C.: Government Printing Office, 1974), p. 31.

Table I. Leading Causes of Death, 1900, 1960, 1970. (reproduced from reference 17, p. 9).

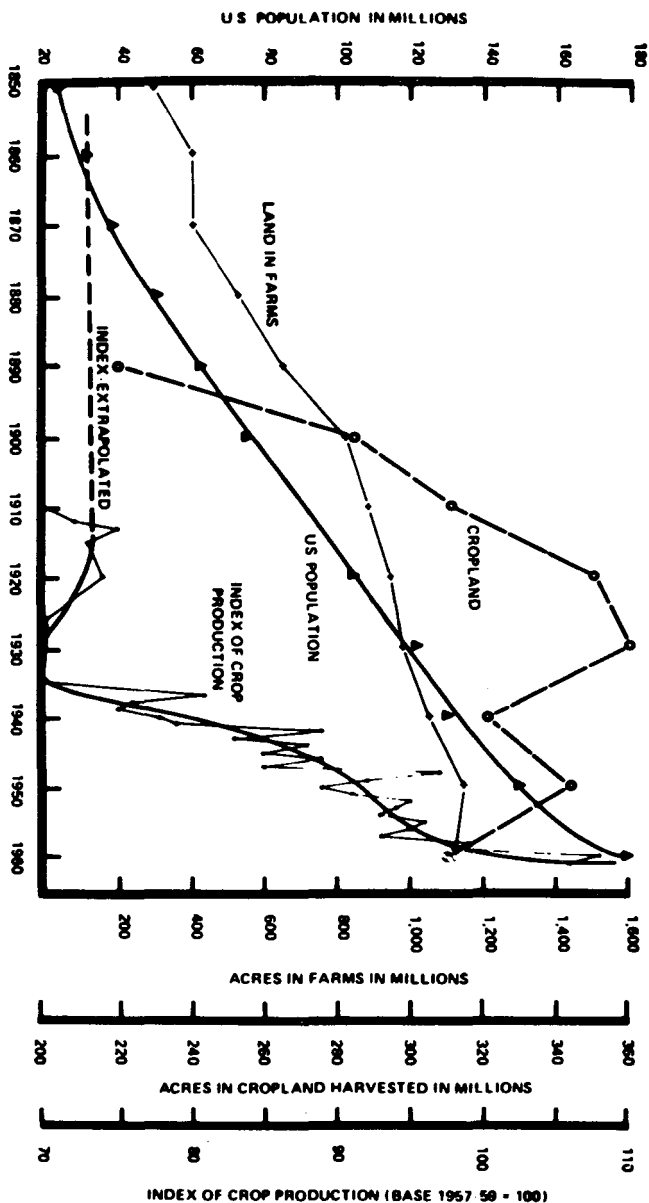


Figure IV. Crop Production, Cropland, Land in Farms, and U. S. Population, 1850-1960 (reproduced from reference 15, p. 17, with permission).

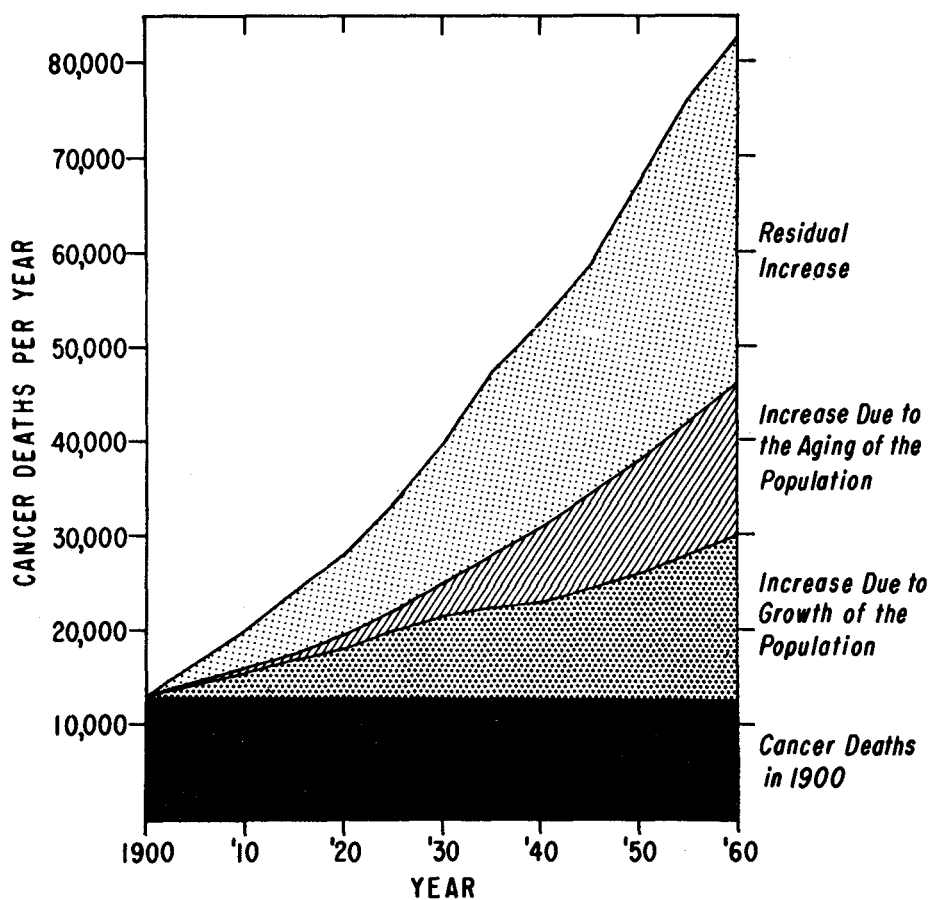


Figure V. Cancer Deaths, 1900-1960 (reproduced from reference 16).

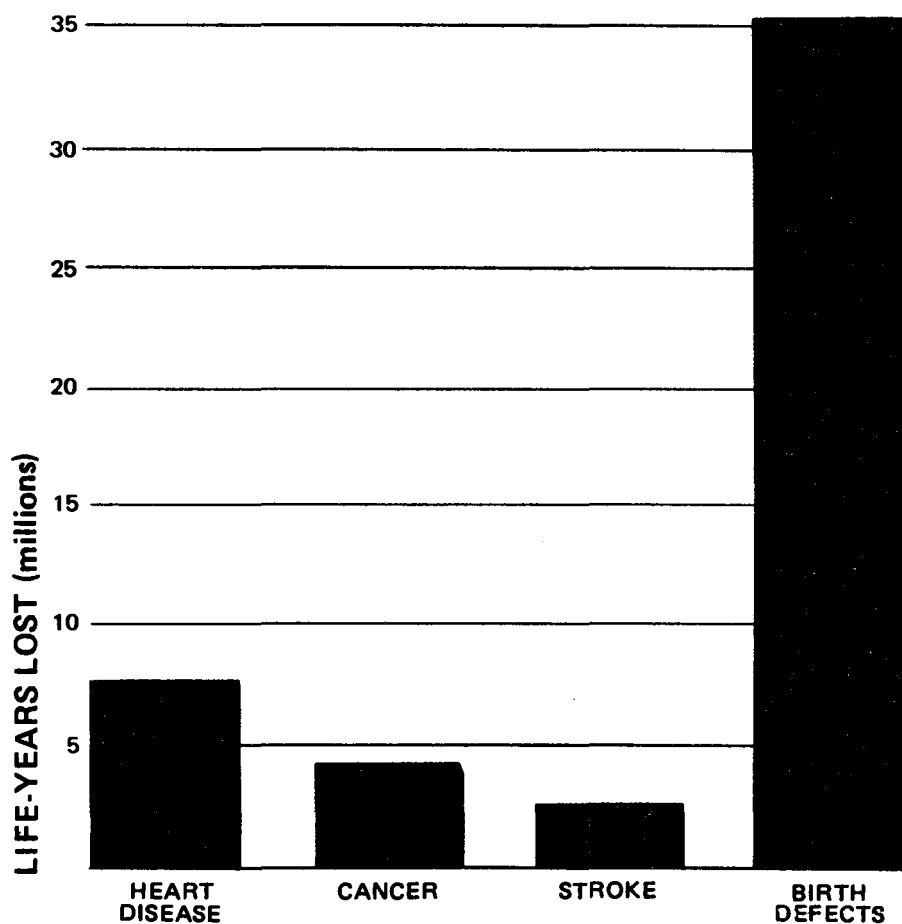


Figure VI. Life-Years Lost Due to Heart Disease, Cancer, Stroke, and Birth Defects (reproduced from reference 20, p. 8). Eighty percent of the birth defects are thought to be genetic in origin.

TOXIC SUBSTANCES AND CONGENITAL MALFORMATIONS

Josef Warkany, M.D.
Children's Hospital
Research Foundation
University of Cincinnati
College of Medicine

The thalidomide catastrophe made the general public aware of the fact that a pregnant woman's exposure to a toxic substance could endanger her unborn child. The shock produced by this event resulted in reactions which were partly healthy and partly unjustified, emotional and unscientific.

Let me put into historical perspective the question of embryonic and fetal injury by adverse environmental conditions. There are old reports on women working in industry and mines exposed to lead and mercury who suffered sterility and repeated abortions. The medical literature of the 19th century that deals with congenital malformations is replete with statements which blame children's defects on the father's or mother's alcoholism, tuberculosis or syphilis, etc. Many of these correlations were accidental and not causative, they could not stand critical analysis in later years. In the 1930's it seemed more "scientific" to consider most, if not all congenital malformations, as genetic and hereditary. One reason for this was that there was no experimental proof that the mammalian embryo can be deformed by adverse conditions of the mother. Such proof became available from the 1930's and 1940's and is now known under the name of experimental teratology, a very well developed science.

There is also ample proof by now that adverse environmental factors can deform human embryos and fetuses. These proven factors are not numerous and they are very heterogeneous. Environmental teratogens in man include iodine deficiency (which caused endemic cretinism), carbon monoxide, X-rays, toxoplasmosis, rubella, and organic mercury, which caused Minamata disease. Other proven environmental teratogens in man are cytomegalic inclusion disease, aminopterin, virilizing progesterones, thalidomide, chronic alcoholism, anticonvulsants, and warfaring.

And now to my topic: teratologic animal experiments. During the 19th century, countless teratologic experiments were done with eggs of chickens, amphibians, and fishes. Although many of these tests were of great scientific value they were not applicable to human conditions. It was thought that the human embryo and fetus were so well protected by the mother within the uterus that they could not be deformed by environmental factors. Aside from radiation, the first mammalian congenital malformations were produced by dietary deficiencies of pregnant animals. The following list describes some of the well established deficiencies that can result in typical malformations if strict experimental conditions are observed:

TERATOGENIC DEFICIENCIES

1933/45	VITAMIN A	RAT
1954/56	VITAMIN A	RABBIT
1940/44	RIBOFLAVIN	RAT
1946/56	FOLIC ACID	RAT, MOUSE
1948/57	PANTOTHENIC ACID	RAT
1950/57	OXYGEN	MOUSE
1953/57	VITAMIN E	RAT
1934/38	COPPER	SHEEP
1966/72	ZINC	RAT
1954/56	FASTING	MOUSE

These experiments were (and are) of great theoretical interest. They proved once and for all that environmental hardships imposed on pregnant females could cause structural malformations in the young. They showed that such hardships could induce syndromes of malformations, an effect which before was thought to be exclusively due to gene anomalies. And, being ultimately attributable to enzyme deficiencies, they imitated gene effects in many respects. Most important, they alerted physicians, epidemiologists and geneticists to the possibility that congenital malformations in children could also be caused by environmental adverse conditions. The list of factors teratogenic in humans that I described before was in part made possible by the animal experiments I have just listed.

And yet, as far as I know, none of these remarkable nutritional experiments has been simulated by human conditions. None of the deficiencies listed has been shown to produce malformations in man. And, vice versa, iodine deficiency which was such an important teratogen in man, has not been simulated in animal experiments. This taught us early that greatest caution is necessary in extrapolation of experiments to human situations. Early teratologists were fine scientists and neither propagandists nor alarmists.

I turn now from deficiencies to "positive" teratogens. So many substances and procedures have been used by now in teratologic experiments that it would take hours to read you the names of them. Instead I'd like to list some teratogenic substances which were found to be effective early, and which are used frequently for various reasons as research tools. To begin with, sex hormones can change secondary sex characteristics and virilize females and interfere with virilization of males.

Nitrogen mustard is a chemical compound which was a forerunner of many alkylating agents employed for production of severe malformations in rodents.

Trypan blue proved to be an excellent teratologic research tool which permitted production of exencephaly, hydrocephaly, spina bifida and other malformations. Its mode of action is still not

fully understood but it seems to interfere with the proper function of the yolk sac placenta of rodents. It has, of course, no direct importance for human malformations.

Many antimetabolites and antitumor drugs are good teratogens. Some of them have in rare cases caused congenital malformations in humans. I mentioned aminopterin before. Methotrexate, myleran, cyclophosphamide and chlorambucil were responsible for a few cases of malformations in children.

Some antibiotics are good teratogens in animals. We have used one, streptomycin; it yielded most interesting malformations in rats which had not been produced by other teratogens before.

Cortisone played an important role in experimental teratology because it produces cleft palate in very high incidences in certain strains of mice and not in others. It was used to demonstrate the combination of genetic with environmental factors in the expression of a specific congenital malformation. It is still widely used in cleft palate experiments.

Oral hypoglycemics and insulin have been teratogenic in rodents.

Hypervitaminosis A is an excellent teratogen in rats and mice. It results in exencephaly in many specimens, and has been used a great deal in research on anencephaly. A special form of vitamin A, retinoic acid, is quickly excreted and removed from the organism after producing malformations. For this reason, it was used in hamsters for exact timing of exposure and subsequent registration of malformations.

As early as 1953 and 1955, it was shown that vaginal application of phenylmercuric acetate in pregnant rats caused malformations in the young, and after the recognition of Minamata disease additional experiments have been done along this line.

Nicotine and caffeine have mild teratogenic effects in mice.

Among the most interesting teratogens are salicylates, including aspirin. These wonderful drugs, which are used by almost everyone and available in lethal amounts in every drug store, proved to our surprise to be teratogenic. In rats, aspirin is such a reliable teratogen that we use it as a standard procedure to test modifying factors. Although it is well known that salicylates taken by pregnant women in large amounts for suicidal purposes can result in fetal death, I do not know of a case that ended in congenital malformations of the child.

Thalidomide is the best known teratogen in man. In our context it is of interest because its teratogenicity in animals was demonstrated after the fact. In rabbits and in monkeys malforma-

tions have been produced a few months or years after the epidemia in human beings was over. Teratogenic effects described in rats and mice may not be due to the chemical properties of the drug. The malformations observed in these rodents do not resemble the thalidomide syndrome in man.

At the end of the list is sodium chloride which under certain experimental conditions can induce congenital malformations in mice. You can draw your own conclusions concerning regulatory procedures based on this animal experiment. Other teratogenic agents that are not defined chemically include X-irradiation in rats, mice, and rabbits; viruses in sheep, swine, rats, and hamsters; mechanical agents in the rat and the mouse; and antisera in the rat.

My discussion was limited to mammals and limited in the choice of teratogens. There are much more complete lists available (Cahen, Wilson) and there is the special Catalog of Teratogenic Agents by Shepard which discussed critically observations in man and animals.

From the choice of my examples of animal models you will conclude that I am opposed to uncritical application of results obtained in experimental animals to man. To show on television one deformed animal, or to publish in a newspaper results of teratologic experiments with the suggestion of stopping fabrication or consumption of the inducing substance, is not the way to proceed. Remember sodium chloride! In spite of this admonition, I consider animal experiments as very important. However, they must be interpreted with caution, with knowledge of the experimental conditions, and with rationality. If one finds a chemical compound teratogenic in animals, it should alert us to vigilance in human conditions. Experiments can show how environmental agents can deform an embryo or fetus. They may even contribute to prevention of certain adverse effects. But they must not be equated with human teratogenicity.

MALE AS WELL AS FEMALE MICE ARE AFFECTED BY
IN UTERO EXPOSURE TO DIETHYLSTILBESTROL

John A. McLachlan, Ph.D.
Environmental Toxicology Branch
National Institute of Environmental Health Sciences

Although many compounds are continuously introduced into our environment, relatively few of them have been examined for their potentially toxic effect on reproduction and development. For many agents, the prenatal organism is more sensitive to chemical injury than the corresponding adult. Many examples of teratogenic agents in animals as well as humans are available (Shepard, 1973); these are, however, the acute results of chemical exposure during the prenatal period.

Of equal importance are the long-term consequences of gestational exposure with chemicals to the offspring without gross malformations. In animals, at least, one such consequence of transplacental exposure to chemicals is increased incidence of certain types of cancer (Rice, 1973). Agents which require large doses and long exposure periods to induce cancer in adult animals do so at very much lower doses and/or after single administration during fetal development. Moreover, the latency period between exposure to a carcinogen and the onset of cancer is often shortened by transplacental exposure. Unfortunately, only a limited number of chemicals have been evaluated for latent toxicities (including cancer) which may follow prenatal treatment with the compound. The report of Herbst et al (1971) concerning the association of a previously rare genital tract neoplasm (clear-cell adenocarcinoma of the vagina) with gestational exposure to diethylstilbestrol (DES) represents the first example of transplacental chemical carcinogenesis in humans. More recent reports (Herbst et al, 1975) of vaginal and/or cervical malformations confirm that DES is also an organ-specific teratogen in human females.

In our laboratory, we have recently been studying the effects of prenatal exposure to DES on the development of the genital tract in male as well as female offspring; the mouse has been used as our animal model for the transplacental toxicity of DES.

In order to gain better understanding of the toxic effects of DES, its absorption, distribution within the body, metabolic pattern by the liver and other extrahepatic sites, and excretion were determined. An understanding of comparative metabolism and distribution of a xenobiotic is essential in the extrapolation of animal toxicity data to man. Although much is known about the metabolism and distribution of DES in nonpregnant animals, very little is known about its fate during gestation. In one of the few reports available, whole body autoradiography using mice, showed that radioactivity associated with maternally administered ^{14}C -DES reached the fetus more slowly and in lower concentra-

tions than that associated with ^{14}C -estradiol or estrone; a partial placental barrier to DES was proposed (Bengtsson and Ullberg, 1963; Ullberg and Bengtsson, 1963). Since localization of a biologically active drug in the target tissue may help understand the mechanisms of DES fetotoxicity, a study of the quantitative aspects of the maternal-fetal transfer of DES was performed (Shah and McLachlan, 1976).

The plasma disappearance curve of ^{14}C -DES, following its intravenous administration to 16-day pregnant mice, has been resolved into 4 major components with half-lives of approximately 4 seconds, 1 minute, 13 minutes, and 14 hours. The extremely rapid initial disappearance of ^{14}C -DES from the plasma can be attributed to several factors: (1) rapid uptake of ^{14}C -DES into red blood cells, the red cell/plasma ratio for ^{14}C -DES being close to 1 within 7 seconds after administration, (2) accumulation of ^{14}C -DES by the maternal liver, the parent compound and its metabolites reaching concentrations 4 and 24 times their corresponding plasma levels by 2.5 minutes after treatment, and (3) extensive metabolism of ^{14}C -DES, the parent compound accounting for less than half the total plasma radioactivity within 5 minutes after dosing.

Although radioactivity corresponding to DES rapidly penetrated all the maternal tissues examined (uterus, muscle, liver, placenta), it reached the fetal compartment relatively slowly. ^{14}C -DES levels in the placenta were higher than the corresponding values in the fetus and did not reach comparable levels in the fetus until almost one-half hour after injection. However, when selected fetal tissues were analyzed for their ^3H -DES content, one-half hour after treatment, an approximately four-fold accumulation of the parent compound relative to fetal plasma was found in the genital tract.

These results indicate that the initial movement of DES from the mother to the fetus is restricted, probably by the placenta. However, after an appropriate time, the drug does reach the fetal compartment and accumulates in the fetal reproductive tract.

It should be stressed that the relationship of the toxicity of a xenobiotic and its localization in a target site is far from clear. Bollengier et al. (1972) failed to demonstrate selected accumulation of ^3H -estradiol in the testes or pituitaries of mice susceptible to estrogen-induced tumors in these sites as compared to non-susceptible strains. However, the importance of drug distribution and metabolism studies to overall toxicological evaluations cannot be emphasized enough. In the case of DES, Klaassen (1973) was able to decrease the LD_{50} of the compound in rats 140 times by ligating the bile ducts and thus blocking its major route of excretion. And phenobarbital pretreatment has been reported to increase the metabolism and decrease the uterotrophic activity of orally administered DES (Levin et al., 1968).

In order to evaluate the prenatal toxicity of exposure to hormonally active environmental chemicals, experiments were done in our laboratory in which pregnant mice were treated with DES. Following subcutaneous treatment with DES in corn oil on the 9th through 16th day of gestation, mice were permitted to deliver their young. The offspring were then evaluated for altered function of the genital tract.

In the female offspring, one of the most striking effects was the dose-related decrease in reproductive capacity as determined by repetitive forced breeding techniques (Table 1). Over the 32 week period that the animals were observed, the effects ranged from a minimal subfertility at the lowest dose to essential sterility at the two highest doses. It should be pointed out that the highest daily dose used in this study (100 ug/kg) was roughly 1/20th the average daily dose, based on body weight, given to pregnant women therapeutically (100 mg/woman).

Lesions of the genital tract contributed to the infertility in the DES treated female mice. The incidence of lesions was dose-related and included cystic hyperplasia of the endometrium and uterine adenocarcinoma. Histological changes in the vaginal epithelium which may resemble adenosis and/or adenocarcinoma included the presence of glandular elements and cellular atypia and require further evaluation.

Although the effect of prenatal exposure to DES on the female genital tract has been extensively studied (see, for example, McLachlan and Dixon, 1976 for review), the effect of such exposure on the male offspring is unclear.

Male mice exposed to DES in utero also showed impaired genital tract function (McLachlan et al., 1975). For example, 60 percent of the males derived from mice treated with DES (100 ug/kg) were sterile. This effect was not seen in mice exposed to the lower doses studied. In addition, when groups of these males were sacrificed and examined at 9 to 10 months of age, no significant lesions were noted in those exposed to the lower doses of DES. However, the genital tracts of 75 percent of the male offspring of mice treated with the highest dose of DES (100 ug/kg) were altered.

The seminal vesicle and/or coagulating gland of the prostate in 6 of 24 animals had nodular enlargements. The enlargements in five of these animals were associated with squamous metaplasia, but adjacent to and in the duct of the coagulating gland of one DES-exposed offspring, there were downgrowths and cellular pleomorphism suggesting a more serious, and possible preneoplastic, lesion. Inflammatory lesions were also seen in those mice with nodules in the seminal vesicle.

Although squamous metaplasia of the accessory sex glands of

male rodents given high doses of estrogen has been described (Burrows, 1935; Greene et al., 1940; Arai, 1968; Triche and Harkin, 1971), carcinoma of the male rodent accessory sex glands is very rare. For example, treatment of mice with large doses of DES as adults results in mammary (Okey and Gass, 1968) or testicular tumors (Andervont et al., 1957), but other genital tract neoplasms were not noted. Administration of DES at a dose of approximately 1 gm/kg during neonatal life failed to produce cancer of the male accessory sex glands although epididymal cysts and fibrotic testes were observed (Dunn and Green, 1963).

In our experiments, testicular changes were found in 15 of 24 mice exposed to DES in utero. Six of the affected mice had at least one intraabdominal testis, which was hypoplastic, fibrotic and usually contained small nodules and sheets of interstitial cells. Eight animals had epididymal cysts which were usually associated with a fibromuscular outgrowth from the testis.

The correlation between animal studies (McLachlan et al., 1975) and human studies (Bibbo et al., 1975) of male offspring from DES-treated pregnancies demonstrates the importance of developmental studies to toxicological evaluations. Since epididymal cysts and prostatic inflammation were observed in the males of both species exposed prenatally to DES, the further question of reduced fertility in humans should be considered. Compromised reproductive capacity resulting from prenatal chemical exposure is certainly of importance in safety evaluation of chemicals. A future role of transplacental toxicology is suggested by the studies outlined in this report. Lesions were induced in the genital tract of fetal animals in a matter of days as contrasted with months of exposure in adult animals. Thus, prenatally-induced cancer or subfertility, such as demonstrated by these studies with DES, suggest unique problems in safety evaluation.

REFERENCES

1. Andervont, H.B., Shimkin, M.B. and Canter, H.Y. 1957. Effect of discontinued estrogenic stimulation upon the development and growth of testicular tumors in mice. *J. Natl. Cancer Inst.* 18: 1-39.
2. Arai, Y. 1968. Metaplasia in male rat reproductive accessory glands induced by neonatal estrogen treatment. *Experientia* 24: 180-181.
3. Bengtsson, G. and Ullberg, S. 1963. The autoradiographic distribution pattern after administration of diethylstilbestrol compared with that of natural oestrogens. *Acta Endocrinol.* 43: 561-570.
4. Bibbo, M., Al-Naqeeb, M., Baccarini, I., Gill, W., Newton, M., Sleeper, K.M., Sonek, M. and Wied, G.L. 1975. Follow-up study of male and female offspring of DES-treated mothers. A preliminary report. *J. Reprod. Med.* 15: 29-32.
5. Bollegnier, W.E., Eisenfeld, A.J. and Gardner, W.U. 1972. Accumulation of ³H-estradiol in testes and pituitary glands of mice and strains differing in susceptibility to testicular interstitial cell and pituitary tumors after prolonged estrogen treatment. *J. Natl. Cancer Inst.* 49: 847-852.
6. Burrows, H. 1935. Pathological conditions induced by oestrogenic compounds in the coagulating gland and prostate of the mouse. *Am. J. Cancer* 23: 490-512.
7. Dunn, T.B. and Green, A.W. 1963. Cysts of the epididymis, cancer of the cervix, granular cell myoblastoma, and other lesions after estrogen injection in newborn mice. *J. Natl. Cancer Inst.* 31: 425-455.
8. Greene, R.R., Burrill, M.W., and Ivy, A.C. 1940. Experimental intersexuality. *Am. J. Anat.* 67: 305-345.
9. Herbst, A.L., Ulfelder, H. and Poskanzer, D.C. 1971. Adenocarcinoma of the vagina. Association of maternal stilbestrol therapy with tumor appearance in young women. *N. Engl. J. Med.* 284: 878-881.
10. Herbst, A.L., Poskanzer, D.C., Robboy, S.J., Friedlander, L. and Scully, R.E. 1975. Prenatal exposure to stilbestrol. A prospective comparison of exposed female offspring with exposed controls. *N. Engl. J. Med.* 292: 334-339.
11. Klaassen, C.D. 1973. The effect of altered hepatic function on the toxicity, plasma disappearance, and biliary excretion of diethylstilbestrol. *Toxicol. Appl. Pharmacol.* 24: 142-149.

12. Levin, W., Welch, R.M. and Conney, A.H. 1968. Decreased uterotropic potency of oral contraceptives in rats pretreated with phenobarbital. *Endocrinology* 83: 149-156.
13. McLachlan, J.A., Newbold, R.R. and Bullock, B. 1975. Reproductive tract lesions in male mice exposed prenatally to diethylstilbestrol. *Science* 190: 991-992.
14. McLachlan, J.A. and Dixon, R.L. 1976. Transplacental toxicity of diethylstilbestrol: a special problem in safety evaluation. In, *Advances in Modern Toxicology*, Volume 1, part 1, ed. M.A. Mehlman, Hemisphere Publishing Corporation, Washington, D.C. (in press).
15. Okey, A.B. and Gass, G.H. 1968. Continuous versus cyclic estrogen administration: Mammary carcinoma in C3H mice. *J. Natl. Cancer Inst.* 40: 225-230.
16. Rice, J.M. 1973. An overview of transplacental chemical carcinogenesis. *Teratology* 8: 113-126.
17. Shah, H.C. and McLachlan, J.A. 1976. The fate of diethylstilbestrol in the pregnant mouse. *J. Pharmacol. Exp. Ther.* 197: 687-696.
18. Shepard, T.H. 1973. *Catalog of teratogenic agents*. Baltimore: John Hopkins Univ. Press.
19. Triche, T.J. and Harkin, J.C. 1971. An ultrastructural study of hormonally induced squamous metaplasia in the coagulating gland of the mouse prostate. *Lab. Invest.* 25: 596-606.
20. Ullberg, S. and Bengtsson, G. 1963. Autoradiographic distribution studies with natural oestrogens. *Acta Endocrinol.* 43: 75-86.

TABLE 1
FERTILITY OF FEMALE MICE EXPOSED TO DES PRENATALLY

<u>Maternal DES Dose (ug/kg)</u>	<u>Number of Offspring</u>	<u>Total Reproductive Capacity of Female Offspring* (% control)</u>
0	74	100.0
0.01	55	89.4
1	54	76.0
2.5	18	49.4
5	16	22.6
10	61	8.0
100	39	4.3

Timed pregnant CD-1 mice (at least 20 animals per group) were treated subcutaneously with diethylstilbestrol from the 9th to the 16th day of gestation, *determined by repetitive forced breeding techniques and expressed as the total number of live young born per mouse over a 32 week interval; (McLachlan, Shah, and Dixon, 1976, unpublished).

TRANSPLACENTAL EFFECTS OF DIETHYLSTILBESTROL
ON THE HUMAN MALE FETUS:
ABNORMAL SEMEN AND ANATOMICAL LESIONS
OF THE MALE GENITAL TRACT

W.B. Gill, G.F.B. Schumacher, and M. Bibbo
Departments of Surgery (Urology), Obstetrics
and Gynecology, and Pathology
University of Chicago

ABSTRACT

The in utero effects of DES (Diethylstilbestrol) on the human male genital tract are reported in this follow-up study of male offspring of DES-treated mothers. Both anatomical and functional abnormalities were significantly greater in the DES-exposed males as compared to placebo-exposed control males whose mothers were all participants in a prospective, randomized, double-blind study of the effects of DES on pregnancy at the Chicago Lying-In Hospital during the early 1950's.

Epididymal cysts, hypotrophic testes, capsular induration of the testes, and microphallus (flaccid penis <4 cm in length) were among the more common genital lesions found in over 25% of 163 DES-exposed males as compared to a 6.5% incidence in 168 control males. Spermatozoa analysis revealed severely pathological changes (Eliasson score ≥ 10) in 28% of 39 DES-exposed males and 0% of 25 control males. Pathological spermatozoa (Eliasson score ≥ 5) were found in 46% of 39 DES-exposed males vs. 12% of 25 placebo-exposed control males.

Abnormal findings on physical examination combined with pathological semen (Eliasson score ≥ 5) were found in 28% of the DES-exposed males vs. 0% of the control males. Cytologic examinations did not reveal malignant cells from the following materials: urine before and after prostatic massage or ejaculation, prostatic fluids and aspirates from epididymal cysts.

Diethylstilbestrol (DES), a synthetic estrogenic hormone, has been extensively used in several ways that bring this chemical into contact in utero with the developing human fetus (1,2,3). The transplacental hazards of DES on the developing human female fetus were originally described by Herbst and co-workers in their report of clear-cell adenocarcinoma of the vagina in

This study was supported in part by a contract (Dr. Marluce Bibbo, Principal Investigator) from the National Institute of Child Health and Human Development NIH-H01-HD-4 2850MB.

adolescent women (4).

The transplacental effects of DES on the human male fetus was originally reported by our group (5,6). Both anatomical (epididymal cysts, hypoplastic testes....) and functional (abnormal semen) abnormalities were significantly greater in the DES-exposed males as compared to placebo-exposed control males whose mothers were all participants in a prospective, randomized, double-blind study of the effects of DES on pregnancy at the Chicago Lying-In Hospital during the early 1950's.

MATERIALS AND METHODS

The effects of DES on pregnancy were evaluated two decades ago at the University of Chicago (1) in a prospective, double blind randomized study of 2,000 consecutive pregnancies which resulted in 840 women receiving DES and 806 women receiving placebos. DES was administered orally at the rate of 5 mg per day starting with the seventh week of gestation and increased by 5 mg per day every second week up to a maximum daily dose of 150 mg by the thirty-fourth week. Over 160 males in each of the two groups (DES-exposed and placebo-exposed) have been traced and evaluated to date. Neither the patients nor the examining physician and laboratory personnel were aware of the group (DES or placebo) to which the male offspring belonged.

Anatomical abnormalities were sought by: 1) meticulous physical examination, 2) urine cytology (cytology of urine pre and post prostatic massage or ejaculation), prostatic fluid, and aspirated epididymal cysts and 3) biopsies, where feasible (epididymis...).

Functional abnormalities were searched for by: 1) evaluation of the medical history (age of puberty, first ejaculation, first intercourse, urinary tract infections, venereal infections and fathering of children), 2) determination of the level of hormones in the blood, follicle stimulating hormone (FSH) and interstitial cell stimulating (ICSH = LH), by means of radioimmunoassay (7) and plasma testosterone (8), and 3) semen analyses (9,10) on ejaculates produced during the clinic visits after sexual abstinence of three days or more. The volume of the whole ejaculate was measured, and sperm count and percent motile spermatozoa were determined between one and two hours after ejaculation (double or triple determinations). Smears were fixed with methanol and stained with Giemsa solution (20 drops of Giemsa stain (Fisher Scientific Company) per 5 ml of distilled water); a differential count of normal (oral) and abnormal forms was performed, including primitive cells of the germinal epithelium.

The semen quality was estimated by a score system for sperm concentration, percent motile sperm, motility grade and morphology (percent normal forms). The sum of the scores was used for the

following classification: <1 = normal; 2 - 4 = doubtful; 5 - 10 pathological; >10 = severely pathological (Eliasson) (10).

RESULTS

Anatomical Abnormalities

Table 1 summarizes the abnormal physical findings in these men in their early twenties. The DES-exposed offspring had a statistically significant higher incidence of epididymal and testicular abnormalities as compared to the unexposed controls. The exact nature of abnormal physical findings in the testes and epididymal areas was difficult to ascertain without biopsies. However, nine DES-exposed patients allowed their epididymal masses to be aspirated which revealed a straw-colored fluid that did not contain spermatozoa in six cases, and a slightly milky fluid that did contain sperm in three cases. Aspirates of two control males contained spermatozoa in only one case. Cytologic examination revealed only epithelial cells and amorphous precipitates without any material suggestive of malignancy.

One of the larger (3 cm diameter) cystic masses was surgically excised from the area of the superior epididymis of a DES-exposed patient. A thin wall structure was found which contained clear, straw-colored fluid and was lined by columnar epithelium without apparent secretory cells. Since no spermatozoa were found in the fluid, the criterion for spermatocele was absent, and a diagnosis of an epididymal cyst was made.

Cytologic examination of the urine specimens (pre- and post-prostatic massage or ejaculation) and prostatic fluids was negative for tumor cells in all of the DES-exposed as well as the control males.

Functional Abnormalities

The results of circulating blood hormone assays are summarized in Table 2. There were no significant differences demonstrable in the averages of any of the circulating blood hormones assayed. Two of the DES-exposed patients had the following association of abnormalities: bilateral hypotrophic testis (<2 x 1 cm), azoospermia, decreased plasma testosterone, elevated plasma ICSH, elevated FSH and eunuchoid body habitus. Without chromosomal analyses and testis biopsies, we are not yet certain whether these represent Klinefelter's syndrome or another type of hypergonadotropic hypogonadism.

The distribution of spermatozoa analyses is given in Table 3 by Eliasson's method which combines the sperm count, the sperm motility, the motility grade, and the sperm morphology into one quantitative number. Note that 11 of 39 (28%) of DES-exposed

males vs. zero of 25 controls had severely pathological spermatozoa analyses. The combination of pathological (5 - 10) and severely pathological (10 <) Eliasson scores occurred in 46% of the DES-exposed males and in 12% of the controls.

Abnormal semen analyses are elaborated in Table 4. Perhaps the most familiar frame of reference is the sperm count of less than 20 million/ml, which occurred in 10 of 39 (26%) DES-exposed vs. zero of 25 control males.

The combination of severely pathological semen quality with an anatomical abnormality on physical examination occurred in 7/11 (64%) of the DES-exposed males who had severely pathological semen and 11/18 (60%) who had pathological semen (Eliasson score >5). None of the controls had such a combination. Therefore, It appears that in utero DES-exposure tends to produce abnormalities in the semen as well as anatomical lesions in the genital tract. Since either lesion can occur independently, however, they are not necessarily causally related.

DISCUSSION

The present study clearly indicates that transplacental effects of DES on the human male do occur. Administration of DES during pregnancy appears to be followed by latent effects on the fetal male genital tract that have shown up after puberty in the form of structural and functional changes that probably will impair fertility in a certain number of patients. With the delays in family planning prevalent today, it will probably be another decade before the actual infertility rate is known. However, since semen analyses give some insight into the probability of male infertility (10), one needs to carefully follow these and expanded numbers of patients with regard to the probable association of DES-exposure and subnormal fertility.

Although our study has not demonstrated carcinogenesis in human males to date, it has demonstrated that prenatal exposure to DES produces detectable anatomical changes in the male reproductive tract. Epididymal cysts, hypoplastic testes and induration of the testicular capsule have all been found in a greater incidence in the DES-exposed males. McLachlan's group (11) has very recently published data on the reproductive tract lesions in male mice exposed prenatally to DES. These workers found that of the male mice studied, 60% were sterile, 33% had epididymal cysts, 25% had undescended testes, 25% had nodular enlargement of the seminal vesicles and/or coagulating glands, which were associated with squamous metaplasia.

It is too early to definitely determine whether malignant lesions comparable to the vaginal and cervical clear-cell adenocarcinomas in DES-exposed female offspring will develop in the prenatally DES-exposed human males. Herbst (12) has proposed that

the embryonic origin of DES-induced vaginal adenocarcinomas in women is the persistence of Mullerian duct tissue in the squamous vaginal plate. Therefore, one probably should worry more about Mullerian duct remnants in the prostatic utricle and appendices of the testis of the human male. The cysts in the area of the efferent ductules and superior epididymis may indeed be abnormally DES-stimulated Mullerian duct remnants.

TABLE 1

ABNORMAL PHYSICAL FINDINGS IN THE MALE GENITAL TRACT

	<u>CONTROL MALES</u>	<u>DES-EXPOSED MALES</u>	<u>p VALUE</u>
Total Number Examined	168	163	
Epididymal Cysts			
Unilateral	8	16	<0.01
Bilateral	0	6	
Testicular Abnormalities			
Hypotrophic Testis			
Unilateral	2	8	< 0.005
Bilateral	0	4	
Capsular Induration	1	5	
Hypoplastic penis (microphallus) (≤4 cm flaccid length)	0	4	
Total Number of Patients with Some Abnormality on Physical Exam	11(6.5%)	41(25.2%)	<0.0005

TABLE 2
BLOOD HORMONE ASSAYS

	CONTROL MALES	DES-EXPOSED MALES
Total No. of Patients Analyzed	163	168
FSH (normal range)	80 - 400 ng/ml	
Average ng/ml	155	187
Standard Error	11.6	16.3
Range	7 - 760	5 - 1760
ICSH (normal range)	20 - 150 ng/ml	
Average ng/ml	34	40
Standard Error	2.6	2.9
Range	3 - 250	4 - 188
Testosterone (normal range)	270 - 1150 ng percent	
Average ng percent	765	718
Standard Error	20.1	29.1
Range	300 - 3048	5 - 1499
Number below normal range	0	1

TABLE 3
DISTRIBUTION OF ELIASSEN SCORES OF SPERMATAZOA ANALYSIS

<u>SUM OF ELIASSEN SCORES*</u>	<u>CONTROL MALES</u>	<u>DES-EXPOSED MALES</u>
0-1 Normal	18/25 (72%)	14/39 (36%)
2-4 Doubtful	4/25 (16%)	7/39 (18%)
5-10 Pathological	3/25 (12%)	7/39 (18%)
10< Severely Pathological	0/25 (0%)	11/39 (28%)

*ELIASSEN SCORES = Sum of scores for sperm count, % motility, motility grade, morphology.

TABLE 4
ABNORMAL SEMEN ANALYSES

	<u>Control Males</u>	<u>DES-Exposed Males</u>
Semen Volume <1.5 ml	0/25 (0%)	10/39 (26%)
Sperm Count <20 x 10 ⁶ /ml	0/25 (0%)	10/39 (26%)
Sperm Motility <40%	4/25 (16%)	10/37 (27%)
Sperm Morphology <60% (Oval)	0/25 (0%)	8/37 (22%)
Eliasson Score >10 (severely pathological)	0/25 (0%)	11/39 (28%)

TABLE 5
PATHOLOGICAL SEMEN AND ANATOMICAL ABNORMALITIES

	<u>Control Males</u>	<u>DES-Exposed Males</u>
Pathological Semen Quality		
Score \leq 5	3/25 (12%)	18/39 (46%)
Score > 10	0/25 (0%)	11/39 (28%)
Genital Abnormalities on Physical Examination	5/25 (20%)	21/39 (54%)
Pathological Semen Quality Without Physical Abnormalities		
Score \leq 5	3/25 (12%)	7/39 (18%)
Score > 10	0/25 (0%)	4/39 (10%)
Physical Abnormalities with Normal or Doubtful Semen Quality	5/25 (20%)	10/39 (26%)
Pathological Semen Quality Associated with Physical Abnormalities		
Score \leq 5	0/25 (0%)	11/39 (28%)
Score >10	0/25 (0%)	7/39 (18%)

REFERENCES

1. Dieckmann, W.J., Davis, M.E., Rynkiewicz, L.M., et al: Does the Administration of Diethylstilbestrol During Pregnancy Have Therapeutic Value? *AM. J. Obstet. Gynecol.*, 66:1062, 1953.
2. Kuchera, L.K.: The Morning-After Pill. *J.A.M.A.*, 224:1038, 1973.
3. Marx, J.L.: Estrogens and the FDA. *Science*, 191:839, 1976.
4. Herbst, A. L., Scully, R.E.: Adenocarcinoma of the Vagina in Adolescence: A Report of Seven Cases Including Six Clear-Cell Carcinomas (So-Called Mesonephromas). *Cancer*, 25:745, 1970.
5. Bibbo, M., Al-Naqueeb, M., Baccarini, I, Gill, W, et al: Follow-Up Study of the Male and Female Offspring of DES Treated Mothers: A Preliminary Report. *J. Reprod. Med.*, 15:29, 1975.
6. Gill, W.B., Schumacher, G.F.B., Bibbo, M.: Structural and Functional Abnormalities in the Sex Organs of Male Offspring of Mothers Treated with Diethylstilbestrol (DES). *J. Reprod. Med.*, 16:147, 1976.
7. Odell, W.D., Rayford, P.L., Ross, G.T.: Simple Partially Automated Method for Radioimmunoassay of Human Thyroid Stimulating Growth, Luteinizing and Follicle Stimulating Hormones. *J. Lab. Clin. Med.*, 70: 973, 1967.
8. Ismael, A.A., Niswender, G.D., Midgley, A.R.: Radioimmunoassay of Testosterone without Chromatography. *J. Clin Endocrinol. Metab.*, 34:177, 1972.
9. Amerlar, R.D.: Infertility in Men: Diagnosis and Treatment. Philadelphia, F.A. Davis Company, 1966.
10. Eliasson, R.: Analysis of Semen. In *Progress in Infertility*. Edited by S.J. Behrman, R.W. Kistner, Second Edition. Boston, Little, Brown and Company, 1975, p. 691.
11. McLachlan, J.A., Newbold, R.R.: Reproductive Tract Lesions in Male Mice Exposed Prenatally to Diethylstilbestrol. *Science*, 190:991, 1975.
12. Herbst, A.L., Poskanzer, D.C., Robboy, S.J. et al: Prenatal Exposure to Stilbestrol (A Prospective Comparison of Exposed Female Off-Spring With Unexposed Controls. *N. Eng. J. Med.*, 292:334, 1975.

TRANSPLACENTAL CARCINOGENESIS: PRENATAL
DIETHYLSTILBESTROL (DES) EXPOSURE,
CLEAR CELL CARCINOMA AND RELATED ANOMALIES
OF THE GENITAL TRACT IN YOUNG FEMALES

William R. Welch, M.D., Ann B. Barnes, M.D.,
Stanley J. Robboy, M.D., and Arthur L. Herbst, M.D.

From the Departments of Pathology and Gynecology, Harvard Medical School, the James Homer Wright Pathology Laboratories and Vincent Memorial Hospital (Gynecological Service of the Massachusetts General Hospital), Boston, Massachusetts, and the Registry of Clear-Cell Adenocarcinoma of the Genital Tract in Young Females, Chicago, Illinois.

The historical roots of DES extend into the late 19th century when the concept of hormones was first conceived and research undertaken to identify internal secretions as well as to identify the organs which secreted and responded to them. During the 1920's, the "estrus hormone", or estrogen, was identified. By the early 1930's, the laboratory synthesis of the non-steroidal stilbene molecule which had weak estrogenic properties proved that this activity was not limited solely to compounds incorporating the steroidal structure of the naturally occurring estrogens. In 1938, Dodds in England synthesized a markedly potent estrogenic compound composed of the stilbene molecule with added diethyl groups; this was called diethylstilbestrol, or DES. Three unique features contributed to the immediate and sustained acceptance of DES; it was inexpensive to prepare; it was almost as potent as natural estrogens; and it could be administered orally. Hence, by 1940, an orally effective, inexpensive non-steroidal estrogen was commercially available, one usage of which became the treatment of high-risk pregnancy. The drug was used most extensively during the late 1940's and 1950's but continued to be administered for this condition until 1971. During this interval 500,000 to perhaps 2,000,000 women received DES during pregnancy.

Between 1966 and 1969, Dr. Arthur Herbst and several other of our colleagues at the Massachusetts General Hospital unexpectedly encountered seven young females with clear-cell adenocarcinoma of the vagina, a tumor which was extraordinarily rare in the medical literature up to that time. Subsequent investigation revealed that these young ladies were offspring of mothers who had received DES during pregnancy (1). This was the first apparent example of transplacental carcinogenesis in humans. The relationship between the development of this rare tumor and a prenatal exposure to DES or its related compounds, dienestrol and hexestrol, was soon confirmed by other investigators and led to a warning by the Food and Drug Administration on the use of DES in pregnancy. Because of the obvious implications of this finding on the lives of exposed

Supported in part by Grant R01-CA13139-05 and Contract NO1-CN-45157 from the National Cancer Institute and by a Junior Faculty Fellowship (to William R. Welch) from the American Cancer Society.

females, all of whom were young and in good health otherwise, the Registry of Clear-Cell Adenocarcinoma of the Genital Tract in Young Females was established by my colleagues with support from the American Cancer Society and the National Cancer Institute in order to centralize data on all cases occurring in women born after 1940 (2). In this way, data could be accumulated on a rare tumor permitting rapid dissemination of epidemiological, diagnostic and therapeutic information to the medical community.

As of June, 1976, in spite of extensive search, approximately 150 cases of cancer with a positive maternal DES history have been accessioned in the Registry. Most have been from North America, but some with documented histories of prenatal DES exposure have come from Europe, Australia and Africa. In every case for which accurate maternal histories are available, the drug had been administered prior to the 18th week of gestation. At the time of diagnosis, the patients ranged in age from 7 to 28 years with the average age in the late teens. One interesting epidemiological observation that has emerged from this study has been the marked increase in frequency of the cancer after the age of 14, raising the possibility that some event related to puberty, such as secretion of estrogen by the patients' own ovaries, might play a role in the development of the tumor. Most of the patients sought medical attention because of vaginal bleeding and discharge. However, one sixth of the patients were asymptomatic and the tumor was detected only because of a routine gynecological examination or Papanicolaou smear, which sometimes was performed only because the patient realized she had been exposed to DES. It is cautioned, however, that cytological smears alone are not an absolutely reliable method of detection and therefore must be combined with adequate pelvic examination and appropriate biopsies. Subsequent to the discovery of the relation between DES and the vaginal tumors, it became apparent that the tumor may arise in the cervix also. The tumors range from 3 mm. to more than 10 cm. in diameter and usually appear nodular or polypoid but occasionally may be flat or present as an indurated mass in the wall of the vagina. Microscopically, the tumors resemble the clear-cell cancers found in the ovary and endometrium of older women. The most characteristic cell of the tumor is a large polygonal cell which contains abundant glycogen; the glycogen is leached from the cells during the processing of tissues for histological examination and leaves the clear cytoplasmic space which accounts for the name "clear-cell" carcinoma.

Radical surgical and radiation therapy have been effective in treating most cases diagnosed at an early stage. A strong argument for early detection of these tumors is the fact that almost every patient with an asymptomatic tumor is alive and free of disease after therapy. In contrast, almost a fourth of the remaining patients in the Registry have developed recurrences or have died.

Although many characteristics of this malignancy are now known, there has been much confusion and uncertainty concerning the non-malignant anomalies also associated with DES exposure, especially vaginal adenosis and vaginal and cervical ridges. The latter, which occur in approximately 20 per cent of the exposed population, have been given a variety of descriptive names (collar, rim, hood, cockscomb, pseudopolyp) based on the configuration each has imparted to the vagina or cervix (3,4,5). Adenosis is an abnormal but benign condition wherein glandular tissue is present in the vagina. It may be suspected when the vaginal mucosa contains red, granular patches or fails to stain when an iodine solution is applied to the vaginal surface. The glycogen-rich squamous epithelium that normally lines the vagina stains a deep brown color when exposed to iodine; the presence of glycogen-poor non-staining epithelium provides a useful guide for areas that require biopsy and potentially harbor adenosis or, rarely, the carcinoma.

The reported prevalence of adenosis in the exposed population varies from 35 to 90 per cent depending on, among other things, the definition of adenosis used by individual investigators and the method used to sample the vaginal lining (6). The prevalence is also greatly affected by the week during pregnancy in which the mother began taking DES. In one study, adenosis was present in 73 per cent of offspring where the drug was administered prior to the 8th week of gestation but in none of those exposed after the 18th week (3).

Three questions often asked about adenosis are: 1. What happens to adenosis as the patients grow older? 2. Is adenosis the precursor of the cancer, and if so, what is the rate of malignant transformation? and, 3. What is the recommended treatment, if any? At the present time the natural history of adenosis is unknown. Adenosis may provide the bed from which the cancers arise, but actual malignant transformation of adenosis has been difficult to document. The numeric risk of any exposed individual developing carcinoma is impossible to state with certainty since to date the size of the exposed population and the natural history of the events preceding cancer have not been adequately defined. Even among patients with adenosis, it is extraordinarily small. Whether other new conditions may develop as this population ages is speculative but possible. Some investigators have already predicted that the glycogen-poor (metaplastic) squamous epithelium which is associated with adenosis will result in increased rates of squamous-cell malignancy. This problem is currently under intensive scrutiny. Because of the above problems, the Division of Cancer Control and Rehabilitation of the National Cancer Institute has recently commissioned the DESAD (DES Administration) project, of which the Massachusetts General Hospital is a participating center, and which has as its charge the study of the incidence and natural history of vaginal and cervical abnormalities associated with prenatal exposure to DES. We anticipate that the project will answer many vital questions and will help formulate needed guidelines for the future health care requirements of this population.

REFERENCES

1. Herbst AL, Ulfelder H, Poskanzer DC: Adenocarcinoma of the vagina. Association of maternal stilbesterol therapy with tumor appearance in young women. *New Eng J Med* 284:878-881, 1971.
2. Herbst AL, Robboy SJ, Scully RE, et al: Clear-cell adenocarcinoma of the vagina and cervix in girls; analysis of 170 Registry cases. *Am J Obstet Gynec* 119:713-723, 1974.
3. Herbst AL, Poskanzer DC, Robboy SJ, et al: Prenatal exposure to stilbesterol. A prospective comparison of exposed female offspring with unexposed controls. *New Eng J Med* 292:334-339, 1975.
4. Robboy SJ, Scully RE, Herbst AL: Pathology of vaginal and cervical abnormalities associated with prenatal exposure to diethylstilbesterol (DES). *J Repro Med* 15:13-17, 1975.
5. Robboy SJ, Scully RE, Herbst AL: Vaginal and cervical abnormalities related to prenatal exposure to diethylstilbesterol (DES). In Pathology of the Female Genital Tract, ed. A Blaustein, Springer Verlag, 1976.
6. Herbst AL, Scully RE, Robboy SJ: Problems in the examination of the DES exposed female. *Obstet Gynec* 46:353-355, 1975.

BLOOD DYSCRASIAS AND CHILDHOOD TUMORS AND EXPOSURE TO CHLORINATED HYDROCARBON PESTICIDES

Peter F. Infante
Division of Surveillance, Hazard Evaluations
and Field Studies
National Institute for Occupational Safety and Health

Samuel S. Epstein
Professor of Environmental Occupational Medicine
School of Public Health
University of Illinois

INTRODUCTION

Chlordane is an active ingredient in many household and garden pesticides that have been in common use for about 25 years (1). The total usage of chlordane in the U.S. for 1975 is estimated at 25 million pounds. About 70 percent was applied around the home, while the remainder was used for agricultural purposes, primarily corn crops (2). There have been various reports on possible association between exposure to chlordane-based formulations, and to related chlorinated hydrocarbon pesticides, and blood dyscrasias in humans (3-15). Evidence also indicates that chlordane and heptachlor, as well as other chlorinated hydrocarbon pesticides, are carcinogenic in experimental animals (2,16-19). Following administration of chlordane, both hepatic and extrahepatic tumors have been induced in mice and rats (1). Perinatal administration of low doses of heptachlor have induced rare "lipomatous" renal tumors, similar to those reported following treatment of rats with pyrrolizidine alkaloid carcinogens (20). Heptachlor epoxide residues have been detected in cord blood and in the organs in stillborn human infants, indicating transplacental passage (21). Technical grade chlordane is also mutagenic in *S. Typhimurium*, strain TA 100, without metabolic activation (V. Simmon, personal communication). To date, no epidemiologic studies have been undertaken to negate the laboratory studies demonstrating the carcinogenicity and mutagenicity of chlordane. Nor is there currently a reporting mechanism to gather pesticide-related pathologic conditions in our population.

For these reasons, five new cases of childhood tumors associated with pre- and postnatal exposure to chlordane, plus six additional cases of aplastic anemia and acute leukemia are presented.

CASES OF NEUROBLASTOMA

The children with neuroblastoma were diagnosed at a single pediatric hospital between December, 1974 and February, 1976. During this period, a total of 14 cases of neuroblastoma were admitted. A history of exposure to toxic agents indicates that

five cases had prior exposure to chlordane formulations. These cases are summarized in Table 1.

Neuroblastoma, Case #1

A two-year and eight-month-old girl was diagnosed in December, 1974, as having neuroblastoma. Excisional biopsy and tissue examination indicated a poorly differentiated (Stage III) neuroblastoma of the right adrenal gland involving the kidney. Both organs were removed, the child was given radiation treatment and she is currently receiving chemotherapy. This case has been recently reported elsewhere in detail (22).

During the first trimester of gestation, the 28-year-old mother's home was treated for termite infestation with 125 gallons of a one percent chlordane formulation. This was made by mixing one gallon of technical chlordane with 95 gallons of water. About equal amounts of chlordane were used inside and outside the house. On the outside of the house, the entire perimeter was treated with subsurface injection, and the ground surface also was sprayed in an area extending one to two feet from the house. On the inside of the house, holes were drilled into the cement blocks in the basement, chlordane was injected into the holes, and the holes were recemented. The mother states that the odor from the pesticide was offensive inside the house and, since she was concerned about the possible effects on the developing fetus, she slept at a neighbor's home on the evening of the first day of application. On the second day of application, the spraying of chlordane was completed outside the house and that evening the mother slept at her own home. The mother relates that the odor was very strong for only 3-4 days, but was noticeable for two weeks after the initial treatment. On the evening of the second day of treatment, the weather turned cold, the windows were closed and the furnace was turned on. This created a particularly strong odor in the basement, where the mother spent 25-30 hours a week typing. One year later, chlordane was again sprayed around the outside of the house. Other noteworthy information from the prenatal history is that the mother took two Valium tablets in early pregnancy and also had received a general anesthetic for a tonsillectomy during the first month of pregnancy. The general anesthesia was induced with thiopental sodium and was maintained with halothane and nitrous oxide.

Neuroblastoma, Case #2

A four-year-old boy presented in July, 1975, with a two-week history of lethargy, decreased appetite and right leg pain. Abdominal palpation and subsequent diagnostic radiographs demonstrated a mass in the right paravertebral area. Exploratory laparotomy revealed a tumor in the right paravertebral area with metastases to regional lymph nodes. Bone marrow biopsy showed malignant cells. Extent of the diseases and histopathologic

evaluation indicated a metastatic neuroblastoma (Stage IV).

A history of exposure to toxic substances revealed that the mother had been exposed to chlordane during her entire pregnancy. One year prior to conception, the house was treated with chlordane for roach infestation using procedures indicated in case #1. The operator returned every six months to spray under the sink and baseboards. Prior to and subsequent to birth, there was no history of exposure to other chemicals for the mother or child. Aspirin was the only medication taken during pregnancy.

Neuroblastoma, Case #3

A four-year-old girl was diagnosed in November, 1975, as having a neuroblastoma. Two weeks prior to admission, the child experienced abdominal pain. At the time of hospitalization, an upper left quadrant and flank mass was present. A radiograph of the chest revealed a mediastinal enlargement. An intravenous pyelogram showed an abnormal left kidney; bone marrow analysis showed no diagnostic abnormality. Exploratory laparotomy revealed an unresectable abdominal tumor arising from the left suprarenal area. Tumor biopsy and histopathologic evaluation indicated a Stage IV metastatic neuroblastoma.

When the child was 23 months old, the parents moved into a recently purchased house. Just prior to moving, the newly acquired house was treated for termites by spraying chlordane around the baseboards and on the inside of the kitchen cupboards. Holes were drilled into the basement walls and procedures were carried out as described in case #1. Additional exposure to other chemical agents known to induce tumors in either experimental animals or humans was not identified through interview with the parents.

A history of prior illness revealed that the child had a urinary infection at ages 18 and 24 months, at which time diagnostic radiographs were made. She also was given an x-ray for trauma to the head in May, 1974.

Neuroblastoma, Case #4

A three-year and nine-month-old boy began to limp and changes were observed in his gait five weeks prior to admission in September, 1975. Radiographs and bone scan showed involvement of several areas of the skeleton, particularly the skull and long bones. Bone marrow aspiration indicated an almost complete replacement of the marrow cells by clumps of tumor cells which were consistent with neuroblastoma (Stage IV). A primary tumor site could not be identified.

Two years prior to birth and also when the child was two years old, the house was treated for termite infestation with chlordane, using the usual procedures for application. Between

these two periods of chlordane application, a commercial pesticide was used intermittently. Other noteworthy information is that the child was given x-rays to rule out a possible bowel obstruction within the first seven days of postnatal development. The remainder of the history was negative.

Neuroblastoma, Case #5

A six-year-old girl was diagnosed in February, 1976, as having neuroblastoma, Stage IV. The child developed leg and hip pains two months prior to admission. Subsequently, bone marrow analysis indicated tumor cells. Histopathologic evaluation indicated neuroblastoma.

Because of ant infestation, the father purchased a container of chlordane dust which he applied several times around the outside foundation of the house in the Autumn of 1973, when the child was three years and eight months of age. The process was repeated in the Spring of 1974. The parents could not recall having applied chlordane after the latter period. A history of prior illness was unremarkable.

Histories of pre- or postnatal exposure to additional toxic agents indicated that the mother of case #1 had a halothane/nitrous oxide general anesthetic in the first month of pregnancy. Cases #3 and #4 had postnatal exposure to x-rays. Thus, five of fourteen children diagnosed at the hospital during this period of time had a known home exposure to chlordane. Of the nine additional cases of neuroblastoma, history of exposure to chlordane is not known, because no formal study has yet been conducted.

CASES OF APLASTIC ANEMIA AND LEUKEMIA

The cases of blood disease associated with chlordane exposure had been treated at several hospitals in the past several years. These cases are summarized in Table 2.

Blood Disease, Case #1 (Aplastic Anemia)

A 15-year-old boy had been well until February, 1975, when he gradually developed pallor, bleeding gums, and fatigue, and was diagnosed as having aplastic anemia. A history revealed that the boy sprayed Isotox* and chlordane on bushes and around the foundation outside the house with a garden hose attachment once a month between June and October of 1974. His clothing frequently

*Active Ingredients: Carbaryl (1-naphthyl N-methyl-carbamate; S(2-(Ethyl-sulfinyl ethyl) o,o-dimethyl phosphorothioate; 1, 1-bis (p-chlorophenyl)-2,2,2-trichloroethanol.

became soaked with the formulation. Chlordane also was applied along door sills and in some kitchen cabinets where food staples were stored. There was no history of exposure to other substances which have been incriminated as possible etiologic agents for aplastic anemia.

On admission to the hospital in March, 1975, the patient appeared pale, otherwise the physical examination was within normal limits. Examination of the peripheral blood showed: hemoglobin, 5.3 gm. percent; reticulocyte count, 2.6 percent; white cell count, 3,500, with a differential of banded cells, 20 percent; segmented cells, 22 percent; lymphocytes, 53 percent; and monocytes, five percent and a platelet count of 20,000. A bone marrow examination showed that all the normal cell types were present, but decreased in number, and the bone spicules were devoid of cells. The hematological findings were consistent with a diagnosis of aplastic anemia. The subject is currently being followed as an outpatient. On follow-up through December, 1975, his blood count has remained the same: megakaryocytes were rare and erythropoiesis and myelopoiesis were diminished. Symptoms of fatigue and, at times, headache and dizziness persist.

Blood Disease, Case #2. (Aplastic Anemia)

A 28-year-old man had been hospitalized in February, 1975, with a two-month history of easily bruising, fatigue, and light-headedness. One week prior to admission, he noticed bleeding from his gums and nose and black stools (generally indicative of upper gastrointestinal hemorrhage). The patient was diagnosed as having aplastic anemia. Upon admission, the initial hematologic evaluation of peripheral blood indicated; hemoglobin, 4.8 grams percent; red blood cells, 1.42×10^6 ; white cell count, 3,300; with a differential of polymorphonuclear cells, 10 percent; banded cells 1 percent; monocytes, 3 percent and lymphocytes 86 percent; platelets were 35,000. A bone marrow biopsy revealed absence of hemotopoietic material, no megakaryocytes or hemotologic precursors were present. The patient was given multiple platelet and red cell transfusions, and he became nearly refractory to these. He was given antigen therapy with no response and eventually bone marrow transplant was performed. On January 11, 1976, the patient expired.

The subject was self-employed as a realtor and in the year prior to hospitalization he had used various insecticides, paints, thinners and varnishes while restoring homes. In the six months prior to admission, he had used rather extensively a 74% chlordane formulation (Ortho-Klor 74) as well as Diazinon.

Blood Disease, Case #3 (Aplastic Anemia)

A 68-year-old man was hospitalized on October 28, 1969, because of increasing dyspnea, weakness and the development of a

pallid complexion. An analysis of the peripheral blood indicated that the hemoglobin was 6.1 gm percent and that the white blood cell count was 1,000. Following administration of two units of packed cells, the hemoglobin rose to 10.2 gm percent, while the white blood cells remained at 1,200 with a cell differential of polymorphonuclear, 18 percent; monocytes, two percent; eosinophils, four percent and lymphocytes, 76 percent. The platelet count was 70,000 and the reticulocyte count was 1.6 percent. A bone marrow biopsy indicated aplasia for all cell elements, including megakaryocytes. The hematologic evaluation was consistent with aplastic anemia. Treatment with prednisone was started on November 9, 1969, and the patient was discharged a few days later in greatly improved condition. The subject developed heart congestion and expired January 30, 1970.

A history of exposure to toxic agents ascertained from the surviving spouse revealed that the subject had purchased a bottle of chlordane which he had used for soil treatment around rose bushes, for crab grass control and for termite control in the basement of their house. The wife stated further that her husband had sprayed a 74 percent chlordane formulation (Ortho-Klor 74) in the basement more than once during a three-year period prior to onset of symptoms.

Blood Disease, Case #4 (Acute Stem Cell Leukemia)

A nine-year-old girl was diagnosed to have acute stem cell leukemia. Three weeks prior to admission the child developed symptoms of fatigue associated with a low grade fever. On admission to the hospital in December, 1969, she appeared pale with a few scattered purpuric areas. Analyses of peripheral blood indicated: hemoglobin, 4.5 percent; reticulocyte count 0.1 percent; white cell count 6,250 with a differential of blast cells, 35 percent; lymphocytes, 60 percent and neutrophils, five percent and a platelet count of 2,500. A bone marrow aspirate showed that erythropoiesis was depressed, myelopoiesis was replaced by stem cells and megakaryocytes were absent. The patient expired in October, 1973.

The child was born in November, 1960. Beginning in the spring of 1961, the house was treated annually with chlordane for termite infestation. In 1967, floorboards were removed from the entire house and 220 gallons of a two percent chlordane formulation were poured over the ground inside the house. The father relates that the chlordane took about 10 days to soak into the ground. The floorboards were then replaced, and the family moved back into the house. Two years later, the child developed leukemia. The mother did not take any medication nor did she receive any x-rays during pregnancy. The remainder of the history also was negative.

A 23-year old man was diagnosed to have acute lymphoblastic leukemia in July, 1973, and is currently in remission. Past history reveals that he had been in good health, except for a chronic ear infection, until two weeks prior to hospital admission, when he developed an external otitis and adjacent facial cellulitis, which did not respond to penicillin. At the time of admission in July, 1973, the patient had a temperature of 105°F, pancytopenia, with a white cell count of 1,800 consisting of 95 percent lymphocytes; a hemoglobin of 10.9 gm. percent; and a platelet count of 90,000. The bone marrow was consistent with a diagnosis of acute leukemia.

Before hospitalization, the only medication the patient had received was ear drops. He had been employed by a lawn care firm for a three-year period prior to hospitalization, during which he sprayed lawns with chlordane, Banvel D, Diazinon, and 2,4-D*. Also, a week before hospital admission, the patient removed the paint from his car and repainted it over a one-week period, using various paints, strippers and thinners.

The patient responded to antibiotic treatment for the acute infection and was treated with a regimen of prednisone. One month later, his platelet count rose from 90,000 to 220,000, the white cell count rose from 1,800 to 6,000; and the hematocrit rose from 23 to 30 with two units of whole blood. Remission was induced with prednisone and vincristine. He also was treated with cranial irradiation and intrathecal methotrexate to prevent symptomatic central nervous system leukemia.

Blood Disease, Case #5 (Acute Lymphoblastic Leukemia)

A 37-year-old male was hospitalized for possible leukemia in August, 1975. For two months prior to admission, he had been feeling weak and fatigued. Ten days prior to admission, he had his teeth cleaned by a dentist. He noticed that the gums did not stop bleeding until the second day after his visit. At the same time, he noticed bruise marks over his extremities.

Upon hospitalization, his initial hemoglobin was 10.7 gm percent, reticulocyte count was 1.6 percent and platelets were 30,000. Bone marrow biopsy initially showed 100 percent cellularity with markedly depressed erythropoiesis. Almost all of the recognized cells seemed to be immature cells. Granulocytic stem cells were 82 percent. The impression was consistent with acute

*Banvel D; Dicamba or 3,6-dichloro-o-anistic acid or 2-methoxy-3,6-dichlorobenzoic acid; Diazinon, 0,0-diethyl 0-(2-isopropyl-6-methyl-4-pyrimidinyl) phosphorothioate; Dursban; 0,0-diethyl 0-(3,5,6-trichloro-2-pyridyl) phosphorothioate; 2,4-D; 2,4-dichlorophenoxyacetic acid.

myelomonocytic leukemia. The patient was placed on acute leukemia protocol. He expired October 24, 1975.

A history of exposure to toxic agents revealed that the subject frequently used chlordane around the house for insect control for the past 10 years prior to admission. He often placed a full strength 44% chlordane formulation (Ortho-Klor 44) in a paint pan and brushed it on all of the basement windows, both inside and outside the house several times a year. At times he complained of headache for a few hours after the application. Once a month, he also sprayed chlordane on the lawn and on the side of the house with a garden hose attachment apparatus for protection against grubs. His wife states that he purchased the chlordane in a pint bottle several times a year. A history of exposure to additional agents possibly associated with aplastic anemia or leukemia was negative.

A history of exposure to other toxic agents indicated that blood disease case #1 was exposed to Isotox; case #2 was exposed to Diazinon and various paints; case #5 was exposed to Banvel D, Diazinon, Dursban and 2,4-D and one week before hospital admission, he was exposed to paints and paint thinners.

REVIEW OF CASES IN THE LITERATURE

Table 3 summarizes 25 previously reported cases of blood dyscrasias associated with exposure to chlordane or heptachlor, either alone, or in combination with other drugs. From these data, it can be seen that in six cases, chlordane or heptachlor alone was implicated; in 15 cases, chlordane exposure was accompanied with unspecified drugs some indicated as "toxic", or as "now known to be associated with blood dyscrasias;" and in four other cases, chlordane was accompanied with other specified pesticides or drugs. Of the 25 cases shown in Table 3, 19 were diagnosed as aplastic anemia, two as leukopenia and one each as hypoplastic anemia, hemolytic anemia, megaloblastic anemia, and thrombocytopenia.

In addition to the cases documented above, unspecified combinations of chlordane, lindane, and DDT were associated with 40 cases of pancytopenia, 10 cases of thrombocytopenia and 10 cases of leukopenia reported in the United States between 1955-1961 as shown in Table 4 (23). The data in Table 4 are taken from a report by the American Medical Association's Study Group on Blood Dyscrasias. Through a voluntary reporting mechanism, the Study Group compiled cases of blood disease suspected as being etiologically related to drug or chemical exposure (23). The type of blood dyscrasia is presented either by pesticide exposure alone or in combination with other unspecified "drugs not known to be toxic," plus those "known to be potentially toxic." In a majority of the cases, pesticide exposure was either alone or in combination with "drugs not known to be toxic." A maximum of

eight cases in Table 4 may have appeared in Table 3; i.e., the cases reported by Conley, 1955, (4), Muirhead et al., 1959, (9), and Huguley et al., 1961, (24), since the time period for the actual occurrence of these cases overlaps the period for the cases shown in Table 3. The case reported by Moore, 1955 (Table 3), occurred in 1953, which was prior to the existence of the Study Group and is probably not represented in Table 4.

Although chlordane and heptachlor have been suspended for major agricultural uses, no attention has been given to possible short- or long-term health risks involved with particular reference to carcinogenicity and blood dyscrasias through home application of chlordane, particularly for termite infestation. The five cases of blood dyscrasias in association with chlordane reported by Conley (4), Moore (8), Furie and Trubowitz (6) and Muirhead et al., (9), plus the 11 new cases of blood dyscrasias and childhood tumors reported in the present communication total 16 cases available with detailed exposure histories. Of these 16 cases, 12 had exposure to chlordane through home applications, three were involved with farm work and one worked for a lawn care firm. The greater number of cases associated with home use may be a reflection of a greater population at risk through home application. A second indication that health risks might be greater through home exposure can possibly be seen in the data for systemic chlordane poisoning presented by the American Medical Association's Committee on Pesticides. (4) Excluding one suicide, nine of 14 poisonings occurred through home use; four occurred through agricultural exposure and only one case occurred through industrial manufacture.

In the absence of epidemiologic studies, the statistical demonstration that any chemical or drug has an etiologic relation to blood dyscrasias, such as aplastic anemia and leukemias, is difficult to make because of the relatively low incidence of these conditions (11). For example, chloramphenicol-induced aplastic anemia was observed sporadically soon after the drug was released in 1949, but so few case reports appeared in the literature that its potential toxicity was not apparent until several years later (23,25). Because a large number of substances has been associated with blood dyscrasias (23) and the occurrence in the general population is rare, some type of idiosyncratic response might be involved. However, prior to exposure, the sub-population that might have such a response as a result of normal pesticide usage would be virtually impossible to identify.

RELATION OF APLASTIC ANEMIA TO LEUKEMIA

In the United States, the mortality rate for leukemia is about seven deaths per 100,000 population while mortality from aplastic anemia is about 0.5 deaths per 100,000 population. Whether aplastic anemia is a preleukemic process or a potentially leukemic disease most likely depends upon the interaction of the

etiologic agent with the host and the genetic make-up of the host's cells. The likely relation between aplastic anemia and leukemia may be a hereditary defect in the stem cells, which makes them susceptible to an agent which causes cell injury and leads to either aplastic anemia, leukemia or both.

Several conditions and agents associated with aplastic anemia also have been associated with an increased incidence of acute leukemia. The aplasia may be a phase in the development of leukemia, or the absence of normal marrow cells might increase the susceptibility of the patient to leukemogenesis. Congenital conditions such as Down's and Fanconi's syndromes and ataxia-telangiectasia that show either abnormal chromosomes and/or immune deficiencies show a high rate of leukemia (26-28). This suggests that instability of chromosomes and immune suppression predispose to leukemia. In Fanconi's anemia, increased chromosomal breaks, aplasia, and an increased incidence of leukemia are seen (29). Radiation is known to induce both aplastic anemia and leukemia (30-32), the latter peaking at about six to seven years after exposure (33). Chloramphenicol also has been associated with aplastic anemia (34). Some patients have later developed acute leukemia (35-37). The usual effect of this agent upon cells is to inhibit mitochondrial protein synthesis; however, in some people, because of a genetic biochemical defect, therapeutic concentrations of chloramphenicol inhibit DNA synthesis, which causes the bone marrow aplasia (38). Bone marrow cells resistant to chloramphenicol have been shown to occur in some patients with aplasia (39), suggesting the presence of at least two stem cell populations. Benzene has clearly induced aplastic anemia (40-42), and many times within a relatively short period after exposure. Benzene apparently acts by suppressing DNA synthesis of differentiated bone marrow cells and not by damaging stem cells. The subsequent development of acute myelogenous leukemia in some patients with benzene-induced aplastic anemia is well documented (40,41,43,44).

Patients who present with marrow aplasia, and who later developed leukemia, show a persistent reduction in normal cells, and the appearance of abnormal leukemic cells. Perhaps, the few remaining cells in aplastic anemia are either more susceptible to leukemogenic viruses or more susceptible to transformation to malignant blasts by the continuing efforts of the offending agent. The process appears to be similar in Fanconi's anemia, ionizing radiation, and in chemically-induced aplasia. Although the type of leukemia may vary in all of these diseases, the incidence of acute myelogenous leukemia is increased most markedly.

The association of paroxysmal nocturnal hemoglobinuria (PNH) with aplastic anemia (45-47) supports the presence of the development of two distinct cell populations. In PNH red cells from one of the populations are lysed by complement in the presence of

acidified serum. In aplastic anemia, the marrow stem cell may be transformed to an abnormal cell which cannot replicate, or one of the cell clones may be susceptible to a cytotoxic effect of an offending agent (28,45). Thus, aplasia may be the result of either an abnormality of the pluri potential stem cell or an altered micro-environment due to defective stromal cells, or both (48). An epidemiologic study to determine the proportion of patients with chemically-induced aplastic anemia who later develop acute leukemia would seem appropriate.

DISCUSSION OF NEW CASES OF BLOOD DYSCRASIA AND NEUROBLASTOMA IN RELATION TO REPORTS IN THE LITERATURE

It is apparent that our six blood disease cases are consistent with previous reports in the literature on the occurrence of various blood dyscrasias in association with the use of several chlorinated hydrocarbon pesticides. Since chlordane, heptachlor, toxaphene, lindane, DDT, dieldrin and aldrin are similar in chemical structure (Figure 1), chemical properties, and in toxicity in humans (49), findings that more than one of the above-mentioned substances might be associated with blood dyscrasias in humans would not be entirely unexpected.

Although exposure to chlorinated hydrocarbon pesticides has been related to aplastic anemia, to our knowledge neither chlordane, heptachlor, nor other chlorinated hydrocarbon pesticides have been reported in association with acute leukemia. However, several reports have indicated that subjects with aplastic anemia associated with various agents have later developed acute leukemia (35-37,40,41,43,45). In some cases, the conversion from hypoplastic anemia to leukemia occurred within a relatively short period of time (41,51). Although the leukemia is usually of the myeloblastic type, acute lymphatic leukemia also has been reported in association (50). Thus, substances such as chlorinated hydrocarbon pesticides that might be capable of inducing bone marrow suppression might also be capable of inducing acute leukemia within a relatively short period of time.

In regard to our cases of childhood neuroblastoma, we are not aware of previous reports, which have suggested associations between perinatal pesticide exposure and childhood tumors. However, in view of the evidence of transplacental passage of chlordane metabolites (21) and experimental animal studies, which have induced the carcinogenicity of chlordane, heptachlor (2,16,17) and heptachlor epoxide (52), the possibility of a transplacental carcinogenic mechanism should be experimentally investigated. (Another halogenated hydrocarbon, vinyl chloride, has been demonstrated to be a transplacental carcinogen in rats) (53). Because chlordane and the structurally similar chlorinated hydrocarbon pesticides are known to be fat-soluble and neurotoxic (49), tissue of the sympathetic nervous system may be target sites for related tumors in humans. Epidemiologic study in this area of investigation

also is needed.

STUDIES OF OCCUPATIONAL EXPOSURE TO CHLORDANE

Although the need for field studies to determine the consequences of chlordane use by the general population was expressed over twenty years ago (54), little epidemiologic data from which to develop valid inferences are available. Because of the small sample sizes, high turnover rates, and poor response to health interviewing, the results of several studies on possible ill-health effects among pesticide workers exposed to chlordane appear to be inconclusive. In three studies (54-56), the sample sizes of the current work force on which conclusions were based ranged from 15-34 workers and are totally inadequate. For example, Alvares and Hyman (55) reported no ill-health effects among 24 men, who had worked in the manufacture of chlordane for periods of time ranging from two months to five years. Inspection of their data shows that eight workers had been employed a year or less and only 12 subjects had exposure periods ranging between three to five years. The data indicate a high turnover rate and an extremely small sample size on which to base any conclusions. In another study, based on survey questionnaires (57), only 12 percent of the companies responded and less than 20 percent of the personnel at risk completed questionnaires. The latter study (57) indicates that the turnover rate of servicemen, the high exposure risk group, was 20 percent annually. Thus, subject selection as well as the response may have biased the results. Also, no mention is made of any attempt to contact employees who had discontinued service in any of these studies.

It is also interesting to note that the relative risk for developing leukemia appears to be greatest in farmers (25). One may question whether or not pesticide exposure, especially the chlorinated hydrocarbons, might be a contributing factor.

ENVIRONMENTAL DISTRIBUTION AND BODY BURDEN OF CHLORDANE AND HEPTACHLOR

Chlordane and heptachlor have a chemical structure similar to that of other chlorinated hydrocarbon pesticides such as dieldrin, lindane, and DDT. Technical chlordane consists of a complex mixture of compounds, whose ratios have been standardized since about 1950, and contains: 38-48% alpha- and gamma-chlordane; 7-13% pure heptachlor; 5-11% nonachlor; 17-25% chlordane isomers; plus other compounds in lesser amounts (17). Technical chlordane is formulated in varying strengths in different pesticidal preparations. Exposure to chlordane can occur through the intact skin, by inhalation of dust or sprays and by ingestion (58). Human poisonings and fatalities have been reported from dermal and oral exposure to chlordane (59-61).

Chlordane is one of the most widely used household and garden

pesticides. In 1974, 21 million pounds were used. Seventy percent was applied for termite control and other household use, while the remainder was used for agricultural purposes (2). Chlordane and heptachlor, or their metabolites, are persistent in the environment long after use (17). Treated soil is subject to water erosion ultimately leading to aquatic contamination. Chlordane also is widely distributed in ambient air and in household dust (62). Although low in water solubility, their affinity for lipids make chlordane and heptachlor subject to possible bioaccumulation and transfer in the food chain (17). From market basket surveys, components of technical chlordane and its metabolites were found commonly in dairy, meat, fish and poultry components of the diet (17).

As with other chlorinated hydrocarbon pesticides, metabolites of chlordane and heptachlor may accumulate in man (63). Oxychlordane and heptachlor epoxide residues have been detected in the adipose tissue in over 90% of large samples of hospital patients studied in 1970-72.

Heptachlor epoxide residues have been detected in the organs of stillborn infants (21) and also in samples of human milk (17). Because of this environmental accumulation, humans may be exposed from the time of conception on throughout adult life.

SUMMARY

1. New cases of blood dyscrasias and childhood tumors are reported following exposure to chlordane.
2. The cases of aplastic anemia are consistent with previous literature reports on associations between chlordane, similar chlorinated hydrocarbon pesticides and blood dyscrasias.
3. The cases suggesting association between chlordane exposure and leukemia are noteworthy in view of previous reports indicating that subjects with aplastic anemia convert to leukemia.
4. The cases suggesting association between chlordane exposure and neuroblastoma are of interest in view of recent data on the carcinogenicity and mutagenicity of chlordane and heptachlor and in view of data on environmental distribution and body burdens.
5. Previous epidemiologic studies are too limited to allow the development of valid inferences regarding the neoplastic risk of chlordane.
6. There is a need for epidemiologic study to evaluate short and long-term health risks associated with

chlordane in home use and in occupational use for the agricultural setting, for termite exterminators and for lawn care operators.

ACKNOWLEDGEMENTS

The authors thank Dr. Inta J. Ertel for helpful discussions on the relation of aplastic anemia to leukemia. The assistance of Dr. William A. Newton Jr. is also greatly acknowledged.

REFERENCES

1. Special Pesticides Review Group. (Fitzhugh OG and Fairchild HE Chairmen): Pesticidal aspects of chlordane in relation to man and the environment. US Environmental Protection Agency, Government Printing Office, Washington, 1975.
2. Epstein SS: The carcinogenicity of heptachlor and chlordane. *Sci Total Environ* 6:103-154, 1976.
3. Best WR: Drug-associated blood dyscrasias. *JAMA* 185:140-144, 1963.
4. Conley BE: The present status of chlordane. Council on Pharmacy and Chemistry, Committee on Pesticides. *JAMA* 158:1364-1367, 1955.
5. Friberg L and Martensson J: Case of panmyelophthisis after exposure to chlorophenothane and benzene hexachloride. *Arch Ind Hyg Occup Med* 8:166-169, 1953.
6. Furie B and Trubowitz S: Insecticides and blood dyscrasias: Association of chlordane exposure and self-limited refractory megaloblastic anemia. *JAMA* In Press.
7. Loge JP: Aplastic anemia following exposure to benzene hexachloride (Lindane). *JAMA* 193:110-114, 1965.
8. Moore C: Exposure to insecticides, bone marrow failure, gastrointestinal bleeding, and uncontrollable infections (Clinico-Pathologic Conference). *Am J Med* 19:274-284, 1955.
9. Muirhead EE, Groves M, Guy R, Halden ER and Bass RK: Acquired hemolytic anemia, exposure to insecticides and positive Coombs test dependent on insecticide preparations. *Vox Sang* 4:277-292, 1959.
10. Registry on Blood Dyscrasias: Report to Council, *JAMA* 179: 888-890, 1962.
11. Sanchez-Mendal L, Castanedo JP and Garcia-Rojas F: Insecticides and aplastic anemia. *New Eng J. Med* 269:1365-1367, 1963.
12. Stieglitz R, Stobbe H and Schuettmann W: Knochenmarkschaden nach beruflicher einwirkung des insektizids gamma-hexachlorcyclohexan. *Acta Haemat* 38:337-350, 1967.
13. Williams DM, Lynch RE and Cartwright GE: Drug induced aplastic anemia. *Sem Hemat* 10:195-223, 1973.
14. Woodliff HF, Connor PM and Scopa J: Aplastic anemia associated with pesticides. *Med J Australia* 1:628-629, 1966.

15. Wright CS, Doan CA and Haynie HC: Agranulocytosis occurring after exposure to a DDT pyrethrum aerosol bomb. *Am J Med* 1: 562-567, 1946.
16. Davis KJ, Hansen W and Fitzhugh OG: Pathology report on mice fed aldrin, dieldrin, heptachlor or heptachlor epoxide for two years. FDA Memorandum, July 19, 1965.
17. Train RE: Pesticide products containing heptachlor or chlordane. *Fed Register* 39:41298-41300, 1974.
18. Epstein SS: The carcinogenicity of Dieldrin. Part I. *Sci Total Environ* 4:1-52, 1975.
19. Epstein SS: The carcinogenicity of Dieldrin. Part II. *Sci Total Environ* 4:205-217, 1975.
20. Schoental R, Hard GC and Gibbard S: Histopathology of renal lipomatous tumors in rats treated with the "natural" products pyrrolizidine alkaloids and A,B-unsaturated aldehydes. *J Natl Cancer Inst* 47:1037-1044, 1971.
21. Curley A, Copeland F and Kimbrough RD: Chlorinated hydrocarbon insecticides in organs of stillborn and blood of new born babies. *Arch Env Health* 19:628-632, 1969.
22. Infante PF and Newton Jr WA: Prenatal chlordane exposure and neuroblastoma. *New Eng J Med* 293:308, 1975.
23. Erslev AJ and Wintrobe MM: Detection and prevention of drug-induced blood dyscrasias. *JAMA* 181:114-119, 1962.
24. Huguley CM, Erslev AJ and Bergsagel DE: Drug-related blood dyscrasias. *JAMA* 177:23-26, 1961.
25. Scott JL, Cartwright JE and Wintrobe MM: Acquired aplastic anemia: An analysis of thirty-nine cases and review of the pertinent literature. *Medicine* 38:119-172, 1959.
26. Levin DL, Devesa SS, Godwin II JD, Silverman DT: How is cancer associated with other diseases or conditions? *Cancer Rates and Risks*, 2nd edition, 1974, p. 72.
27. Hirschman RJ, Shulman NR, Abuelo JG, Whang-Peng J: Chromosomal aberrations in two cases of inherited aplastic anemia with unusual clinical features. *Ann Int Med* 71:107-117, 1969.
28. Hecht F, McCaw BK, Koler RD: Ataxia-telangiectasia "Clonal Growth" of translocation lymphocytes. *New Eng J Med* 289:286-291, 1973.

29. Higurashi M, Conen PE: In-vitro chromosomal radiosensitivity in Fanconi's anemia. APS, SPR Program, 1971. p. 88.
30. Court-Brown WM and Doll R: Leukemia and aplastic anemia in patients irradiated for ankylosing spondylitis. MRC Spec Rep #295; H M Stationary Office, London, 1957. pp 1-135.
31. Cronkite EP: Radiation-induced aplastic anemia. Sem Hemat 4: 273-277, 1967.
32. Wald N: Leukemia in Hiroshima city atomic bomb survivors. Science 127:699-700, 1958.
33. Armenian HK and Lilienfeld AM: The distribution of incubation periods of neoplastic diseases. Am J Epid 99:92-100, 1974.
34. Awwaad S, Khalifa AS, Kamel K: Vacuolization of leukocytes and bone marrow aplasia due to chloramphenicol toxicity. Clin Pedia 14:499-506, 1975.
35. Brauer MJ and Dameshek W: Hypoplastic anemia and myeloblastic leukemia following chloramphenicol therapy. New Eng J Med 277: 1003-1005, 1967.
36. Cohen J and Greger WP: Acute myeloid leukemia following seven years of aplastic anemia induced by chloramphenicol. Am J Med 43:762-770, 1967.
37. Mukherji PS: Acute myeloblastic leukemia following chloramphenicol treatment. Brit Med J 1:1286-1287, 1957.
38. Yunis A: Chloramphenicol-induced bone marrow suppression, Sem Hemat 10:225-234, 1973.
39. Howell A, Andrews TM, Watts RWE: Bone marrow cells resistant to chloramphenicol in chloramphenicol-induced aplastic anemia. Lancet 1:65-69, 1975.
40. Vigliani EC and Gaiti G: Benzene and Leukemia. New Eng J Med 271:872-876, 1964.
41. Forni A and Moreo L: Cytogenetic studies in a case of benzene leukemia. Europ J Cancer 3:251-255, 1967.
42. Mohler DM and Leavell BS: Aplastic anemia; an analysis of 50 cases. Ann Int Med 49:326-362, 1958.
43. De Gowin RL: Benzene exposure and aplastic anemia followed by leukemia 15 years later. JAMA 185:748-751, 1963.
44. Williams MJ: Myeloblastic leukemia preceded by prolonged hematologic disorder. Blood 10:502-509, 1955.

45. Dameshek W: Riddle: What do aplastic anemia, paroxysmal nocturnal hemoglobinuria (PNH) and "hypoplastic" leukemia have in common? *Blood* 30:251-254, 1967.
46. Fraumeni JF: Bone marrow depression induced by chloramphenicol or phenylbutazone. *JAMA* 201:828-834, 1967.
47. Quagliani JM, Cartwright GE and Wintrobe MN: Paroxysmal nocturnal hemoglobinuria following drug-induced aplastic anemia. *Ann Int Med* 61:1045, 1964.
48. Stohlman Jr. F: Aplastic anemia. *Blood* 40:282-286, 1972.
49. Stevens H: Neurotoxicity of some common halogenated hydrocarbons In, Sunderman FW and Sunderman Jr FW eds *Laboratory Diagnosis of Diseases caused by Toxic Agents*. Warren H Green, Inc, St. Louis, 1970. pp 196-198.
50. Rejsek K and Rejskova M: Long term observation of chronic benzene poisoning. *Acta Med Scand* 152:71-78, 1955.
51. Delamore IW and Geary CG: Aplastic anemia, acute myeloblastic leukemia, and oxymetholone. *Brit Med J* 2:743-745, 1971.
52. Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health, Carcinogenicity of Pesticides. United States Department of Health, Education and Welfare, Government Printing Office, 1969. pp 459-506.
53. Maltoni C and Lefemine G: Carcinogenicity bioassays of vinyl chloride. Current results. *Ann NY Acad Sci* 246:195-218, 1975.
54. Princi F and Spurbeck JH: A study of workers exposed to the insecticides chlordane, aldrin, dieldrin. *Arch Ind Hyg Occup Med* 3:64-72, 1951.
55. Alvarez WC and Hyman S: Lack of toxic manifestations in workers exposed to chlordane. *Arch Ind Hyg Occup Med* 8: 480-483, 1953.
56. Fishbein WI, White JV and Isaacs JH: Survey of workers exposed to chlordane. *Ind Med Surg* 33:726-727, 1964.
57. Stein WJ and Hayes Jr WJ: Health Survey of pest control operators. *Ind Med Surg* 33:549-555, 1964.
58. Ambrose AM, Christensen HE, Robbins DJ and Rather LL: Toxicological and pharmacological studies on chlordane. *Arch Ind Hyg Occup Med* 7:197-210, 1953.
59. Dadey JL and Kammer AG: Chlordane intoxication, report of a case. *JAMA* 153:723-725, 1953.

60. Derbes JV, Dent JH, Forrest WW and Johnson MF: Fatal chlordane poisoning. JAMA 158:1367-1369, 1955.
61. Lensky P and Evants HL: Human poisoning by chlordane. JAMA 149: 1394-1395, 1952.
62. Starr Jr. HG, Aldrich FD, McDougall III WD and Mounce LM: Contribution of household dust to the human exposure to pesticides. Pestic Monitor 8:209-212, 1974.
63. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some organochlorine pesticides. International Agency for Research on Cancer, Lyon, France, vol 5, 1973.

TABLE 1
CASES OF NEUROBLASTOMA ASSOCIATED WITH
EXPOSURE TO CHLORDANE

<u>Case No.</u>	<u>Pathologic Condition</u>	<u>Sex</u>	<u>Age at Chlordane Exposure</u>	<u>Age At Diagnosis</u>	<u>Chlordane Use</u>
#1	Neuroblastoma (Stage III)	Female	1st Trimester of Pregnancy	2 yr. 8 mo.	Termite Infestation
#2	Neuroblastoma (Stage IV)	Male	1st Trimester of Pregnancy and every 6 months thereafter	4 yr. 5 mo.	Roach Infestation
#3	Neuroblastoma (Stage IV)	Female	1 yr. 11 mo.	4 yr. 4 mo.	Termite Infestation
#4	Neuroblastoma (Stage IV)	Male	2 yr. 5 mo.*	3 yr. 9 mo.	Termite Infestation
#5	Neuroblastoma (Stage IV)	Female	3 yr. 8 mo.	6 yr. 5 mo.	Ant Infestation

*House also treated with chlordane two years prior to child's birth.

TABLE 2

SUMMARY OF NEW CASES OF BLOOD DYSCRASIAS ASSOCIATED WITH
EXPOSURE TO CHLORDANE OR HEPTACHLOR, EITHER ALONE,
OR IN COMBINATION WITH OTHER AGENTS

<u>CASE NO.</u>	<u>AGE AT DIAGNOSIS</u>	<u>CHLORDANE OR HEPTACHLOR ALONE</u>	<u>CHLORDANE OR HEPTACHLOR WITH OTHER DRUGS</u>	<u>BLOOD DYSCRASIAS</u>
1	15 yr.	0	Isotox	Aplastic Anemia
2	28 yr.	0	Diazinon, various paints	Aplastic Anemia
3	68 yr.	0	Undetermined	Aplastic Anemia
4	9 yr.	1	None	Acute Stem Cell Leukemia
5	23 yr.	0	Banvel D Diazinon Dursban 2,4-D Paints, strippers, thinners	Acute Lymphoblastic Leukemia
6	37 yr.	1	None	Acute Myelo-Monocytic Leukemia
ALL CASES COMBINED		2	4	

TABLE 3

SUMMARY OF CASES OF BLOOD DYSCRASIAS REPORTED IN THE LITERATURE, ASSOCIATED WITH EXPOSURE TO CHLORDANE OR HEPTACHLOR, EITHER ALONE, OR IN COMBINATION WITH OTHER AGENTS

AUTHOR YEAR	TOTAL CASES	NUMBER OF CASES REPORTED			BLOOD DYSCRASIAS
		CHLORDANE OR HEPTACHLOR ALONE	CHLORDANE OR HEPTACHLOR WITH OTHER DRUGS UNSPECIFIED	SPECIFIED*	
Conley 1955	1	0	0	Sulfonamide	Hypoplastic Anemia
Conley 1955	1	0	0	Toxaphene Sulfonamide	Aplastic Anemia
Moore 1955	1	0	0	DDT and Lindane	Aplastic Anemia
Muirhead et al. 1959	1	0	0	Dieldrin Toxaphene	Hemolytic Anemia
Huguley et al. 1961	5	2	3	0	Aplastic Anemia
Loge 1965	3	3	0	0	Aplastic Anemia
Loge 1965	9	0	9	0	Aplastic Anemia
Loge 1965	2	0	2	0	Leukopenia Agranulo- Cytosis
Loge 1965	1	0	1	0	Thrombo- Cytopenia
Furie & Trubowitz 1976	1	1	0	0	Refractory Megalo- Blastic Anemia
Reports Combined	25	6	15	4	

*Specified Drugs Are Named.

TABLE 4

Number of Cases of Blood Dyscrasias Associated with Pesticides (Chlordane, Lindane, and DDT) Reported by the American Medical Association's Study Group on Blood Dyscrasias for the Years 1955-61.*

Blood Dyscrasias	EXPOSURE HISTORY			
	Pesticides	Pesticides "With Other Non-Toxic Drugs"	Pesticides "With Other Potentially Toxic Drugs"	Exposures Combined
Pancytopenia	10	19	11	40
Thrombocytopenia	8	2	0	10
Leukopenia	1	1	8	10
Dyscrasias Combined	19	22	19	60**

* Ref: Erslev and Wintrobe, 1962. In this report, exposure histories for chlordane grouped together so that it is not possible to determine the number of exposures for the individual pesticide.

** Because the time periods for the cases reported by Conley, 1955, Muirhead et al. 1959, and Huguley et al. 1961, (Table 3), overlap with the cases reported in Table 4, a maximum of eight cases shown in Table 3 might also be included in Table 4.

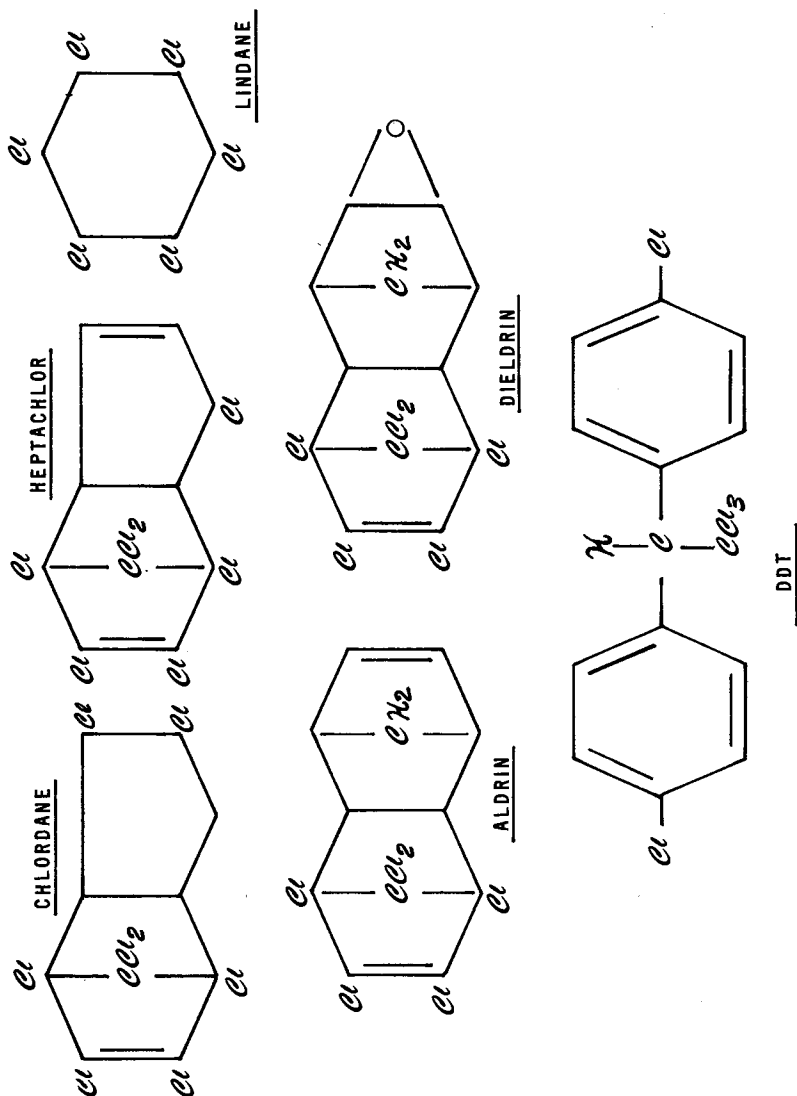


FIGURE LEGEND

Chemical structure of chlordane, heptachlor, similar chlorinated hydrocarbon pesticides.

CONGENITAL MINAMATA DISEASE:
METHYL-MERCURY POISONING AND BIRTH DEFECTS IN JAPAN

Aileen M. Smith
New York City

Minamata Disease is the first occurrence of widespread methyl-mercury poisoning caused by man-made environmental pollution that the world has experienced.

Minamata Disease was discovered in 1956 and its cause proved by 1959. Nevertheless, the present situation of the 100,000 persons who live within reach of the original mercury contamination of the environment is still basically unclear.

The term "disease" is not an accurate description of a condition that is actually a poisoning, but the term quite naturally entered popular usage before scientists were able to determine the origins of the condition and use of the term has persisted.

The Shiranui Sea is a quiet inland sea located in the western part of Japan's southern island, Kyushu. Minamata Bay is located on the eastern shore of the Shiranui Sea. Leading into the northeast depth of Minamata Bay is the drainage channel of the Chisso Corporation's Minamata factory. The mainstay of the economy of the city of Minamata (population 1959: 50,000 1976: 37,000) was and still continues to be the Chisso factory.

Unusual changes were detected in Minamata Bay as long ago as 1950. Fish floated on the surface of the sea, shellfish frequently perished, and some of the seaweed died. In 1952, some birds such as the crow and the amedori -- a type of sea bird -- began to drop into the sea while flying. The area of the sea where dead fish could be seen floating spread throughout the bay and out into the Shiranui Sea. Sometimes octopus and cuttlefish floated so weakened that children could catch them with their bare hands.

By 1953, not only cats but even some pigs and dogs went mad and died. The cats' "dancing disease" -- so called by the local inhabitants -- was particularly striking. Cats would stagger about as though drunk, salivating, convulsions would suddenly strike them or they would impulsively whirl in violent circles, often jumping into the sea (1).

However, fish continued to live in Minamata Bay, fishermen

Large portions of this report were taken from Minamata Disease; A Medical Report, by Masazumi Harada, M.D. and Aileen M. Smith. [From the book Minamata by W. Eugene Smith and Aileen Smith. Holt, Rinehart and Winston, 1975.]

continued to go out to catch them, and the people continued to eat them.

In April of 1956 a five-year, eleven-month-old girl entered the pediatrics department of the Chisso Corporation's Minamata factory hospital with brain-damage symptoms such as disturbance of gait, disturbance of speech and delirium. Within five weeks, her younger sister and four members of a neighbor family were found to be suffering from the same symptoms.

On May 1, 1956, Dr. Hajime Hosokawa, the head of the Chisso factory hospital, reported to the Minamata Public Health Department: "An unclarified disease of the central nervous system has broken out." This is the official "discovery" date of what is now called Minamata Disease.

An investigation quickly uncovered 30 cases.

By October, 1956, the cause was tracked down to some type of heavy metal poisoning from fish, and by October 1959 the causal element was identified as methyl-mercury from the waste water of Chisso's acetaldehyde and vinyl chloride plant. Fish and shellfish collected in Minamata Bay showed high mercury contents (5:61 - 39ppm) (2).

Unfortunately counter-measures were not taken: fishing in the bay was not banned nor the factory facilities causing the poisoning stopped. The number of patients continued to increase rapidly. By late 1956 there were 52 known victims and by the end of 1962 there were 121 officially verified Minamata Disease patients of whom 46 had died, at which point the outbreak was thought to be over.

CONGENITAL MINAMATA DISEASE

However, a major question still remained unanswered by the researchers. Many cases of congenital idiocy accompanied by various neurological symptoms had broken out on the coast of Minamata during the same period as the outbreak of Minamata Disease. From the beginning, it was suspected that these children might be cases of Minamata Disease (3). Since none of them had eaten the fish and shellfish of Minamata Bay, these children affected from birth were generally diagnosed as having cerebral palsy. In none of these cases, however, could there be found abnormal factors during the mother's pregnancy, at delivery, or in the postnatal period that might have caused cerebral palsy; the one common and notable factor was that the mothers of these children had all eaten a great deal of the fish and shellfish of Minamata Bay during their pregnancy.

Usually the mothers of these children began to notice that something was wrong when their babies had difficulty in holding

up their heads even after they were six months old. In some cases, tonic convulsion, failure of the eyes to follow, or weak response, attracted the mothers' attention to the abnormality of the infants.

The clinical characteristics of these children consist of serious mental retardation, primitive reflexes (such as grasping, opening the mouth and sucking), cerebellar symptoms of asynergy such as disturbance of coordination, ataxia, adiadochokinesis, dysmetria, intention tremor, dysarthria and nystagmus. Many cannot hold a sitting position, and disturbances in gait are observed even in lighter cases. The children also manifest disturbance of growth, akinesia, hypokinesia, hyperkinesia (chorea, athetosis, etc.) Rigidity and spasticity often coexist. Hypersalivation and strabismus are quite frequently observed. The children show character disorder (such as unfriendliness, apathy, abnormal shyness, irritability, sullenness, stubbornness, restlessness, fearfulness, and abnormal propensity to laugh or cry), and psychomotor seizure, loss of consciousness, myoclonic jerk, grand mal epilepsy, and also deformity of limbs, and pathological reflex (4). Deficiencies in growth and nutrition were observed in all cases in the early period, but there has been gradual improvement. One of the reasons for malnutrition is motor dysfunction such as disturbances of ingestion, mastication and swallowing.

The clinical picture of these patients is similar to certain symptoms in cases of exogenous idiocy, serious feeble-minded and physically handicapped infants, and cerebral palsy. Therefore, diagnosis is sometimes difficult. But when a comparison is made between the symptoms of the Minamata cases with those having similar diseases in other parts of Japan, the characteristic of the disease becomes clearer. Compared with congenital Minamata disease patients as a group, victims of exogenous idiocy show cerebellar symptoms or strabismus less frequently; pyramidal symptoms, palsy, differences in left and right sides, and factors causing cerebral dysfunction, are observed more often. Mental retardation is milder in the cerebral palsy group, cerebellar symptoms are milder, and chorea and athetosis are conspicuous.

Of the 220 births between 1955 and 1958 in the Tsukinoura, Detsuki, Yudo and Modo districts (the Minamata areas most affected by Minamata Disease), 13 such cases were found by 1962 -- a rate of 5.9%. This is much higher than the 0.2 - 0.3% rate of cerebral palsy cases in other areas of Japan.

A check was made on their families. In 1962, 64% of these children had some member of their family who had typical acute Minamata Disease. All the mothers of these patients were thought to be healthy at the time. However, when carefully observed, certain neurological symptoms were present in 73% of the mothers: for example, ataxia, adiadochokinesis, nystagmus, dysarthria, sensory disturbance. Ten years later, these symptoms were observed to have increased.

The mercury content in the hair of these children who were born between 1955 and 1958 was still high in 1961. For example, one child's hair registered 100 ppm. The mercury content in the hair of the mothers who had given birth to these children was also as high as 191.0 ppm and 172.9 ppm in some cases (5). The preserved umbilical cords of these children had higher methyl-mercury contents than those of other children (6).

Then in 1961 and 1962 two of these children died. The autopsies gave significant findings. Typical pathological findings found in methyl-mercury poisoning cases (such as granular cellular atrophy in the cerebellum) were observed. Also microcephalia, general hypoplasia of the medullary substance (subcortex), hypoplasia of the corpus callosum and hypoplasia of the pyramidal tract were found, thereby showing that damage occurred in the early fetal stages (7).

The clinical, epidemiological, and pathological findings indicated that methyl mercury had passed through the placenta from the mother to the fetus, thereby causing methyl-mercury poisoning. This was diagnosed as congenital (fetal) Minamata Disease in 1962. At present (1976), 40 such congenital cases have been found.

Furthermore, autoradiographical experiments on pregnant animals and histological study of their embryos later showed that methyl mercury passed through the placenta and caused damage to the central nervous system in the embryos (8).

The methyl-mercury content in the milk of mothers who have been contaminated by methyl mercury has been proven to be high. Since the ingestion of milk containing methyl-mercury causes poisoning, it cannot be denied that congenital cases may also have been affected by their mothers' milk after birth.

TREATMENT

Treatment for these children is severely limited and has basically been rehabilitative. To be of any help at all, rehabilitative treatment must be done on a case-by-case basis. This form of treatment has had some good results on light cases, but is limited in its effectiveness. Although their symptoms were alike when the children were discovered, they have changed in the course of more than ten years. There has been some improvement of ataxia, primitive reflex and disturbance of intelligence. Symptoms most difficult to overcome are strabismus and dysarthria. When motor functions improve, mental retardation becomes the main symptom.

AWAKENING TO THE "NON-CONCLUSION" OF MINAMATA DISEASE

During the years 1962 to 1970 it was generally believed that Minamata Disease had been "concluded". It was believed that the number of patients was 121, that they had fallen ill between the

years 1953-1960, and that the outbreak had occurred in the 50 kilometer area north and south along the coast of Minamata (9). Medically, up to then, the only cases diagnosed as Minamata Disease were acute, severe cases and cases which showed the typical methyl-mercury symptoms (reported by Hunter and Russell in England, 1940).

In 1970, surveys were begun again by individual medical researchers (10). As a result, it gradually became clear that the influence of methyl mercury on the population in general was much greater than was assumed before. Also, women who had had miscarriages and stillbirths were found to have relatively lighter symptoms than other members of their families (11); it was suggested that methyl mercury was discharged from their bodies through the placenta and into the fetus.

Since family members of the patients with typical Minamata Disease had been eating the same contaminated fish, they were examined first. It was found that 84% of these individuals had some symptom connected with Minamata Disease and 55% had some neuropsychiatric disturbance in their daily life (12).

Health surveys conducted by the Second Kumamoto University Research Group in the most severely contaminated Minamata areas showed that the neurological symptoms among the inhabitants of this area are clearly more common than they are in a control group (13).

Moreover, the survey showed that the appearance of symptoms was not limited to the 1953-1960 period as was thought before.

SCHEMATIC VIEW OF MINAMATA DISEASE (14)

Since Minamata Disease is a contamination by methyl-mercury of the entire population through environmental pollution, gradations from serious cases to apparently healthy cases should be perceived as a continuous spectrum. Epidemiological data indicate that the effects of methyl mercury on the human body vary according to the degree of contamination.

In the schematic view of congenital Minamata Disease, if the mother's methyl-mercury intake is so great that she falls acutely ill with Minamata Disease, she does not become pregnant. If the dosage is somewhat less, the woman becomes pregnant but the child is spontaneously aborted or is born dead. If the dosage is even less, a child with congenital Minamata Disease, accompanied by severe neurological symptoms, is born. Even in such cases, the mother's own symptoms may be relatively light. If the mother's mercury dosage is even less, there is a chance that the child -- even with no remarkable neurological symptoms -- may be mentally deficient. In such cases the mother may have almost no neurological symptoms. It would be difficult to differentiate the symptoms of these children from mental deficiency due to a different cause.

As a matter of fact, in the most heavily contaminated Minamata area of Tsukinoura, Yudo and Modo, mental deficiency among children born between 1955-1959 was 29% (1962). This figure excludes the already recognized congenital cases. It is astonishingly higher than in the control area (15).

Moreover, in 1970 an examination of junior high school students in this contaminated area was made, and 223 children born between April of 1955 and March of 1958 were examined. These children are attending regular public school classes and do not include the already recognized congenital cases. The following observations were made. Mental deficiency was found among 18%, sensory disturbance among 21%, clumsy speech among 12%, and clumsy movements among 9%. These examinations were conducted by qualified neuropsychiatrists directly examining the children. These rates are higher than in any other part of Japan examined (16). (Mental deficiency among junior high school children is considered to be 9.7% in Japan).

SAFETY LEVEL

The present "safety" level of total mercury in fish is 0.4 ppm in the U.S.A. and Japan (17). This level was determined on the basis that it was "safe" by a factor of ten, that an adult male weighing 50 kg. and consuming an average amount of fish will not get Minamata Disease by eating fish with less than 0.4 ppm mercury.

The definition used of Minamata Disease was the acute type originally found in Minamata. Also, the safety level determinants ignore the biological half-life of methyl mercury in the brain (which is 230 days), but use the half-life of methyl mercury in the blood instead (which is 70 days).

When considering the safety level one must consider not the average but the exception. The safety level should be calculated to definitely avoid even the lightest of symptoms. It should be calculated to be safe for those consuming more than the average amount of fish and shellfish. Also, the calculation should consider the pregnant woman, the young and the weak as well as the healthy adult.

It must also be noted that there are no experiments as yet to prove how safe the presently calculated safety level is when amounts just on the so-called safe side are consumed continuously over a period of many years.

And finally, in this age of multiple pollutants in the environment, this safety standard has not considered the possibilities of multiple contamination: mercury from other sources and also the cumulative effects of other poisons in the environment.

Therefore, when these various factors are considered, it be-

comes clear that it is necessary to seriously re-examine the present approach in figuring a safety level.

CONCLUSION

The schematic views of Minamata Disease and congenital Minamata Disease indicate that there are types and levels of methyl-mercury poisoning that are as yet undefinable, undetectable, or unknown in present medical practice. In Minamata the levels of poisoning were so great that the heavier dosages of contamination could be readily detected. It is quite possible, however, that in other areas with lower levels of contamination, symptoms may go undetected as methyl-mercury poisoning. The world-wide implications of the possible effects of long-term, low-level poisoning become self-evident.

In the case of Minamata, there are approximately 100,000 inhabitants living on or near the shores of the Shiranui Sea. One can only speculate on how many have been affected by this mercury contamination.

At this point (June 1976), there are 880 officially verified patients, of whom 107 have died. Over 3,000 more are applying for verification.

REFERENCES

1. Kitamura, S., et al., Journ. Kumamoto Med. Soc. 31 (Suppl. 2) p. 238 (1957), in Japanese.
2. Minamata Disease, Minamata Disease Study Group, Kumamoto University, Japan, 1968.
3. Kitamura, S., et al., Journ. Kumamoto Med. Soc. 34 (suppl. 3) p. 477 (1960), Nagano, S., et al., Journ. Kumamoto Med. Soc. 31 (suup. 3) p. 511 (1960), 34 (suup. 3) p. 511 (1960), in Japanese.
4. Harada, M., Psychiat. Neurol. Japan, 66, p. 429 (1964); Harada, M., Constitutional Medicine, 38, p. 29 (1974).
5. Harada, Y., Minamata Disease, Minamata Disease Study Group, Kumamoto University, Japan, 1968, pp. 93-117.
6. Fujiki, M. op. cit.; Harada, M., Advances in Neurol. Sci. 16, p. 870 (1972), in Japanese. It is an old custom in Japan to keep the dried umbilical cords of newborn infants.
7. Takeuchi, T., Minamata Disease, Minamata Disease Study Group, Kumamoto University, Japan, 1968, pp. 141-228; Matsumoto, H., et al., Journ. Neuropath. Experiment. Neurol., 24, p. 563 (1965); Takeuchi, T., International Congress on Environmental Mercury Contamination (1970), pp. 247-301, Ann Arbor Sci. Publ. Inc. (1972).
8. Morikawa, N., Kumamoto Medical Journal, 14, p. 87 (1961); Fujita, E., Journ. Kumamoto Med. Soc., 43, p. 47 (1969) in Japanese; Shiraki, H., Journal of Kyogyo Chem., 17, p. 93 (1972).
9. Minamata Disease, Minamata Disease Study Group, Kumamoto University, Japan, 1968.
10. Harada, M., Science, 41, p. 250 (1972), in Japanese.
11. Harada, M., Constitutional Med., 38, p. 29 (1974)
12. Harada, M., "Advances in Neurol. Sci.", 16, p. 870 (1972) in Japanese; Harada, M., Constitutional Med., 38, p. 20 (1974).
13. Epidemiological, Clinical and Pathological Studies on Minamata Disease Ten Years After Outbreak, a report of the Second Medical Study Group, Kumamoto University, Japan (1973) in Japanese.
14. Harada, M., Journ. of Public Health, 37, p. 171 (1973) in Japanese.
15. Harada, M., Psychiat. Neurol. Japan, 66, p. 429 (1964).

16. Ibid.

17. Ekmann, L., Nord. Med., 79, p. 450 (1968).

DISCUSSION

Dr. Charles Shaw, Professor of Biology
University of Texas
M.D. Anderson Hospital and Tumor Institute

Dr. Umberto Saffiotti, Chief, Experimental Pathology Branch
Carcinogenesis Program
National Cancer Institute

Dr. Eldon Sutton, Vice President for Research
University of Texas at Austin

DR. SHAW: I would just make a couple of brief points. First, to try to inject a note of perspective into this presentation and to the whole meeting. As a person who works in carcinogenesis and the chemical causes of cancer, I've been asked by a number of laymen, in light of all of the horror stories that we read about, whether it is true that everything causes cancer? I even had one man say that he'd been told that water causes cancer, if you take enough of it. Well, the answer is definitely not. Most chemicals, we feel, do not cause cancer, do not cause mutations and do not cause congenital malformations.

The problem is, how do we know which ones are safe? I think Dr. Warkany and I would certainly continue to eat table salt without any concern. I think Dr. Legator has made the important point that it is difficult to prove the safety of any substance. Certainly, if you run a compound through a test and get a negative result, that's by no means an indication that it's safe.

It's easy to prove that a substance is hazardous, or potentially hazardous, but when you try to prove that it is safe, absolutely safe, under all concentrations, I think most of the scientists here would agree that there's really no way you can come up with such a definite conclusion, what we call proving the null hypothesis.

One more brief point: I think we're really talking here about the differences between men and women. I think there are differences. As a geneticist, I recognize that women are different from men, especially in that they have 2 X chromosomes. I think this whole meeting is a laudable effort to bring into open and free discussion the fact that there are such differences: that such differences are carried into the workplace; and that women and men, as two different populations, may be differentially at risk to certain conditions.

As one whose main interest is the study of metabolism of chemical carcinogens, I am interested in differences in such metabolism between men and women, as well as among all men and women.

Most of the compounds we're talking about here this morning, most of these horrible chemicals that do such terrible things to the body and to the offspring, are metabolized in the body. Most of them are converted to the active forms, which is called "activation". Dr Legator alluded to this in some of the tests.

Some of them are then further metabolized beyond the active forms to the inactive or detoxified forms. We know from a number of studies that people vary a great deal in the amounts of enzymes that they contain, which activate and then inactivate these compounds. So I think it's important that we look at this aspect of the question in studying the differences between men and women, as well as any other groups.

Thank you.

DR. SAFFIOTTI: I would like to address a few comments to the general topics of the discussion today. The first part of the session was designated under the general heading of Laboratory Approaches. My field is particularly that of carcinogenesis. And we have had very limited experience up to a few years ago, on this particular area of transplacental effects of carcinogenesis. In the last decade, a great deal of additional work has developed in this area.

We are still really just beginning to learn the various conditions of exposure that are linked to a transplacental effect. One of the important challenges in carcinogenesis studies is that of trying to correlate the experience gained by experimental studies in animal systems with the human experience.

We are seeing, now, a number of instances -- the case described here today of vaginal adenocarcinoma due to DES exposure is a good example -- where a well-known experimental carcinogen has been confirmed to be active in the human species. What we want, obviously, is to relate the experience obtained from laboratory studies with that obtained from human observations.

The National Cancer Institute's carcinogenesis program, which I've been privileged to direct for about eight years, until recently, has developed a considerable effort in the area of transplacental carcinogenesis studies, particularly under the leadership of Dr. Jerry Rice, whom Dr. McLachlan has quoted. Dr. Rice recently organized and chaired a conference on perinatal carcinogenesis, which was held in Tampa, January 19-21, 1976. At this conference, the current knowledge of transplacental carcinogenesis was reviewed.

In considering carcinogenesis in women, one must take into account the specific role of the woman as mother, because of transplacental exposures. In addition, we should not forget the role of the woman as a member of the population having certain special

physiological characteristics and susceptibilities. The point that Dr. Shaw mentioned -- the study of how environmental chemicals really react in the body to affect the target tissues -- is one that is beginning to be more extensively studied. And this is very important in trying to identify exactly the levels of susceptibility of different members of a population to a given type of exposure.

We know that there are a number of conditions that are peculiar to each of the two sexes in their response to certain types of environmental chemicals, and particularly carcinogens. Some will be characterized by sex-related target tissues. For example, there are the interesting observations of Dr. Albert Segaloff on breast cancer in experimental systems in which exposure to x-radiation, combined with exposure to hormonal carcinogens, shows a remarkable enhancement of the induction of breast cancer.

The approach that the laboratory can perhaps best contribute to all this is to bridge these gaps between different levels of observation. My main philosophical approach to our research has been to develop good documentation at different levels of observation that could be interrelated.

We now have models that have been developed, particularly in the last decade, for the induction of cancer in specific organs in various animals. For example, better models for the induction of breast cancer in animals have been developed by Dr. P. Gullino of the NCI. And Dr. Bela Toth of the University of Nebraska has reported a very high instance of ovarian cancers induced in animals by specific chemical exposure to hydrazines.

One of the major thrusts of our work has been that of developing organ-culture and cell-culture studies for specific target tissues, particularly epithelial tissues, which are the main targets of carcinogenesis in the human population. We have now begun to develop what looks to me like an extremely promising methodology for applying these culture techniques to human tissues. We cannot do carcinogenesis studies on living human beings, other than what, unfortunately, people do to themselves by working in hazardous operations, by smoking, and by a variety of other factors. But we can take human tissues and cultivate them in vitro, and in appropriate culture conditions, we can now study the binding, the metabolic requirements, and the interaction of environment chemicals directly with the human target tissues in vitro.

Dr. Curtis Harris and his colleagues in our laboratory have been working with Dr. Benjamin Trump and his colleagues at the University of Maryland to develop some very exciting techniques that enable us to study the effect of carcinogens directly on human target tissues such as the bronchi, the large intestine, and other organs. All these will give us tools that will enable us to pin down more precisely the interaction of environmental chemicals with target tissues that relates to people. Is that very far from

the problem of occupational risk? I don't think it is.

If we learn how to identify early reactions, the way carcinogens link to the target tissues of individuals, and if we learn to identify those individuals who are at particularly high risk for a variety of congenital or acquired reasons, then we can be much more selective in our prevention. And while continuing to strive for the elimination of carcinogens that we have identified, we can also put a lot of effort into selective protection of high-risk individuals in a population.

DR. SUTTON: I would like to elaborate a bit on some ideas put forward by the previous two discussants, concerning human sensitivity. In terms of mutagenesis, one perhaps should distinguish two types of mutagenesis; that which affects the somatic cells (non-germ cells) of the person exposed and, therefore, according to some theories, may lead to malignancy; and that mutagenesis which occurs in the germ cells of the person and, therefore, would be transmitted to future generations.

And, of course, one should also consider under somatic mutation, any mutations induced in a fetus exposed in utero. These present somewhat different ethical problems for us, since we may feel quite capable of taking the responsibility of our own somatic mutations and taking them to the grave with us, as opposed to the mutations that would be burdens for future generations.

This is a problem that we have not learned to cope with effectively. We are now on a pendulum-swing that says each person should be able to make decisions about himself or herself and yet we permit, at the same time, individuals to make decisions that affect later generations.

Another, let's say, influence of heredity, or genetics, is mutagenesis, per se. That is, the induction of new mutations, as opposed to the inherited sensitivity to environmental agents, whether those agents cause mutation, or whether they are directly responsible for somatic effects.

And I should especially like to consider the variation among individuals in the population, and their sensitivity to environmental agents. This is not a new concept, of course. We recognize that certain persons, because of their skin pigmentation, may be especially susceptible to sunlight.

Some people enjoy a tan; others suffer from reddening of the skin. And such persons may, indeed, have a high propensity to develop skin cancers. Other examples: we now cite as our classical example in which sensitivity and mutagenesis are tied together, the disease of xeroderma pigmentosum. Where there is a defect in mutagenesis repair, such persons are unusually sensitive to ultraviolet light and, as a result of this, have a high risk of induction

of mutation from ultraviolet light.

We know very little about repair systems in mammals. We know that there are many others, and one would expect that the sensitivity of certain persons to environmental mutagenesis must be very much higher than of other persons.

Mutation, of course, is not the only end point that is measured. Other examples of sensitivity would include G6PD deficiency; that is, glucose six phosphate dehydrogenase, where persons with this X-linked recessive gene are sensitive to a variety of chemical agents, some of which are commonly used drugs.

Since it is an X-linked trait, this, of course, makes the males more sensitive or, if you wish to look at the other side of the coin as I'm sure some of you do at this conference, it makes the females more resistant. So, perhaps, thinking in terms of industrial exposures, one should consider 2 X chromosomes are superior to 1, for that purpose only, of course.

We may need to move to -- or, let's say, more generally accept -- a concept that geneticists are using. That concept is genetically significant exposure, in which risk to the radiation and other environmental mutagens, in terms of mutation in future generations, is assessed in terms of the likelihood that the exposure will cause such mutations. This is not just a chemical problem. A genetically significant exposure reflects the amount of reproductive period left for each person. In addition to age, of course, it would vary upon whether one is male or female, and it would also depend upon how much of the exposure actually occurs in the germ cells.

In the case of radiation, this has been applied most vigorously. I might just inject a footnote here that induction of mutations in female mice by radiation is apparently very low -- so low, in fact, that in the most recent National Academy Report on the risk of radiation, it was assumed that only human males are sensitive and females are resistant. This, of course, is germinal mutation and not somatic mutation. Some of these concepts leave us with a few problems we're going to have to face, not just as scientists but as citizens. How do we identify and protect persons who are especially sensitive to environmental agents?

Some of the sensitivity may, indeed, be associated with sex, either male or female, but certainly much of it is associated with individuals. And, after all, our ultimate goal is to be recognized as individuals, and not simply as one of two major categories.

How do we identify and protect risk to the future generations? Here we have to keep in mind that a fetus and its germ cell, because of its own separate genetic identity, may be relatively more or less sensitive compared to the parents.

And, finally, how do we achieve equality of opportunity for persons who are biologically unequal? I think this is a very difficult matter. I, of course, have already given examples of biological inequality. We do not wish that special problems prevent some individuals from entering into full participation in our economic benefits and social benefits, but having recognized such problems, what do we do about the situation?

These are some issues that, I think, must be resolved. Unfortunately, I have no solutions to them.

QUESTIONS, ANSWERS AND COMMENTS

MR. HARRY SLATON: I am a consultant in industrial hygiene. My company is Belair. I have some brief experience with diethylstilbestrol. Most, if not all, diethylstilbestrol made in this country was made by Lilly. The original plant was at their home base in Indianapolis.

After a few years, the men operating the plant developed secondary sexual characteristics of females, which was an undesirable thing. And so, they moved their plant and constructed a new plant in Omaha, Nebraska, which was set up as a clean plant. And up until a few years ago, that plant was still operating and under the new conditions, they got no sex changes, as it were, in their personnel.

But the chemical is a very powerful chemical. It is shipped in bags. The bags are broken, and they are opened. The chemical was used mainly in animal feed. And the way you make animal feed is as follows: you get a great big mixer and you put in so many pounds of this and so many pounds of that, and you twirl it back and forth. Naturally, the material dusts all over the place. So, there is still a possibility that DES may get around to other people.

MR. WALTER LEAR: I'm with the Pennsylvania Department of Health. I wanted to ask Dr. Infante about the practical consequences of the many people that are using chlordane in insect control work, lawn work, and so forth. Is that being studied or is anything being done about that?

DR. INFANTE: One of the points of my presentation was that there have not been any epidemiological reports which would allow us to preclude the absence of neuroblastic risks from chlordane. And I realize there are many people that have to work with it and that there just simply isn't any human data available from which I could draw that conclusion.

I think Dr. Legator made the point this morning that there is concern about concluding from inadequate testing or assessments that there is no risk involved. I think this has been the problem

with chlordane; for example, there have been three, quote, studies that have been done in humans exposed to chlordane with the sample sizes ranging from 12 to 36 employees.

And when you're assessing diseases like acute leukemia -- or just leukemia which, you know, only occurs in the population at a rate of about 8 per 100,000 persons -- I doubt you could detect it even if the risk were in the order of 100-fold.

DR. JANETTE SHERMAN: I have a two part question for Dr. Infante. Since we can buy chlordane, heptachlor and other pesticides in the supermarket the same -- one aisle over from where we buy our food -- and since, I understand, about 60 percent of pesticide use is in the home, and since people tend to believe that if a little bit works then a lot works even better, what do you suggest we do about limiting access to these deadly chemicals?

And the second question is this: in the last month I've had two patients who have insecticide-induced malignancy, and have two more that I suspect very highly. Where do I go to get help? Where do I report these cases so that there is some central registry? And how do I get further investigation of the patients that I suspect?

DR. INFANTE: They're some very profound questions and, of course, I don't have any profound answers.

In regard to the latter, where do you report them is exactly the problem. The AMA study group on blood dyscrasias set up their registry after the epidemic of chloramphenicol induced aplastic anemia. But, this was voluntary reporting, and this mechanism no longer exists. One of the problems is that the busy practitioner has no place to report these cases, and they are not being reported in the literature or anywhere else. So I am just not really capable of answering your question.

In regard to people buying pesticides, like chlordane, over the counter, it has been suspended for agricultural and I understand also for home use. However, it's still going to be permitted for termite infestation. And also, you can still go to just about any hardware store today and buy chlordane because they are allowed to use up the existing supplies.

And one of the problems with people using this around the home is that they don't have any respect for the hazards involved. In fact, I think the label says: "Warning, Keep Out of the Reach of Children".

Perhaps there is someone from the Environmental Protection Agency here who might like to address this question and answer it.

MR. KEVIN LEE: I am with the Environmental Protection Agency. In

answer to parts of the question about the reporting, I think that it might be helpful if it were reported to the Environmental Protection Agency, OPP, Office of Pesticides. Unfortunately, we don't have a program at this time, but we'd like to get one going.

As to the availability of chlordane, it has been suspended for use around the home with the exception of the termiticide use. And it is currently being looked at, in administrative hearings, for cancellation.

This hearing is going to take place this summer. That's as much as I can tell you.

DR. HECTOR BLEJER: I have a partial answer to Dr. Sherman's question. And, parochially, it's about California, which is the only state in the Union I know of that has a system of mandatory reporting of pesticide-induced illnesses, whether official or not. They are reported to the local health department and also the Departments of Health and Agriculture and the Division of Industrial Safety.

Now, obviously, this doesn't apply to the 49 other states nor to the federal government. But, maybe Dr. Sherman should write to her Congresspeople and see what actions she can get for the state of Michigan, and, similarly, for the rest of the nation, since I doubt that a national reporting mechanism would ever work.

One other DES item here, if I may. Lately, there was a report from Kansas concerning cattle feeding bunkers. Two brothers who worked in construction were demolishing some of these old bunkers. Their clothes became contaminated with the DES-impregnated feed. They went home and two, I believe, of the children of each of the workers developed, at a very early age -- they were five, six, seven, thereabouts -- premature, secondary sexual characteristics as a result of the parents' occupation and the transmission of the DES in the parents' clothing. So, there are many other ways of being exposed to DES.

SESSION II

BIRTH DEFECTS, CANCER, AND MISCARRIAGES ASSOCIATED WITH ANESTHETIC GASES, VINYL CHLORIDE, AND OTHER INDUSTRIAL CHEMICALS

MODERATOR: Dr. John Finklea
Director, National Institute for
Occupational Safety and Health

DR. FINKLEA: I think I have a close relationship with all of the people on the panel this afternoon. I know we are all very pleased that there's such great interest in this topic. We feel this is one of the more important topics in occupational health and it will be one that we'll be dealing with for a number of years.

The papers and discussions this afternoon are on specific important social problems and technical problems that illustrate much larger problems. We're going to be talking about anesthetic gases and vinyl chloride monomer. But, I think over the next few months -- the next year or so -- you'll see similar questions about health risks arising from a number of important halogenated compounds that are related chemically and structurally to some of the compounds we're going to discuss. These include perchloroethylene in the dry cleaning industry, and epichlorohydrin in the chemical industry. And they will include such solvents as trichlorethylene and its substitute methyl chloroform.

So, we have an important series of problems and compounds to address. They are illustrative of the sorts of questions we'll be asking about related substances.

As we move through the afternoon, you'll hear different views expressed by labor, industry, the legislative community, the academic community, and government. But I want to emphasize before we begin these discussions that all of us in this room probably share four major social goals. First of all, we'd like to have jobs for workers. Secondly, we all believe in equal employment opportunity for all workers. In the third place, I think we all are very much committed to public health and to a proper health heritage for our children. And, finally, I think all of us are concerned about protecting individual liberty and free choice, even if some things might not be the best for your public health.

We are, in a sense, a real consensus. That which unites us is much deeper and much more dear than any divisions we may have. I would hope that before the conference ends we will have a lot of heated discussions, but I also hope all of us remember that only

by working together in a national consensus to assure equal employment opportunity and the protection of public health can we get these problems solved.

So, I have taken the liberty of making a few remarks and I'll now set the clock for our initial speaker of the afternoon, Dr. Thomas H. Corbett.

CANCER, MISCARRIAGES AND BIRTH DEFECTS ASSOCIATED WITH OPERATING ROOM EXPOSURE

Thomas H. Corbett, M.D.,
Clinical Investigator, USVA Hospital
Ann Arbor, Michigan;
Associate Professor of Anesthesiology,
University of Michigan Medical Center,
Ann Arbor, Michigan

Inhalation anesthesia was first administered in 1842 by a Dr. Crawford Long of Jefferson County, Georgia. At that time, he anesthetized a friend with diethyl ether and removed a tumor from his neck. He was later run out of town by the local townsfolk who thought he was in league with the devil.

Since that time, we've come a long way in many respects, with regard to inhalation anesthesia, but in other respects, we haven't come quite so far.

It has been estimated that approximately twenty million anesthetics are administered to patients each year in the twenty-five thousand operating rooms throughout the United States. Approximately fifty thousand operating room personnel are exposed daily to low concentrations of anesthetic gases which permeate the operating room throughout the administration of inhalation anesthesia. This figure does not include surgeons, who usually do not operate every day. The following table lists the number of operating room workers in the United States by professional group:

TABLE I

<u>Professional Group:</u>	<u>Membership (1973)</u>	<u># Women</u>
American Society of Anesthesiologists	11,192	1,399
American Association of Nurse Anesthetists	14,594	11,967
Associations of O.R. Nurses & Technicians (combined)	23,799	<u>22,133</u> 35,499

Epidemiologic observations first drew attention to anesthetic gases as possible environmental hazards to operating room personnel in 1967, when Vaisman reported an unusually high incidence of headache, fatigability, nausea, and pruritus among 354 Russian anesthesiologists. He also noted that 18 of 31 pregnancies reported ended in spontaneous abortion. In addition, two of the pregnancies ended in premature delivery and one in a congenital malformation.

In 1968, Bruce, et al., published a study of the cause of death among 441 members of the American Society of Anesthesiologists who had died between 1947 and 1966. In addition to an inordinately high suicide rate, there was a high incidence of reticuloendothelial and lymphoid malignancies.

A germane study by Li, et al., in 1969 found a significantly higher rate of cancer among chemists compared to professional men in general. Nearly half the excess cancer deaths were due to malignant lymphomas and cancer of the pancreas. The relevance, if any, is that both chemists and anesthesiologists are exposed chronically to low concentrations of volatile chemicals and gases in the course of their work.

Further studies from Denmark, California, and Great Britain all demonstrated a high incidence of spontaneous miscarriage of pregnancy among operating room nurses. In 1972, we conducted our own survey of the 621 female nurse anesthetists in the state of Michigan, obtaining a response rate of 85%. A total of 33 malignancies in 31 nurse anesthetists was reported. Several unusual types were noted, including hepatocellular carcinoma, malignant thymoma, malignant melanoma, and leiomyosarcoma. Ten malignancies, including skin cancers, were diagnosed during 1971. Excluding skin cancers, the expected incidence adjusted for age distribution, based on statistics from the Connecticut Tumor Registry, was 403/100,000. The age-adjusted incidence among the Michigan nurse anesthetists was 1333/100,000, or three times the expected rate. This difference was significant at the 3.1% level. Analysis of the data from this survey regarding congenital abnormalities among children born to nurse anesthetists revealed a significantly higher incidence of anomalies in children born to mothers who administered anesthesia during the pregnancy. In the same study, exploring the possibility of transplacental carcinogenesis, we found that three neoplasms had occurred in two of the 434 children whose mothers had practiced during pregnancy. One had a neuroblastoma at birth and later developed a thyroid malignancy at puberty. Another child developed a parotid tumor at 22. Among the 261 children whose mothers did not work while pregnant, there was one case of leukemia, at age three. The figures are not statistically significant, but neither are they reassuring.

All the above studies were of small population groups and the need for a nation-wide study of large numbers of operating room personnel became apparent. This study was undertaken by the American Society of Anesthesiologists with the financial support of the National Institute for Occupational Safety and Health (NIOSH). Fifty thousand operating room professionals were surveyed, with 24,000 unexposed medical and nursing professionals serving as controls. The study found 1.3-2 times the incidence of spontaneous abortion in exposed, compared with unexposed, female personnel. Women physician-anesthetists suffered the highest risk, followed by nurse anesthetists. The incidence of congenital abnormalities

among live-born offspring of exposed female physician-anesthetists was double that among offspring of unexposed female physicians. Among exposed nurse anesthetists, the risk of fetal abnormalities was 1.6 times that of their unexposed counterparts. There was also a 25% increase in the risk of congenital abnormalities in children of unexposed wives of male anesthesiologists.

The frequency of cancer was 1.3-2 times greater among exposed women, the highest risk again among the women physician-anesthetists, followed by nurse anesthetists.

Liver disease, excluding serum hepatitis, was found from 1.3-2.2 times more frequently among exposed women than among unexposed counterparts, the highest incidence being among the anesthesia personnel. A similar increase in liver disease was seen among male physician-anesthetists compared with male pediatricians.

None of the above studies has established a cause-and-effect relationship. It has been argued that causative factors could include long hours and tension of the operating room, exposure to radiation from X-rays and radium implant procedures, and exposure to patients with transmissible viruses. However, it would require an extraordinarily recalcitrant bias not to consider the chronic exposure to low concentrations of anesthetic gases to be responsible for the excess of incidence of cancer and birth defects seen among operating room personnel.

It is now estimated that 80% of all human cancers are caused by chemicals. The remainder are thought due to radiation, viruses and other factors. Man and other animals are susceptible to the carcinogenic effects of a wide variety of chemicals, both man-made and naturally-occurring. There are two stages in the production of cancer by chemicals. The first is initiation, during which the carcinogen reacts with the various cell constituents including DNA, RNA, and proteins in many different cell organelles. Initiation may take a relatively short time to occur, usually hours or days. The second stage, promotion, requires considerably more time, usually years for the human species. It is during this stage that the changes from the altered cell constituents manifest themselves and eventually, after many cell divisions, result in malignant transformation.

Most inhalation anesthetics are halogenated hydrocarbons or halogenated ethers. A number of related chemicals are known carcinogens in man and/or animals.

TABLE II

HALOGENATED HYDROCARBONSHALOGENATED ETHERSanesthetics carcinogens

chloroform
 trichloro-
 ethylene
 halothane
 ethylene
 dichloride
 ethylene dibro-
 mide
 DDT
 Aldrin
 Dieldrin
 Heptachlor
 Chlordane
 PCB
 Benzene hexa-
 chloride

anestheticscarcinogens

Isoflurane
 Englurane
 Methoxyflurane
 Bis (chloromethyl)
 ether
 Chloromethyl
 methyl ether
 Bis (B-chloroethyl)
 ether

It is both interesting and disconcerting to know that vinyl chloride, a known human carcinogen, was once considered for use as an anesthetic agent, but was discarded because of its myocardial irritant properties. Perhaps even more disquieting, however, is the structural similarity between the anesthetic trichloroethylene and vinyl chloride. TCE has enjoyed popularity among anesthesiologists in the past, but is used only occasionally at the present time.

The carcinogenicity of chloroform in animals has already been demonstrated. Preliminary carcinogenicity testing with trichloroethylene and isoflurane resulted in data categorized as "highly suspicious" for both chemicals.

Embryolethality and teratogenicity of anesthetic doses of halothane, nitrous oxide, and other inhalation anesthetics were reported in laboratory animals during the 1960s. Whether these effects occur in laboratory animals from occupational exposure levels is unknown, but the question is under study in several laboratories. However, the fact that embryo-lethality and teratogenicity have been demonstrated in animals administered anesthetic doses of these chemicals is cause for concern. Many chemicals known to cause birth defects or cancer react differently in different animals, and humans may be more or less sensitive to a particular chemical than the test animals. For example, thalidomide can cause birth defects in humans with a dose as low as 0.5 mg. per kilogram of body weight. The minimum dose necessary to produce birth defects in other animals is much higher. Humans are 60 times more sensitive to thalidomide than mice, 100 times more sensitive than rats, and

700 times more sensitive than hamsters.

At one time, inhalation anesthetics were administered by the open drop method and even by placing an anesthetic-soaked cloth near the patient's mouth and nose. Modern inhalation anesthesia is administered via an anesthesia machine which accurately mixes the appropriate gases and vapors at the desired concentrations. A high-flow, semi-closed re-breathing system with a carbon dioxide absorber is most commonly used. Flow rates are usually maintained at 5 or 6 liters of gas per minute. The patient takes up less than 0.5 liter per minute, the excess gases escaping into the operating room through a "pop-off" valve located on the anesthesia machine. The valve is usually located within two feet of the person administering the anesthetic.

The occupational exposure to anesthetic gases can be reduced considerably with existing scavenging devices. Operating room levels of the various gases under usual working conditions are shown in Table 3. With installation of the proper scavenging equipment, exhausting the gas through the operating room ventilation system outlet, these concentrations can be reduced to less than 1% of the unscavenged level.

TABLE III

Range of Concentrations of Anesthetic Agents Found
in the Operating Room Environment

<u>Agent</u>	<u>ppm near anesthesiologist</u>	<u>ppm near surgeon</u>
Halothane	1-10	1-2
Methoxyflurane	2-10	1-2
Trichloroethylene	1-103	1-1.5
Nitrous Oxide	330-9700	310-550

The data presented here raise enough serious questions to justify regulations enforcing the installation and employment of gas scavenging devices in the operating room. Future research must be directed toward identification of those anesthetics which are embryolethal, teratogenic, mutagenic, or carcinogenic, with subsequent limitation or removal of these chemicals from the anesthesiologists' arsenal.

SELECTED READING

Cohen, E. N. et al: Occupational disease among operating room personnel: a national study. *Anesthesiology* 41: 317, 1974.

Corbett, T.H.: *Cancer and chemicals*. Nelson-Hall, Inc. (Chicago) 1976.

Rice, J.M.: An overview of transplacental carcinogenesis. *Teratology* 8: 113, 1973.

GENETIC EFFECTS ASSOCIATED WITH
INDUSTRIAL CHEMICALS

Joseph K. Wagoner, S.D.Hyg.
Peter F. Infante, D.D.S., Dr. P.H. and
David P. Brown, M.P.H.

Industrywide Studies Branch
Division of Surveillance, Hazard
Evaluation and Field Studies
National Institute for Occupational
Safety and Health
Cincinnati, Ohio

Each generation makes its own accounting to its children.
-- Robert F. Kennedy

In the past several decades, there has been an explosive proliferation of man-made chemicals in the environment and in the workplace. It is a subject of increasing concern that with few exceptions these chemicals have not been evaluated for their potential danger to this or future generations, either as carcinogens, mutagens or teratogens. At this Conference on Women and the Workplace, it seems only appropriate to review this spectrum of biological activity for select man-made industrial chemicals which have undergone evaluation and what the response of society to these findings has been.

In 1971, the toxicity of vinyl chloride (VC) broadened to include carcinogenesis (1). Inhalation studies by Maltoni and Lefemine demonstrated that VC induced adenomas and adenocarcinomas of the lung, lymphoma, neuroblastoma of the brain and angiosarcoma of the liver (2). Subsequent epidemiologic investigations of workers exposed to VC demonstrated an excessive number of deaths due to cancer of those same four organ systems: brain, liver, lung and lymphatic system (3). This observation of the carcinogenicity of VC, first in animals and subsequently in humans, had a profound positive effect on public health: first, in terms of the recognition of the need for rapid regulatory control of VC in the industrial setting; and second, in terms of an increased confidence and awareness of the value of animal bioassay.

VC also was demonstrated to induce tumors in the offspring of exposed pregnant rats (2). In addition, children born in communities contiguous to VC polymerization facilities were demonstrated to have an increased risk of birth defects, which included defects of the central nervous system (4). These two observations led many to propose that women of reproductive age should be excluded from employment in VC monomer production facilities or VC polymerization facilities, a "public health" approach in lieu of total containment of VC in the occupational setting (5). This public health response

to the toxicity of VC is cause for serious reflection. From a scientific viewpoint, the toxicity of VC on subsequent generations must be assessed not only through exposure of the female (trans-placental carcinogenesis, maternal toxicity and teratogenesis) but also through occupational exposure of the male (mutagenesis).

What evidence exists for the mutagenicity of VC? Several reports have indicated that VC is mutagenic in microbial test systems (*E. coli*, *S. typhimurium*, and *S. pombe*) (6-12), in insects (*Drosophila*) (13,14), in plants (*Tradescantia*) (15). VC metabolites also have induced mutations in mammalian cells (16). Likewise reports from four countries have shown an excess of chromosomal aberrations in lymphocytes of male workers exposed to VC as compared with controls not so exposed (17-20). Further evidence for the mutagenicity of VC has been provided by recent NIOSH investigations of fetal mortality among wives of male workers occupationally exposed to VC (21). Among pregnancies occurring prior to exposure, fetal death rates were 6.9% for the controls versus 6.1% after age adjustment for the primary VC exposure group, a difference not statistically significant (Table 1). Among pregnancies occurring subsequent to the husband's exposure, however, the difference in the fetal death rates between the study and the control group was significant, $p < 0.05$. Whereas, the difference in fetal death between the controls prior to and subsequent to employment, 6.9% versus 8.8%, was not statistically significant, the difference in age-adjusted fetal death rate among pregnancies for the primary VC group for prior to exposure and subsequent to the husband's exposure was statistically significant at $p < 0.02$. Additional analyses restricted to the pregnancies of women who had less than two, less than three and less than four fetal deaths, respectively, did not alter the findings of an association between male exposure to VC and an increased risk of fetal death (Table 2) (22). Unequivocal evidence for the mutagenicity of VC would be the finding of increased congenital anomalies among children born of males occupationally exposed to VC. Studies of abortions, however, have shown that most chromosomally abnormal embryos miscarry (23), thus making such unequivocal evidence for the mutagenicity of VC highly unlikely using the epidemiologic method. The finding of increased fetal mortality associated with occupational exposure of the male worker to VC, when taken in conjunction with the mutagenic response via microbial test systems, insects and plants and the observations of a significant increased risk of chromosomal aberrations among male workers exposed to VC point to germ cell damage in the father through direct VC exposure as the leading mechanism for these human observations. This spectrum of evidence for the mutagenicity of VC demonstrates that the only prudent public health approach to VC is through control of exposure at the source and not through the exclusion of women in the workplace.

Because of the carcinogenicity and mutagenicity of VC, a close look at other structurally similar chlorinated hydrocarbons seems in order. Two structural analogues of VC are vinylidene chloride

(VDC) and trichloroethylene (TCE), (Figure 1).

VDC, a chemical widely used in the manufacture of plastics, has recently been reported to be carcinogenic in the rat (24). In addition to this reported oncogenic response, VDC also has been shown to induce mutations in several microbial test systems (6,11, 25,26) and in a plant assay system (15) (Table 3). To date, evidence in humans is lacking to negate this mutagenic property of VDC.

TCE, a higher order structural analogue of VC, has been used for many years as an anesthetic (27,28). It also is widely used as a degreasing agent for metals and as an extractant in the food-stuff industry (29,30). Like VC, TCE has been demonstrated to induce hepatocellular carcinoma in mice (31). As was the case for VC and VDC, TCE also has been shown to be mutagenic in microbial assays (6,25,32) as well as plant assay (15) (Table 4). No studies of genetic risks among humans, however, have yet been undertaken for TCE.

Another chemically-related compound, 2-chlorobutadiene (chloroprene) is used extensively in the chemical industry, being the starting material for the synthetic rubber, polychloroprene. As early as 1936, Von Oettingen et al. (33) demonstrated damage in the reproductive organs of male mice and rats following skin application and inhalation of chloroprene. Even in low concentrations, 0.434 mg/l (120ppm) in rats and 0.042 mg/l (11.6ppm) in mice, chloroprene by inhalation affected the male reproductive organs, inducing degenerative changes and causing sterility in up to 60 to 70 percent of exposed animals (Table 5). Investigations by Davtyan et al. (34) in 1973 demonstrated that whereas inhalation of chloroprene by male rats at a concentration of 0.0038 mg/l (1.0ppm) did not affect fertilization capacity, it did nevertheless significantly increase embryonic mortality. They reported this same low concentration of chloroprene to induce chromosomal aberrations in bone marrow cells of mice.

A similar pattern of genetic effects in humans has been demonstrated following occupational exposure to chloroprene. Sanotsky reported that Fomenko and colleagues found functional disruption of spermatogenesis among males occupationally exposed to chloroprene for less than or equal to ten years and morphological disruption of spermatogenesis among men having greater than ten years of exposure to chloroprene (35). Sanotsky (35) in a survey of 143 workers exposed to chloroprene and 118 employees of an electro-machine construction plant (controls) also found a threefold excess of cases of miscarriage among wives of workers occupationally exposed to chloroprene.

An excess of chromosomal aberrations in lymphocytes of male workers exposed to chloroprene has been reported (36,37). The spectrum of evidence for the mutagenicity of chloroprene and for VC shows a marked similarity, from microbial assay through animal

assay to human observations (Table 6).

We must ask ourselves, what will be the response of Society to mutagens in the workplace:

Will Society require the enumeration of fetal deaths and/or congenital anomalies in humans before regulatory control is implemented?

Will Society now exclude the male worker from the industrial setting of chloroprene and vinyl chloride as previously proposed for women following the findings of transplacental carcinogenesis in animals exposed to vinyl chloride?

Or will the legacy of Society to future generations be the rapid reduction of human exposure to mutagens at their industrial source?

The questions are now before us. What will be the accounting of this generation to its children?

REFERENCES

1. Viola, P.L., A. Bigotti, A. Caputo. 1971. Oncogenic response of rat skin, lungs and bones to vinyl chloride. *Cancer Res.* 31: 516-519.
2. Maltoni, C., G. Lefemine. 1975. Carcinogenicity bioassays of vinyl chloride: current results. *Ann. N.Y. Acad. Sci.* 246: 195-218.
3. Waxweiler, R.J., W. Stringer, J.K. Wagoner, J. Jones, H. Falk, C. Carter. 1976. Neoplastic risk among workers exposed to vinyl chloride. *Ann. N.Y. Acad. Sci.* 271: 40-48.
4. Infante, P.F. 1976. Oncogenic and mutagenic risks in communities with polyvinyl chloride production facilities. *Ann. N.Y. Acad. Sci.* 271: 49-57.
5. U.S. Dept. of HEW, NIOSH. Recommended standard...Occupational Exposure to Vinyl Chloride. 1974.
6. Greim, H., G. Bonse, Z. Radwan, D. Reichert, D. Henschler. 1975. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes and a function of metabolic oxirane formation. *Biochemical Pharmacology.* 24: 2013-2017.
7. Bartsch, H., C. Malaveille, R. Montesano. 1975. Human, rat and mouse liver-mediated mutagenicity of vinyl chloride in S. typhimurium strains. *Int. J. Cancer.* 15: 429-437.
8. Rannug, U., A. Johansson, C. Ramel, C.A. Wachtmeister. 1974. The mutagenicity of vinyl chloride after metabolic activation. *Ambio.* 3: 194-197.
9. Garro, A.J., J.B. Guttenpaln, P. Milvy. 1976. Vinyl chloride dependent mutagenesis: effects of liver extracts and free radicals. *Mutat. Res.* 38(2): 81-88.
10. Andrews, A.W., E.S. Zawistowski, C.R. Valentine. 1976. A comparison of the mutagenic properties of vinyl chloride and methyl chloride. *Mutat. Res.* 40:273.
11. McCann, J., E. Choi, E. Yamasaki, B.N. Ames. 1975. Detection of carcinogens as mutagens in the Salmonella/microsome test: Assay of 300 chemicals. *Proc. Nat. Acad. Sci.* 72(12): 5135-5139.
12. Loprieno, N., R. Barale, S. Baroncelli. 1976. Evaluation of the genetic effects induced by vinyl chloride monomer (VCM) under mammalian metabolic activation: Studies in vitro and in vivo. *Mutat. Res.* 40: 85-95.
13. Sobels, F.H. 1975. Presented at the international symposium

on new developments in mutagenicity testing of environmental chemicals Oct. 13-17, 1975, Zinkovy Castle, Czechoslovakia. *Mutat. Res.* (In Press).

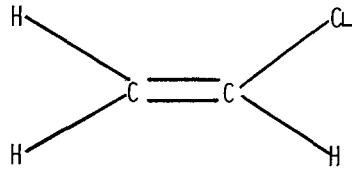
14. Magnusson, J., C. Ramel. 1976. Mutagenic effects of vinyl chloride in *Drosophila melanogaster*. *Mutat. Res.* 38: 114-115.
15. Sparrow, A.H. 1976 (Personal communication).
16. Huberman, E., H. Bartsch, L. Sachs. 1975. Mutation induction in Chinese Hamster V79 cells by two vinyl chloride metabolites chloroethylene oxide and 2-chloroacetaldehyde. *Int. J. Cancer.* 16: 639-644.
17. Ducatman, A., K. Hirschhorn, I.J. Selikoff. 1975. Vinyl chloride exposure and human chromosome aberrations. *Mutat. Res.* 31: 163-168.
18. Funes-Cravioto, F., B. Lambert, J. Lindsten, L. Ehrenberg, A.T. Natarajan, S. Osterman-Golkar. 1975. Chromosome aberrations in workers exposed to vinyl chloride. *The Lancet.* i: 459.
19. Purchase, I.F.H., C.R. Richardson, D. Anderson. 1975. Chromosomal and dominant lethal effects of vinyl chloride. *The Lancet.* ii: 410-411.
20. Loken, E., E. Thiis-Evensen. 1976. Preliminary report on the medical examination of 288 employees at the PVC plant, Norsk Hydro a.s., Porsgrunn Fabrikker, Porsgrunn, Norway. Unpublished manuscript.
21. Infante, P.F., J.K. Wagoner, A.J. McMichael, R.J. Waxweiler, H. Falk. 1976. Genetic risks of vinyl chloride. *The Lancet.* i: 734-735.
22. Infante, P.F., J.K. Wagoner, R.J. Waxweiler. 1976. Carcinogenic, mutagenic and teratogenic risks associated with vinyl chloride. *Mutat. Res.* (In Press).
23. Carr, D.H. 1972. Cytogenetic aspects of induced and spontaneous abortions. *Clin. Obstet. Gynecol.* 15: 203-219.
24. Personal communication reference in Bartsch, H. et al. 1975. Tissue mediated mutagenicity of vinylidene chloride and 2-chlorobutadiene in *S. typhimurium*. *Nature.* 255: 641-643.
25. Bartsch, H., C. Malaveille, R. Montesano, L. Tomatis. 1975. Tissue mediated mutagenicity of vinylidene chloride and 2-chlorobutadiene in *S. Typhimurium*. *Nature.* 255: 641-643.
26. Baden, J., R. Wharton, B. Hitt, M. Brinkenhoff, V. Simmon, R. Mazze. 1976. Mutagenicity of volatile anesthetics. *Fed.*

- Proc. Federation of American Society of Experimental Biology. 35: 410.
27. Defalque, R.J. 1961. Pharmacology and toxicology of trichloroethylene. A critical review of the world literature. Clin. Pharmacol. Ther. 2: 665-688.
 28. Dement, J.M., P.J. Bierbaum. Trichloroethylene usage as an inhalation anesthetic. U.S. Dept. of HEW, CDC, NIOSH: Unpublished data.
 29. Stewart, R.D., C.L. Hake, J.E. Peterson. 1974. Use of breath analysis to monitor trichloroethylene exposures. Arch. Environ. Health. 29: 6-13.
 30. U.S. Dept. of HEW, NIOSH. Criteria for a recommended standard. Occupational exposure to trichloroethylene. 1973.
 31. U.S. Dept. of HEW, (National Cancer Institute). 1976. Carcinogenesis Bioassay of trichloroethylene. Technical report series no. 2, CAS no. 79-01-6. NCI-CG-TR-2.
 32. Ramel, C., 1975. (Personal communication).
 33. Von Oettingen, W.F., W.D. Hueper, W. Deichmann-Gruebler, F.H. Wiley. 1936. 2-Chloro-butadiene (chloroprene): Its toxicity and pathology and the mechanism of its action. J. Ind. Hyg. Tox. 18(4): 240-270.
 34. Davtyan, R.M., V.N. Fomenko, G.P. Andreyeva. 1973. Question of the effect of chloroprene on the generative function of mammals (males). USSR Academy of Medical Sciences. The toxicology of new industrial chemical substances, issue no. 13: 53-58.
 35. Sanotsky, I.V. 1976. Problems of chloroprene toxicology (immediate and long-range effects). Symposium on potential environmental hazards from technological developments in rubber and plastics industry. National Institute for Environmental Health Sciences. Research Triangle Park, North Carolina, March 1-3, 1976.
 36. Katsova, L.D. 1973. Cytogenetic analysis of peripheral blood of workers engaged in the production of chloroprene. Gig. Tr. Prof. Zabol. 10: 30-33.
 37. Bochkov, N.P. 1975. Population studies in the chemical industry. International symposium on new developments in mutagenicity testing of environmental chemicals, Oct. 13-17, 1975, Zinkovy Castle, Czech. Mutat. Res. (In Press).
 38. Vogel, E. Mutagenicity of carcinogens in *Drosophila* as a func-

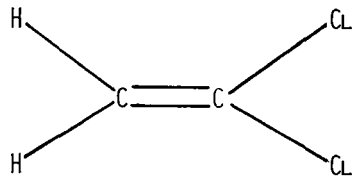
tion of genotype-controlled metabolism. Conference on In-vitro Metabolic Activation, National Institute for Environmental Health Sciences, Research Triangle Park, North Carolina, Feb. 1976.

FIGURE 1

VINYL CHLORIDE
(VC)



VINYLDENE CHLORIDE
(VDC)



TRICHLOROETHYLENE
(TCE)

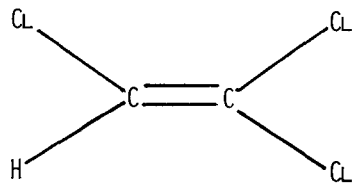


Figure Legend: Chemical Structure of three chlorinated ethylenes

TABLE 1

MEAN PATERNAL AGE, NUMBER OF PREGNANCIES AND FETAL DEATH
RATES ACCORDING TO HUSBAND'S VC EXPOSURE

	<u>CONTROLS</u>	<u>PRIMARY VC EXPOSURE</u>
<u>PRIOR TO HUSBAND'S EXPOSURE</u>		
Number of Families	95	70
Mean Paternal Age at Conception (Years)	23.0	26.4
Number of Fetal Deaths Among Wives	11	15
Number of Pregnancies	159	148
Age-Adjusted Fetal Deaths/100 Preg.	6.9	6.1
<u>SUBSEQUENT TO HUSBAND'S EXPOSURE</u>		
Number of Families	113	62
Mean Paternal Age at Conception (Years)	30.4	30.2
Number of Fetal Deaths Among Wives	24	23
Number of Pregnancies	273	139
Age-Adjusted Fetal Deaths/100 Preg.	8.8	<u>15.8*</u>

SOURCE: Reference 21.

TABLE 2

NUMBER OF PREGNANCIES AND AGE-ADJUSTED FETAL DEATH RATES
 ACCORDING TO HUSBAND'S VC EXPOSURE EXCLUDING
 PREGNANCIES IN WOMEN WITH >2, 3 OR 4 FETAL DEATHS

	<u>Controls</u>		<u>Primary VC Exposure</u>	
	<u>Number of Pregnancies</u>	<u>Fetal Death Rate</u>	<u>Number of Pregnancies</u>	<u>Fetal Death Rate</u>
<u>>2</u> Fetal Deaths Excluded				
Before Husband's Exposure	155	5.8%	126	1.7%
After Husband's Exposure	255	4.7%	111	6.2%
<u>>3</u> Fetal Deaths Excluded				
Before Husband's Exposure	159	6.9%	141	3.1%
After Husband's Exposure	265	6.8%	120	10.8%
<u>>4</u> Fetal Deaths Excluded				
Before Husband's Exposure	159	6.9%	142	5.8%
After Husband's Exposure	265	6.8%	127	11.8%

Rates for the primary VC exposure group are age-adjusted to the Control group.

Source: Reference 22.

TABLE 3
MUTAGENICITY TESTING FOR VINYLIDENE CHLORIDE (VDC)

<u>TEST SYSTEM</u>	<u>RESULTS</u>	<u>REFERENCE-AUTHOR(YEAR)</u>
I. Microbial System		
A. Salmonella Typhimurium	† † †	McCann, et al. (1975) (11), Bartsch, et al. (1975) (25), Baden, et al. (1976)(26)
B. E. Coli	†	Greim, et al. (1975) (6)
II. Plant System		
A. Tradescantia	†	Sparrow (1976)(15)

TABLE 4
MUTAGENICITY TESTING FOR TRICHLOROETHYLENE (TCE)

<u>TEST SYSTEM</u>	<u>RESULTS</u>	<u>REFERENCE-AUTHOR(YEAR)</u>
I. Microbial System		
A. Salmonella Typhimurium	† (?) †	Baden, et al. (1976) (26), Rame1 (1975)(32)
B. E. Coli	†	Greim, et al. (1975) (6)
II. Plant System		
A. Tradescantia	†	Sparrow (1976)(15)

TABLE 5
EFFECT OF INHALATION OF CHLOROPRENE ON THE REPRODUCTION
OF MALE MICE AND RATS

<u>SPECIES</u>	<u>NUMBER OF ANIMALS</u>	<u>PREGNANCIES</u>	<u>FERTILITY RATE (%)</u>
<u>Mice</u>			
0.042 - 0.548 mg/l (11.6 - 151.4 ppm)	14	6	43
Controls	5	5	100
<u>Rats</u>			
0.434 - 22/419 mg/l (120 - 6190 ppm)	19	6	32
Controls	5	5	100

From: Reference 33

TABLE 6

EVIDENCE FOR MUTAGENIC OR REPRODUCTIVE
EFFECTS OF VINYL CHLORIDE AND CHLOROPRENE

TEST SYSTEM	AGENT	
	VINYL CHLORIDE	CHLOROPRENE
<u>LABORATORY</u>		
Microbial (E. Coli, S. Typhimurium, S. Pombe)	† (6-12)	† (25)
Insect (Drosophila)	† (13,14)	† (38)
Plants (Tradescantia)	† (15)	?
Mammals	† (16)	?
Metabolites in hamster cells		
Chromosomal aberrations in male rats	?	† (34)
Reproduction interference following exposure to male mice or rats	- (19)	† (33,34)
<u>HUMANS</u>		
Chromosomal aberrations in male workers	† (17-20)	† (36,37)
Excess miscarriage in wives of male workers	† (21,22)	† (35)
Decrease in motility and number of sperm in workers	?	† (35)

BIRTH DEFECTS AND VINYL CHLORIDE

Mr. Larry Edmonds
Health Services Officer
Birth Defects Branch
Center for Disease Control

I would like to address the question of vinyl chloride monomer exposure and its possible relationships to birth defects. If there is a relationship between vinyl chloride and birth defects, it could be either a mutagenic effect, prior to conception, or a teratogenic effect after conception.

One of the first reports that vinyl chloride may be related to birth defects in humans was presented in March, 1975, by Dr. Infante at the New York Academy of Science. The study conducted by Dr. Infante, then with the Ohio Department of Health, noted increased rates from 1970 to 1973 in three Ohio communities where polyvinyl chloride plants were located. Anomalies of the central nervous system were of greatest concern and most of the excess was attributed to the city of Painesville, Ohio. The concern was that residential and occupational exposure might be associated with increased rates of CNS defects.

At the Center for Disease Control, data are collected through the Birth Defects Monitoring Program (BDMP). Briefly, the BDMP is a national, hospital-based system for monitoring the incidence of birth defects and other newborn conditions from hospital discharge diagnoses; it consists of approximately 1,200 hospitals monitoring a million births per year from 1970.

The BDMP provided data with which we could further examine the suggested increase in CNS rates in Ohio. We identified two cities which had a polyvinyl chloride plant, and one BDMP hospital believed to serve each community. One hospital was in Pottstown, Pennsylvania, and the other was in Painesville, Ohio.

Selected malformation rates for these two hospitals -- 1970 to 1974 -- were compared with rates for their respective states. The rates seen in Pottstown, Pennsylvania, were 21.6 per 10,000, approximately what was expected from the national BDMP rates. The expected number of defects in Painesville was approximately 11. The observed number of 22 cases was twice the expected.

On the basis of this increase, an investigation was undertaken in Painesville. Painesville, Ohio, is an industrial city on Lake Erie, near Cleveland, with a population of approximately 16,000. There are two polyvinyl chloride plants located in Painesville. They are across the street from each other and about two and a half miles from the center of the city. One of them has been in operation since 1949 and the other since 1967.

The investigation was a hospital-based study and was limited to cases of anencephaly and spina bifida. The BDMP data were compared to birth certificate data obtained from the Ohio Department of Health. One additional case of anencephaly was found, making a total of 15 cases.

The hospital records were reviewed to confirm the diagnosis and obtain other demographic data. For each case, two controls were selected from the hospital records; the first normal, white birth before and after the case, since all the cases were white.

The physician of each mother in the case group was contacted and permission obtained to interview the parents concerning their past occupational and residential histories. The investigation did confirm increased rates for anencephaly and spina bifida compared to expected statewide rates.

The hospital rates for anencephaly (14.1 per 10,000 births) and for spina bifida (21.2 per 10,000 births) were greater than two times the expected rates, based on the BDMP state data. None of the 14 cases interviewed had ever worked at either of the two polyvinyl chloride plants in Painesville. One set of parents could not be located, but hospital records indicated that they did not work at the plant at the time of their infant's birth.

Among the 30 control parents, two fathers had worked at a polyvinyl chloride plant at the time of their infant's birth. No parent in either case or control group lived within two miles of either plant.

Statistical comparisons on distances varying from one mile to 10 miles of parental residence to the plant indicated that there was no difference between the cases and control.

Local increases in CNS and other malformation rates, such as seen in Painesville, are not uncommon. In this particular study, no association could be found between VC and the higher rates of CNS defects.

A second and similar study was just completed in Kanawha County, West Virginia. Because of the continued concern about the teratogenic potential of vinyl chloride, BDMP data were again reviewed for all counties having a polyvinyl chloride plant and at least one BDMP hospital.

In the earlier study, the selection was limited to cities with only one hospital serving the entire community. At CDC, we identified 17 counties in the U.S. which have a polyvinyl chloride plant and at least one BDMP hospital.

Four of these 17 counties had rates for CNS defects that were significantly higher than rates for the U.S. for the time period,

1970 to 1974. Two of the counties so identified had less than 50 percent of total births in the BDMP and were excluded from this study.

The two remaining counties with increased rates were Lake County, Ohio, (Painesville) the site of the previous study -- and Kanawha County, West Virginia, where 95 percent of county births were monitored. In Kanawha County there were 59 infants born between 1970 and 1974 with CNS defects. The expected number, based on U.S. rates, was 37.

The principal centers of population in Kanawha County include Charleston and South Charleston, which are located along the Kanawha River which meanders through the valley's hilly terrain. The Kanawha River Valley is known for its chemical industry. There are seven major chemical plants and several smaller ones within 10 miles of Charleston. There is one polyvinyl chloride plant, located in South Charleston, bordering Charleston, which began production of polyvinyl chloride in 1935.

Two sources of data were reviewed to identify all cases of central nervous system defects occurring in Kanawha County between January 1, 1970, and December 31, 1974. One source was a case listing obtained from the Birth Defects Monitoring Program which would list the cases occurring in the BDMP hospitals in the county. The second source was vital records. The West Virginia Department of Vital Statistics provided all cases of CNS birth defects occurring between January 1, 1970, and December 31, 1974.

The two lists were combined and the hospital records reviewed to confirm the diagnosis and the place of residence. For each confirmed case, two controls whose parents resided in Kanawha County were selected from vital records. These controls were the first normal, live-born infants whose certificates preceded and followed a case.

The cases and controls were matched according to month of birth, race, paternal education, and maternal age. With the permission of the local physicians, all cases and one randomly chosen control were interviewed. The parents were told that this was a birth defect study and vinyl chloride was not mentioned. The following information was obtained: history of previous pregnancies; five year residential history, prior to and including the time of birth of the affected infant; and five year occupational history of both parents, including the location of work and any possible chemical exposures.

TABLE 1

INCIDENCE OF CNS MALFORMATIONS IN KANAWHA COUNTY,
W. VIRGINIA AND THE UNITED STATES, 1968-1975
(RATES PER 10,000 TOTAL BIRTHS)

	<u>BDMP Hospital Data</u>		<u>Vital Statistics</u>		<u>Confirmed Cases</u>	
	<u>U.S.</u> <u>Rate</u>	<u>Kanawha Co.</u> <u>Case Rate</u>	<u>Kanawha Co.</u> <u>Case Rate</u>		<u>Kanawha Co.</u> <u>Case Rate</u>	
1968	N.A.	N.A.	(13)	35.4		
1969	N.A.	N.A.	(8)	20.3		
1970	21.8	(12) 35.9	(16)	42.8	(14)	37.5
1971	22.1	(19) 51.7	(13)	34.4	(15)	39.7
1972	21.6	(11) 30.5	(13)	37.9	(12)	34.9
1973	19.9	(11) 28.9	(5)	15.0	(5)	15.0
1974	18.4	(6) 14.4	(1)	3.0	(1)	3.0
1975	18.2	(10) 22.9				
1970-						
1974	20.6	(59) 31.7	(48)	27.2	(47)	26.7

The BDMP rates are given in the first column of the table. The high Kanawha County rates compared to the U.S. rates are what prompted the investigation. The last column gives the rates for the confirmed cases from 1970 to 1974. As you can see, 1970, 1971, 1972 were the highest years, with the rates starting to decline in 1973 and only one case observed in 1974.

Forty-six cases and their matched controls were located and interviewed. Five cases were not residents of Kanawha County at the time of conception and were eliminated from the study, except in calculation of rates, which are based on residency. Close matching was achieved between the cases and controls for paternal education, maternal age, and the Hollingshead index, which is an indicator of socioeconomic status. Reproductive history revealed that the total number of pregnancies were similar between the cases and controls; 49 for the case group and 48 for the control group. The live birth percentages were also very similar between the two groups. The fetal death rates, which include spontaneous abortions and stillbirths, were 22% for cases and 23% for controls.

Occupational histories revealed that two cases and two control fathers were employed at the vinyl chloride plant at the time of conception. Fathers of three additional controls also had worked at the vinyl chloride plant on a contract basis and may have had possible vinyl chloride monomer exposure. None of the mothers in either the case or control group had worked at the polyvinyl

chloride plant, but few women work in the vinyl chloride industry.

The residences of case and control parents, at conception, were plotted on county maps and the distances from the plant were calculated. Using a chi square test, at one mile intervals, no statistical difference was observed between the cases and controls. Matched pairs of cases and controls were also tested, using a matched-pair T test of significance, and no difference was observed. While no difference was observed in the distance of residence from the plant, inspection of the map suggests that, within the five mile area, cases were concentrated to the northeast of the river, and the controls were concentrated to the southwest.

From the polyvinyl chloride plant, X and Y co-ordinates were determined for the parents' residence, at the time of conception, for each case and control. Cases and controls were then compared for a statistically significant difference in their respective centers of gravity, which is the mean position of the group. The only statistically significant difference ($p.02$) noted was within the three mile area. There were 10 controls and 9 cases within this area.

Both cases and controls within the three mile area had a higher socioeconomic index than those outside the area. No difference was noted between the cases and controls in regard to occupation or length of residence within the three mile area.

Cases did have an interesting family history of birth defects. One case mother gave birth to two of the cases within the three mile area. Another case mother had had a sister with spina bifida. A third mother in this case group had reported a cousin with a heart defect. While familial components do not exclude environmental cause, they suggest a possibility of a genetic causation.

Atmospheric and environmental data related to vinyl chloride monomer and other chemical emissions in Kanawha County are limited. Some data were provided by the vinyl chloride plant. These data have been collected over the past several years.

On October the 1st, 1974, the largest single emission was noted and the VC levels were measured in the community. The highest atmospheric levels were .1 and .2 parts per million. At that time the wind was from the southwest, and these levels were found to the northeast of the plant.

Additional data were available from the West Virginia Air Pollution Commission. These data, gathered over the past several years, dealt with suspended and settleable particulate matter. They indicate just the opposite of the VC measurements. They indicate that the highest concentrations of the settleable and suspended particulate matter were to the southwest, suggesting a pre-

dominant wind direction from the northeast.

In addition to these conflicting data, other data available on wind and temperature data, coupled with the terrain, suggest that Kanawha County in the Kanawha Valley is a veritable mixing bowl for any emissions. There have been identified over a hundred chemicals emitted into the valley. The effect of these chemicals, alone or in combination, is not known, and to attribute this case pattern within three miles to the effect of one chemical plant is unwarranted.

In conclusion, both studies did confirm higher CNS rates than expected during the early 1970's. CNS rates are not significantly different from the U. S. rates at the present time. Occupational histories in both studies revealed no difference between the case and the controls in possible work exposure to vinyl chloride monomer. Residential histories showed no difference between the case and controls when compared at varying distances from the poly-vinyl chloride plants.

However, in Kanawha County, we did see a difference in the pattern of residents for cases and controls living within three miles. No explanation of this difference could be found, but the available data suggested no association with vinyl chloride monomer. Further plans are to monitor the counties identified earlier in the birth defect monitoring program. We also are gathering back data from Kanawha County to 1958 in order to determine the CNS rates for these years.

PLACEMENT OF WOMEN IN HIGH-RISK AREAS:
AN INDUSTRY VIEWPOINT

Dr. Harold L. Gordon
Medical Director
Dow Chemical Corporation
Midland, Michigan

Occupational hazards to women fall roughly into one of three categories: those common to all workers, those associated with sex-role stereotyped jobs, and those related to the biological make-up of women. The following discussion deals primarily with one aspect of the third category, namely the "pregnant" female in industry. The discussion is restricted by design because it is felt current policies can be applied successfully to most employees, irrespective of sex. However, the pregnant and soon to become pregnant female employee needs to be included in that small group of workers who, because of their physical or mental status, must receive special consideration with regard to job placement.

By way of background, it must be understood that about 30 to 40 percent of all conceptions usually result in a spontaneous abortion, still birth, or live birth with congenital malformation. An undetermined number of these are probably a result of some environmental factor(s). Industry has a moral and legal responsibility to control environmental factors of the workplace.

A simplistic approach would be to limit the placement of all fertile females to jobs that involve no exposure to real or potential carcinogens, transplacental carcinogens, teratogens or mutagens. No exposure would mean no imposition of risk on female employees; but this approach is unrealistic. This puts a company in possible conflict with E.E.O. and it deprives industry of a large pool of potential workers.

The opposite tack could also be taken and no special provisions made for women. This also is unrealistic. It ignores pregnancy.

So, some middle course must be taken. It must reflect the current "state of the art" and have the capability of reacting to the results of new research. It must be workable. This means that the fewest number of people possible should be involved in making routine decisions -- the patient-employee and the examining physician. Education must be provided to the patient-employee and guidelines to the examining physician. There should be a method for orderly override of the guidelines in those few cases where there may be extenuating circumstances. Perhaps this could best be handled by a committee composed of representatives from Personnel, Legal, Industrial Hygiene, Toxicology (teratology) and Medical. Involvement by the individual and her personal physician must also

be considered in any decision.

This is fine, but how is it to be done? What is the population at risk? How is it identified? What specific agents need to be considered? How are they identified? What restrictions are needed? When do they apply? To whom do they apply? What are the legal and economic considerations? What are the action steps?

What is the population at risk? Theoretically, any woman between puberty and menopause, irrespective of her marital status, who has not had a sterilization procedure is capable of bearing children. Is this the population of concern? In a sense yes, the restriction of "fertile females" would definitely protect both mother and child through the full course of the pregnancy, but for practical reasons, the guidelines on this discussion apply primarily to that smaller population of women who are pregnant or are actively contemplating pregnancy.

How is this population identified? Without the active cooperation of the female employee, it probably isn't, at least not at a stage when our efforts would have the maximum effect. Ideally, we would want to react to the patient just prior to conception. For a small number, this would be possible; but for most, this is not realistic and the best to be hoped for is a diagnosis shortly after conception. Laboratory procedures are available which allow the diagnosis of pregnancy to be made as early as five days after the first missed period; but the diagnosis can't be made unless the patient presents herself for testing; nor, as was facetiously suggested, can a pregnancy test be made with each paycheck. An extensive educational program and perhaps a modification of certain personnel procedures will be needed before routine control of this problem will be successful.

What specific agents need to be considered? Although temperature extremes and emotional stress, for instance, have been reported to be teratogenic agents, the recommended guidelines have been limited, at least initially, to chemical and biological materials. Emotional stress in particular is too difficult to define, quantify and modify for it to be included at this stage.

How are the chemical and biological agents of concern identified? For this we must rely heavily upon the teratologists. By exclusion, all other chemicals fall into the category of unknown. These lists should be reviewed and updated on a periodic basis. The actual hazard will have to be evaluated on a job-by-job basis by the examining physician and/or the special committee.

What restrictions are needed? The following guidelines are suggested for non-pregnant women who are pre-menopausal and have not had a tubal ligation or other sterilization procedure:

TERATOGENS

A. Known human teratogens.

Women should not be hired in positions where there is exposure to human teratogens. Where there are exceptions there should be:

1. An educational program to alert the women employees to the teratogenicity of the specific chemical.
2. Employee acknowledgment in writing of the risks of the job.
3. Discussion with the examining physician.
4. If after consultation with the physician, the woman still wishes to work in the area, the physician could refer the request to the special committee.
5. Further development of retrospective data.

B. Known animal teratogens.

Women are not automatically excluded from areas of potential exposure to known animal teratogens, but for any woman who is to be hired for a position with this hazard there should be:

1. An educational program alerting her to the possibility of teratogenic response in humans.
2. Acknowledgment in writing of her understanding of the potential risks.
3. Discussion with the examining physician, with possible referral to the special committee.
4. Further development of data.

C. Teratogenic by analogy.

1. Educational program alerting the women to the possible teratogenic effects of the material with no discrimination as to placement.
2. Further development of data.

TRANSPLACENTAL CARCINOGENS

Guidelines similar to those of teratogens.

CARCINOGENS, MUTAGENS AND UNKNOWN

A. Educational program.

B. Unless the chemical is also in the teratogen or transplacental carcinogen categories, no additional special provisions on the basis of sex are suggested.

As part of each educational program, it would be expected there would be a discussion of the need for early detection of pregnancy. It would be stressed that because of the unknowns associated with the effects of chemicals upon fetal development, special precautions need to be taken for the potential protection of the fetus. It would be emphasized that an employee who is planning to

become pregnant or suspects she is pregnant should be evaluated and counseled at the Medical Department.

If women are moved out of areas of concern and this proves to have a detrimental effect on their paycheck or careers, industry probably will find that the cooperation of its women employees on early reporting to Medical will be rapidly lost. This cooperation is the keystone to the whole operation. Personnel management should try to develop procedures which promote this cooperation.

To whom do these restrictions apply? There has been some talk as to the need for one policy for salaried employees and another for hourly employees. "Exempt" and "Non-Exempt" may be convenient administrative terms, but they don't apply to biological problems. Any policy should apply across the board to all employees -- salaried, hourly, full-time, part-time and co-ops. The special problems of job assignment to manufacturing or research will have to be handled on an individual basis.

What are the economic and legal problems? The biggest problem is going to be the threat of law suits. On the one hand, there is potential for litigation by E.E.O.: on the other, law suits on behalf of every unsuccessful pregnancy. The Legal Department should be able to define these legal and economic problems in more detail. In any case, industry must be able to defend its policies in court.

While I have noted what industry's posture should be, I feel strongly that a similar posture must be taken by government and academia as well. These problems know no home -- they are ever present.

What are the action steps?

1. Present proposal to management and gain its support.
2. Continue to define lists of teratogens, transplacental carcinogens, carcinogens and mutagens.
3. Develop education programs.
4. Form the committee to review special cases. This committee should include representatives from Personnel, Toxicology (teratology), Industrial Hygiene, Legal and Medical.
5. Generate lists of nonoccupational agents to be avoided during pregnancy.

PLACEMENT OF WOMEN IN HIGH-RISK AREAS:
A WORKERS' VIEWPOINT

Sylvia Krekel
Occupational Health and Safety Specialist
Oil, Chemical and Atomic Workers
International Union

This conference on women in the workplace underscores the growing recognition of women as an integral rather than a peripheral part of the workforce. The sexist theory of women working for "pin money" has been supplanted to some degree by an acknowledgment that women are "real" workers whose jobs support households rather than merely supplementing the family income. The fact that women now make up over 40% of the workforce has spawned a host of articles, conferences, committees and caucuses all devoted to the varied problems encountered by the working women. One can scarcely pick up a magazine or newspaper today without seeing some mention of the social, political, economic or physical repercussions of women in the workplace.

Legally, we've come a long way. The Equal Pay Act, Title VII of the Civil Rights Act and Executive Order 11246 all prohibit sexual discrimination to varying degrees in employment. As a direct result of such federal legislation, specifically Title VII, state protective laws are clearly unlawful and, where still existing, are under heavy attack by those who recognize that theoretical protection almost always means practical restrictions.

Yet, as the topic of this session suggests, we still have a long way to go before women enjoy the job equality afforded the male workers. That "placement of women in high-risk occupations" should be a controversial topic subject to conflicting viewpoints says a lot about where we are in the area of sex discrimination on the job.

Also, I think it's essential to remember that, although this conference deals specifically with the health hazards faced by women at work, these hazards are not discrete and separate from the other problematic aspects of women working. For job health is not a purely scientific or medical problem, but rather, is intimately tied in with economic and political considerations. For instance, why is it that the lead industries refuse to hire fertile women when recent European studies indicate that men suffer a decrease in fertility from lead absorption as well as lead poisoning? If the lead industry is seriously concerned over the reproductive effects of lead exposure, then the emphasis should be placed on the reproductive capacity of both male and female.

To cite another example, take the matter of radiation standards. The current standard observed by both ERDA and the Nuclear Regula-

tory Commission sets exposure limits at 5 rems annually for every worker. At the same time, the regulatory guide published by these agencies recommends that every licensee inform women workers that there is a risk to the fetus posed by the 5 rems standard, and that pregnant women should, therefore, be advised against working at these levels.

The Natural Resources Defense Council has petitioned that the standard be lowered to 1/2 rem per year, a level that will substantially protect all workers, including the fetus, from damage arising from radiation exposure. However, the NRDC petition included only those workers up to the age of 45.

Our union, the OCAW, conducted a survey of our workers in the atomic industries. We found that over 83% of OCAW-represented atomic workers were already working under 1/2 rem per year, so we supported the NRDC petition knowing that it would impose no undue difficulty for plants to meet. However, our members protested the 45 year cutoff point since a 50 year old man is perfectly capable of fathering a child and, in fact, many had. Thus, OCAW recommends that the 1/2 rem standard be applied to all workers without respect to age.

{So it seems that much of the clamor over women working in high-risk occupations is often little more than a smoke screen to conceal industry's reluctance to place a priority on people rather than profits. If a few dollars can be saved by screening out those people who are most visibly affected by toxic substances, then the position seems to be save the money and damn the worker.

Another aspect of the concern over women's exposure to toxic substances may very well be that women have been chosen by corporations to become economic scapegoats. During a period of high unemployment such as we are now experiencing, women would seem to present an attractive choice for job discrimination. There is still a vague fear of women taking jobs away from men who desperately need them.

There still exists a fairly strong vein of ingrained prejudice against women leaving the home. Industry can effectively exploit these and other latent prejudices. And what better rationale can be offered for discrimination than "protection". So we have come full circle from the establishment of state protective laws to their invalidation under Title VII to their reinstitution among industries today.

And screening women from so-called "high-risk areas" means that women suffer from triple discrimination in the workplace. First of all, women are hampered by the fact that they are female, making it hard to break into the workplace initially. Once this hurdle is overcome and they get a job, the woman worker is the first to get laid off since she has the least seniority. With the addition of screening policies aimed at the fertile female, the

position of women in the workplace becomes tenuous at best. The spectre of triple discrimination becomes even more ominous if the woman becomes pregnant, and the company decides she must transfer to another job for the sake of the fetus. She then loses the little seniority she may have acquired and usually must take a job at a lower wage scale.

So where do we look for a solution for this threat of triple discrimination against women in the workplace?

From the workers' viewpoint, the solution is simple and unequivocal. It is based on the law as well as on some basic moral principles. Clearly, the workplace should be made safe for all workers regardless of sex, age or reproductive capacity. The Occupational Safety and Health Act clearly guarantees "every working man and woman in the Nation safe and healthful working conditions". Nowhere in the act is there any distinction made between the sexes in terms of working conditions. So, it is only through promulgation of standards that protect all workers that we will avoid the blatant abuses implicit in discriminatory hiring and placement procedures. Any other policy than that of safety and health for all workers would logically culminate in a kind of labor Darwinism in which those workers considered "unfit" for the job would be selectively weeded out of the labor force. "Survival of the fittest" is the ultimate outcome of discriminatory placement policies.

Therefore, the workers' position is that health standards should be set at levels low enough to protect everyone in the workplace, including the fetus. However, in the interim before such standards are made mandatory by OSHA and until employers effect the requisite engineering controls for these standards, certain administrative controls may be necessary.

For instance, we have to recognize that pregnancy is a special condition and may demand special protections. In such a case and in any case involving the transfer of a worker to another department on the basis of health considerations, the employer has the obligation to keep the worker whole. Not only must the worker's health be protected, but he or she must also be kept economically whole. Any protective transfer must be accompanied by protection of the worker's seniority and rate of pay. The burden most definitely rests upon the employer to bear the costs of keeping the worker economically and physically whole.

Of course, none of this protection, even though written into law, is won without struggle. The labor movement will continue its fight against job discrimination under whatever guise it occurs, using all means available including legal actions, the collective bargaining agreement, and the arena of public opinion.

PLACEMENT OF WOMEN IN HIGH-RISK AREAS:
A LEGISLATIVE VIEWPOINT

Peggy Taylor
Professional Staff
U.S. Senate Subcommittee on Labor
and Welfare

Before I make my remarks, I'd like to make the disclaimer that these remarks are my own, as a staff member of the Senate Labor Subcommittee, and do not necessarily represent the views of Chairman Harrison Williams or the other members of the Subcommittee.

I'm sure all of you are wise enough to know that the very complex problems of how to assure adequate protection to child-bearing women in the workplace are not susceptible to any easy solution -- legislative or otherwise.

In the early 1900's, many states passed so-called "protective legislation" to spare the "weaker sex" the long hours, hazardous jobs and unsanitary conditions. Dr. Alice Hamilton, believing that women might be more susceptible to some occupational health hazards, urged passage of special laws to protect women in the workplace.

Since World War II, the legislative and social movement has been directed at removing institutional distinctions between men and women and treating all parties equally.

To determine what might be the legislative viewpoint on placement of women in situations which may pose a high risk to a certain subset of these women, we have to define, explicitly, what the problem is and identify legislation already in existence which touches on it.

First, what is the client group which works at high risk? While this conference is called Women and the Workplace, there has been some discussion of the greater appropriateness of looking at male and female reproductive systems in the workplace.

However, this would lead you to consider as special a fertile group of women, ages 15 to 44, and men, 15 years and older, since men do not lose their fertility at menopause as do most women. Once we have a group of people so broad as this, we're no longer talking about a special, highly susceptible group which needs to be accorded special treatment. When the issue is hazard to the reproductive systems of both men and women, you are led to the preparation of occupational health standards which have taken into account research that assesses impaired ability among both sexes to produce healthy children.

Men in the workplace may be a problem of its own. I think we

don't yet have even a skeletal idea of how broad the work-related problems of male sterility, flaccidity and so forth may be, and whether these effects occur at exposure levels that also do damage to the reproductive capability of women.

Even when you are talking about women, it is not easy to define very specifically the group you would act to protect. Obviously, the exceptional quality of women is that they carry the fetus and bear the child. And substances to which the mother is exposed are frequently carried through the placenta to the fetus, with the ensuing risk of hazard.

Women of childbearing age are considered to be those of 15 to 44 years of age. In 1975, this represented approximately 25 million workers, 68 percent of the women who were working. Now, OSHA has estimated that over 1 million of these women are working in occupations where there is potential exposure to chemical substances which may cause birth defects and/or miscarriages.

In discussing methods of protection, it would be useful to be able to pare down this group to those women who will bear children. Perhaps this suggests that those women who are or who intend to become pregnant should be covered by a certain set of protections. This group, however, is probably impossible to define. In 1970, 39 percent of the births to married women were unplanned. In 1974, 346,000 children were conceived by unmarried women under the age of 20, and one would presume, in most cases, were unplanned. Another measure of unplanned pregnancy can be inferred from the fact that in 1975, just under 1 million abortions were performed.

These figures would lead one to believe that there is very little assurance of adequate identification of the group at risk if we were to rely on reports of intended pregnancy. So we are dealing with two nonspecific pieces of information:

One, at some level of exposure to the pregnant female, some substances cause damage to the fetus.

Two, there is a group of women who cannot be reliably defined, except by the most gross measures, who will carry these fetuses.

When looking for a way to tackle the problem and protect against injury arising from employment, we have to consider the existing and potential legislation, which requires and/or may limit the means to such protection.

Four pieces of existing and proposed legislation come to mind. The Occupational Safety and Health Act; the Equal Employment Opportunity Act, which is Title VII of the Civil Rights Act; the National Workers' Compensation Act, which has been introduced by Chairman Williams; and the Senate-passed Toxic Substances Control

Act.

The Occupational Safety and Health Act, which was authored by Senator Harrison Williams, who is Chairman of the Labor and Public Welfare Committee, states in Section 6(b) (5) that "The Secretary, in promulgating standards dealing with toxic materials or harmful physical agents under this subsection, shall set the standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capability even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life".

OSHA, in the preamble to the lead standard, has stated that "it is appropriate to consider these sizeable groups, (including women of childbearing age...), who may exhibit a greater susceptibility in setting a level, which applies to all workers, in view of our objective, consistent with Section 6(b) (5) of the Act, to set standards which protect more than just the least susceptible employees and in view of the need to assure the fullest employment opportunities to all American workers".

And the Department of Labor Solicitor has said, with regard to the proposed lead standard, that he would not bar women from the workplace under any circumstances.

It should be understood that these statements by the Department of Labor do not add up to a policy position that OSHA will set the threshold limit value at a level which would protect pregnant women from damage to the fetus, but they do give official recognition to the potential special hazard to women workers.

On the other hand, to protect women from other injustices, we also have Title VII of the Civil Rights Act, also known as the Equal Employment Opportunity Act, which prohibits discrimination based on race, color, religion, sex and national origin of all terms, conditions and privileges by employers, employment agencies and labor unions.

Under the Equal Employment Opportunity Act, a company must prove that an action which treats women differently from men is a business necessity. Otherwise, discrimination will be assumed.

In *Griggs versus Duke Power Company*, the landmark case on "business necessity", the Fourth Circuit Court opined that "Congress has now provided that ... criteria for employment or promotion may not provide equality of opportunity merely in the sense of the fabled offer of milk to the stork and the fox".

The Courts have more humor than we do in what they write. I am trying to find out what the fable of the offer of milk to the stork and the fox was. I asked two lawyers on our committee staff

and three people at the Library of Congress, and nobody knew what the fable of the stork and the fox was, except that it has some relationship to milk being offered in a jug with a long neck, so that the offer of milk, in fact, only enabled the stork to drink and not the fox. I don't know the rest of the fable.

The court decision further states: "On the contrary, Congress has now required that the posture and condition of the job seeker be taken into account. It has -- to resort again to the fable -- provided that the vessel in which the milk is proffered be one all seekers can use. The Act proscribes not only overt discrimination but also practices that are fair in form, but discriminatory in operation. The touchstone is business necessity. If an employment practice which operates to exclude ... cannot be shown to be related to job performance, the practice is prohibited."

A further legislative factor which must be confronted is the proposed National Workers Compensation Act of 1975, which assures compensation for injury or disease "rising out of and in the course of employment".

There has been very minimal discussion of the eligibility for compensation of the child who is injured in the course of fetal presence in the endangering workplace. But case law, after the passage of the Act, may well test the position that such a child may recover damages for life under workers' compensation.

Lastly, the Senate-passed Toxic Substances Control Act, in Section 4(b) (2), states that the health effects for which standards for the development of test data may be prescribed include carcinogenesis, mutagenesis and teratogenesis. This provision creates the opportunity to generate much more data than is presently available on the effects of chemical exposures of the women on the yet unborn child.

The approach of removing the hazard to highly susceptible groups, such as pregnant women, is an obvious first choice solution under existing legislation where that's feasible.

So far as I know, we, as yet, know very little about the levels at which various chemicals cause fetal damage. From the theory that the periods of the most rapid cell division are the times of greatest susceptibility to intrusion of any sort, it would then follow that there is an increasing susceptibility to the ill effect of certain chemicals, as one moves down the age scale, from the adult to the child to the fetus to the embryo.

However, there has been so little research attention given to the effect of occupational exposures on the fetus, that we don't explicitly know what threshold levels to set to assure safety.

There has been concern that the levels which would be required

to protect highly susceptible groups would be significantly lower than those levels otherwise acceptable, and that these new levels may be, at least for the time being, technologically or economically infeasible.

When we consider alternatives to engineering exposures down to a safe level for pregnant women, the most obvious alternatives run you smack into potential problems with the Equal Employment Opportunity Act.

An alternative that was chosen by the Nuclear Regulatory Commission in order to deal with the problem of radiation exposure for pregnant women, was to fully advise the women at the time of employment of the hazards of the job, and to encourage them to turn themselves in when they knew they were pregnant and negotiate for a temporary job transfer. Or, the guidelines said, they might consider simply quitting their job if transfer was infeasible.

This alternative raises serious questions which are applicable to almost any conceivable administrative alternative.

- Does a woman who accepts a job in the face of a declared hazard waive her right to compensation if she's injured in the course of employment? One would think not from the simple examples of industrial injuries where workers are compensated for injuries on machines long recognized as hazardous. Compensation is essentially no-fault. I should mention, by the way, that at the present, workers' compensation is not federally standardized. It's controlled by state legislation on a state by state basis.
- Can a woman sign away the rights of her unborn child to sue for what's been termed "industrial malpractice" if he or she suffers birth defects as a result of the mother's exposure to hazardous substances on the job?
- Wouldn't requiring a woman to turn herself in be in violation of the Equal Employment Opportunity Act, because it would lead to discriminatory treatment based on her sex?
- If the woman were transferred, at protected pay and seniority, into another job, could a man making less pay than she at the same job sue for equal pay?
- If the woman quits her job because of her pregnancy, is this discrimination because the employer refused to afford a safe workplace to a certain class of people of one sex?
- How do you deal with the problem of damage to the fetus during the first four to six weeks before the woman determines she is pregnant?

In a 1974 memo on the Nuclear Regulatory Commission's proposed guidelines, the Justice Department raised the issue of "whether the government has any interest in protecting the health of the fetus in the first trimester of pregnancy or before the onset of pregnancy that would override a woman's right to privacy and her right to equal employment opportunity".

They also stated that "... the Supreme Court decisions in the abortion cases preclude government restrictions on the rights of women for purposes of protecting the fetus in the first (and second) trimester of pregnancy".

All I have done for you is throw out more problems. There obviously is not agreement on how we are going to deal with this issue. There are no legislative precedents which instruct us on how to deal with this complex tangle. The laws on the books do not provide a clear solution. In fact, they could in practice seem to engender conflicting responsibilities for the protection of women.

I would be less than candid if I did not admit that it is not at all clear what a successful legislative solution to this dilemma is, or even if it lends itself to legislative solution.

We need continued and intensive research to more clearly define the issues and the hazards. Meanwhile, however, legislators and administrators must sometimes move ahead of science to address a problem for which the best available evidence presents only a partial solution.

I can only add, by way of encouragement, that public awareness and additional information generated by conferences like this can bring legislative attention to such an issue.

Thank you.

DISCUSSION

Dr. Samuel Milham, Jr., Supervisor, Non-Communicable Diseases Unit
Washington State Department of
Social and Health Services

Dr. Bertram Carnow, Professor and Director
Occupational and Environmental Medicine
University of Illinois
School of Public Health

DR. MILHAM: I'd like to bring you some good news. I don't think things are as bad or as alarming as we may have been led to believe, at least from my experience in this business of teratogenesis and structural defects. It's obvious over the last 50 or 60 years that more and more women are entering the workplace. I think it's also obvious that more and more chemicals, especially toxic ones, have been added to the workplace. Yet, against this background, I'd like to make some very simple points.

Over the last 50 years, the miscarriage rate in this country has stayed essentially stable. The central nervous system defect rate, that is, anencephaly and spina bifida, has shown a very striking precipitous drop, starting about 1910. And, similarly, the fetal death rate in this population has come down considerably in the last 30 years. So, if these chemicals are detrimentally affecting the fetuses of women exposed in the workplace, in terms of structural defects, we don't have any general evidence for the whole population.

I'm certain that there are pockets of problems, and I'm certain there are infants who are defective today as a result of their mothers' exposures. But just common sense and intuition tells me from the time trends that the problem is probably not general and it's probably not severe.

I was very interested in Dr. Edmonds' paper this morning. He looked at the relationship of vinyl chloride to central nervous system defects. I looked at these defects in New York State, in a large population, and there we found that the really critical variables were social class. We found that in most towns, the low social class areas were those that had the highest defects rates, and that there was a strong racial relationship, in that the non-whites had very low rates. So, this is a complicated business and I'd heartily recommend that anybody who performs community studies of defects related to, say, air pollutants do his studies very carefully, and do them with good controls.

I just can't emphasize this too much. Take the first paper, the paper given by Dr. Corbett about anesthetists and operating room exposures. One thing that bothers me is the mechanism by which

an exposure to a father -- say, a male anesthetist -- can cause a structural defect in his child. One obvious suggestion was that of mutagenesis -- a permanent change in the genes, or the genetic makeup of the father, which could be passed on. If this is the case, you'd expect that as men age, through the reproductive years, their load of mutations would increase. The older you are, the more likely you are to be exposed to mutagenesis. Once a mutation has happened, it's there.

The fact of the matter is, in no structural defect that I'm aware of is there an independent father's age effect. Take monogolism, for example, and the striking relationship to the mother's age. None of the other structural defects that I'm aware of have an independent contribution based on the father's age. So, I think this is indirect evidence that if mutational events are happening, the father's genes are not being permanently damaged. With that bit of good news, I'd like to conclude my discussion.

DR. CARNOW: We're dealing with a number of serious questions. One is the whole question of the myth of female inadequacy. I think we heard very little here to suggest that anybody really believes that women are inferior to men in almost any aspect with regard to activities in the workplace. That is, non-pregnant women.

I think we're also concerned here with the special problem of the pregnant woman. And this is, indeed, a problem. You heard the last speaker deal with some of the legal complexities of this problem; the fact that a woman carries another life within her; and the whole question of the protection of that life, and its rights.

I think that we've been preoccupied over the years, however, with the question of women in high risk because of fertility, which is not the same as a fetus being affected after it's there.

As a clinician, early in my career I was faced with couples coming in and saying, "Well, the wife can't get pregnant. Check her out." And when I would suggest a sperm count, the male would say, "Who, me?" And in more than 50 percent of the cases, the "Who, me" was the one who was at fault.

I think that down through the years there has been this concept: you remove women because they're of childbearing age. Now, if we do remove women from known teratogens, as was suggested by one speaker, and if what Dr. Wagoner says about vinyl chloride is true, and if what the Soviets say is true, and if it is a fact that women married to lead workers have abnormal births, then we also have to remove the men, as was pointed out. And we'll either wind up with 60-year-old eunuchs in the workplace, or we'll have to eliminate the teratogens. I suggest that it would be easier to eliminate the teratogens.

What we are really talking about is how to define a safe

workplace, and how to make it safe so that everybody can earn a living. Otherwise, what happens is that after the women, we eliminate the bronchitics, we eliminate the asthmatics, we eliminate everybody with a G6PD deficiency, and we eliminate everybody with an alpha one antitrypsin deficiency. I could go on and on because I have been looking at high-risk populations for the last 15 years, and I can tell you that there are many of them.

I think that when looking at high-risk populations it is important that we do not study just a survival population, which might result in a workplace standard that relates to the strongest individuals in our society. I think we must do adequate studies so that when we formulate a standard, we can formulate it in a way that our concern will be with the pregnant woman and not with anybody else. And I think that many of the standards in the workplace are going to require very serious reassessment in order to do this.

For example, everybody talks about old people being affected by air pollutants. Well, we did a study of octogenarians in Chicago. We did pulmonary function on them. We measured levels in the air and so on. And we found that these people did not get sick when the pollution levels were high. And the reason was that they had lived in Chicago for 70 years and had survived all of this.

When you look at this population, you find that they never smoked, that they had no respiratory infections, that they came from families that never had any problems, that there was no history of asthma, and so on. In short, you're talking about a survival population. And if we had structured air pollution levels to conform to this population, we would have had serious problems, indeed.

Let me just note, for example, the CDC study. Now this is a very careful study, and I don't purport to be able to analyze it just from hearing it once. I would like to see the data on the CDC study. However, the fact is that there were virtually no air quality measurements. So we really don't know what the environmental levels were. I think there was only one measurement or very few measurements.

The fact is that there was a relationship between the high level of VC and abnormal births within three miles, and the fact that there were high particulate levels in the southwest, as compared to the northeast, may have related to the fact that the particulate did not come from the VC plant, but came from another plant. Unless I misunderstood what was said, my impression was that there was a significant relationship between VC and abnormal births within three miles, in a northeast segment, to people. And that the one level of VC that was measured was high in this segment.

By the way, I would suggest that even if this study is valid and there is no relationship in the community between this structural deformity and VC, this does not in any way absolve VC. Perhaps

those people with the structural deformity, those fetuses, died. So, you'll have to look at the structural deformities in relation to numbers of abortions in the community, which I did not hear in the study.

I think that when we do these studies, they have to be done with extraordinary care because the stakes are extraordinarily high. Again, I'm not faulting the study. I think that it represents a very serious and important effort. I'm only suggesting that we do need a lot more environmental measurements in the plants and out of the plants.

We do have to define all of the populations at high risk. We do have to carry out studies which will then look at the level at which these populations are affected. As I said before, there is no threshold for most of these things. At every level of environmental contaminants -- in the plant or out of the plant -- someone may be affected. So what we're really talking about when we do careful studies -- and this, I think, is one of the approaches we can take to this problem -- is that we have to look for what are socially acceptable risks. We have to look at dose response relationships and try to come up with some level in the workplace and in the community which will protect virtually all of the population.

If we do this, then we can look at the question of the pregnant woman as a very special case, and make every attempt to protect her without sacrificing her economic status and so on. I think we'll have more to say about that kind of thing in discussion, but I feel that this really is what we're trying to grapple with today and tomorrow.

QUESTIONS, ANSWERS AND COMMENTS

DR. CLARK COOPER: Along with Dr. Carnow, I think we need epidemiological studies concurrent with the actions that we are going to have to take. We have found it very difficult to develop designs for studies that we feel are satisfactory, and I am particularly interested in Dr. Wagoner's report of fetal deaths associated with male exposures to VCM, because this really has been one of the most startling reports that we have had. It was given to us in a very abbreviated form and I am very interested in just how the study was handled, and how data was obtained.

DR. WAGONER: This was a survey in which both male employees exposed to vinyl chloride and unexposed rubber workers in the same facility were questioned concerning the reproductive histories of their spouses. The data were analyzed for potential interviewer bias, and none was found. The data were analyzed, as I indicated in the presentation, with regard to those individuals having two or more miscarriages, three or more miscarriages, or four or more miscarriages.

ages, in order to eliminate potential bias resulting from chronic aborters unassociated with the place of employment.

The complete methodology of the study is included in the paper which was recently published in The Lancet Magazine, where it is available for peer review.

DR. PETER INFANTE: I am also concerned about the results of the CDC study of vinyl chloride, and about the clustering on the north-west side of the plant. I believe the wind direction was predominantly from the southwest in this part of the country.

In fact, recent studies by the Environmental Protection Agency, in which they tagged chlordane isotopes and spread them for agricultural purposes in Dallas, resulted in this chlordane being detected in Cincinnati's water supply. For this reason, I think the predominant direction of the wind is to the southwest, and that is why I'm a little concerned when I see the clustering near the plant, within the three-mile limit on the north side of the plant.

Also, I am concerned about the case control method for the study -- looking at distances from the plant -- given the mobility of the working population. For example, a worker who doesn't work in the plant can work next to the plant but live ten miles away. Should a birth defect or some other anomaly be related to this, it would show up ten miles away when, in fact, the worker spends eight hours a day closer to the plant.

Again, I think these are questions that need to be resolved. In addition, the method of selection in this study requires examination. They looked at 17 communities, with vinyl chloride polymerization facilities, and four of them had a significant excess of central nervous system anomalies.

Now, at the statistically acceptable five percent level, you would expect five out of 100 communities to show significant excess, and yet they found four out of seventeen with a significant excess of central nervous system anomalies. This is of further concern because in two of the communities, the monitoring system ascertained less than 50 percent of the births. Yet they were still able to identify a significant excess of central nervous system anomalies.

I have some information concerning birth defects in a community that was not in the CDC monitoring program. The data was received from the Michigan Department of Health, and it shows the incidence of birth defects in Midland County, Michigan, where there is a large industrial chemical complex, in relation to the rate for the remainder of the state. During the five-year period, 1970-1974, there was an incidence of 21.3 birth defects per thousand live births. That's compared to 9.5 for the state as a whole. So,

again, we see a repeated pattern of birth defects in areas with vinyl chloride polymerization facilities.

As I said a year ago, this certainly does not mean that these observations are causally related to vinyl chloride. But as data continues to come in, I am becoming more concerned about it.

DR. FINKLEA: Thank you very much. I think maybe Dr. Edmonds might want to make a reply. I might add one thing that would be helpful to you. The Environmental Protection Agency did publish a monograph on the air pollution in the Kanawha Valley. And I think if you will look at that, maybe elevation might be just as important here as a location for a plant. That valley acts as a trough and you have frequent inversions, and you have a lot of almost slushing back and forth in that trough. And your air pollution there should follow a little bit different pattern than you would see if your topography were quite level.

MR. EDMONDS: I would just like to make a few comments. First of all, somebody made a comment earlier to the effect that these studies found no association with vinyl chloride. In fact, these studies cannot rule out such an association.

The first thing we compiled was wind data. Dr. Infante said that the predominant winds were out of the southwest. Kanawha Valley is a deep valley. The very top of the valley is an airport, and wind direction at that airport is out of the southwest. Down in the valley there is a different situation. The wind direction is different during the time of day, and time of year. In addition, you cannot ascribe any one wind direction to any one part of the city. We did try to ascertain wind direction at the time of conception for both cases and controls.

The wind blew a little more out of the southwest than out of the northeast -- about 42 percent of the time versus 37 percent of the time. So the difference is very minimal. And difficult to assess. There is very little information on environmental pollutants in the valley. There are a lot of chemicals. I mentioned the VC levels on one day they measured. That one day's measurements were the only levels we could find in Kanawha Valley. The levels were a little bit higher to the northeast, with the winds out of the southwest.

The particulate and settleable matter I told you about has been measured over several years. These measurements are higher to the southwest, and could be reflecting emissions from other industries in the general vicinity.

The environmental and atmospheric data were very confusing. You cannot pinpoint wind direction to any one part of the county. As for selection of the counties, four counties with the highest rates were identified out of the seventeen. Two of them were

eliminated. In one of them, only three percent of the births were monitored. The other county that was eliminated was a county where 38 percent of the births were monitored between 1970 and 1974, but where the VC plant was not in operation until late 1972. So that community was eliminated, which leaves the two counties of Painesville and Kanawha.

The next comment I would like to make is about the high rates in Midland. I would be interested in knowing what types of defects they are.

DR. INFANTE: The data from Midland County, Michigan, show total defects. With regard to congenital anomalies, thirty-four urogenital defects were observed, whereas only seven were expected; twelve heart anomalies were observed, whereas only four were expected; and fifteen cleft lip and palate defects were observed, whereas five were expected. These observations are highly significant statistically.

MR. EDMONDS: We need to monitor these counties. In Kanawha Valley, we are going to evaluate all birth certificates back to 1958. We still have a concern.

DR. INFANTE: Well, the point I was trying to make is that the population study conducted in the Ohio communities with vinyl chloride polymerization facilities demonstrated a significant excess of birth defects. This study did not conclude that the birth defects were related to vinyl chloride. On the other hand, your study does not rule out the possibility.

DR. GORDON: You cannot rule it out.

DR. INFANTE: In fact, I am more concerned now in that you identify four out of seventeen counties showing a significant excess. To me that is a significant observation.

MS. CLARA SCHIFFER: I am interested in the technologies that are necessary to reduce hazards, and I have two questions. First, I would like to ask Dr. Corbett how much it costs to install a scavenger in an operating room and properly vent it? And, secondly, I know Dr. Gordon was asked to speak on placement and not on what Dow does. But I want to take advantage of his being here to ask him what technological changes Dow is putting in to reduce some of these hazards?

DR. CORBETT: The answer to the first question is that it costs approximately \$100 to install the scavenger device on an operating room anesthesia machine. That is, if the existing remainder of the equipment is suitable for removing the gases from the room. In other words, does the operating room ventilation system carry the gases directly outside, as opposed to recirculating the air? If air is recirculated, an alternate form of exhausting is necessary.

This can be done through wall suction devices if they are adequate. If either of these two systems is not adequate, then an entire new exhaust system must be installed, which is more expensive.

However, in the majority of cases, one can get away with about \$100. In 1974, approximately 20 percent of operating rooms had a scavenger. A number of hospitals have installed these devices since that time, but I don't know exactly what percentage at the present time. It is around 40 or 50 percent.

DR. GORDON: I have been given the opportunity to talk about an excellent program that I think one chemical industry has in controlling environmental hazards. In Midland, the Dow Chemical Company has what I consider one of the finest toxicological laboratories in industry. And this is, to some extent, duplicated in our Texas area.

Many of these problems are currently being studied in animals. In addition to toxicological studies, epidemiological approaches have been taken at most of our manufacturing installations. And, above all, every attempt is being made to reduce work exposures, and fence line exposures, to the lowest possible limit.

This is being accomplished and it's being checked by a staff of excellent industrial hygienists who are the envy of the country. Indeed, many of them have been hired away by industry in other locations, by government, and by academia.

I think that a great deal is being done and I heartily recommend that other industries follow suit. One other thought here concerns what we call "product stewardship". I believe that this is very important. What it means is that every manufacturer should take responsibility for problems downstream in the user.

Now, for one pesticide that has been referred to, we feel it is important for the company to identify problems to the ultimate user, and to tell the pest control operators how to monitor for this substance, both atmospherically and in the individuals. It is required that they attend to this identification of hazards before they are sold the pesticide. And, furthermore, this is monitored from time to time by the company.

DR. FINKLEA: It is also my understanding that if you have a contractual arrangement, and if it is broken by your formulating customer, the contract is removed, and you don't sell him any more. I don't think that's the usual practice now in industry, but I think it's a very responsible one. Dr. Carnow wanted to make one comment about the anesthesiology department.

DR. CARNOW: Just one quick comment. You talked about scavenging operating rooms, and I think that's good. Every once in a while, however, we miss populations. There are a large number of dental

hygienists and dental nurses in offices, for example, who are exposed under awful conditions, both to radiation and to nitrous oxide. I think that we have to look at these people.

This also is a problem, by the way, in small plants. The ones that are not monitored; the ones where nobody knows what's going on.

DR. FINKLEA: I think Pat Breslin, of NIOSH, has been working with the dental hygienists and the dental association on some early studies in this area.

MS. SCHIFFER: I don't know much about nitrous oxide, but at one point I checked with the District of Columbia concerning the X-ray machines of dentists. We had two X-ray technicians to inspect all the X-ray machines in the District of Columbia, which meant that they got to hospitals about once a year, but rarely got to dentists and physicians.

MR. BARRY CASTLEMAN: I am a consultant to the Natural Resources Defense Council, and other environmental groups. There has been a lot of controversy over the cost of meeting regulations. As we find in things like vinyl chloride, there have been enormous controversies over the cost of meeting the OSHA standards.

To give an example, the asbestos textile industry is well on its way to producing in Brazil, Taiwan and Spain. And I am very concerned about the flight of these industries, the economic incentives to move plants to these other countries. My question to the gentleman from Dow is simply this: Does Dow have a formal policy of extending the same degree of protection to workers in all its plants, everywhere in the world? Or are Korean, Brazilian, Mexican, Venezuelan, and Taiwan workers considered less susceptible than U.S. workers to the hazards of chemicals?

DR. GORDON: The answer is, yes. Emphatically, yes. We have standards, and we have had them for many years. Many of them are more stringent than the local standards of the United States or the country in which we have manufacturing installations. The rule is that we abide by the local standards or the Dow standards, whichever are most stringent.

DR. RIEKE: I have two questions, one for Dr. Gordon and one for Ms. Taylor. I have wondered for years, because of the fact that we really have great concern about chlorine and chlorine-containing compounds, what you can tell us about chlorine, as such, or fluorene as such, particularly chlorine, as it might relate to the type of discussion we have today. Have you been able to draw any conclusions about the fertile grouping, or is that something that remains to be studied? And Ms. Taylor, let me congratulate you for your presentation on a very difficult subject -- the laws and their conflicts. We are much aware in the west of violent attacks on

OSHA in the Congress and in the newspapers -- the Wall Street Journal -- and elsewhere. We are very much troubled by accusations about the heritage of the law, the constitutionality of it, the administration of it, and so on. But is the Senate, in your judgment, going to hold the line and get more money needed for studies and needed education for employers and workers? Or are they going to buckle under to the pressure?

DR. GORDON: Regretfully, we have not done studies on birth defects that could result from exposure to chlorine-containing compounds, or to chlorine itself. I believe that chlorine is a ubiquitous material in drinking water, and that it is a natural constituent of the body fluids in the form of electrolytes. It would be very difficult to study this as an entity. But the answer to your question is that we have not studied this specifically, as it relates to birth defects.

MS. TAYLOR: Let me try to tie the question into the subject of the issue. I certainly can't say whether the Senate is going to buckle under this year.

One of our main problems has been with the controversy over OSHA's extending its regulations and so forth to small business. And there is very little understanding in the general public, perhaps among some portions of the Congress, of what hazards exist of any sort, particularly health hazards, in small businesses.

I am sure that the words "mutagen" and "teratogen" are foreign. Last year, the Senate voted an exemption for small businesses having three or less employees from inspections under the Act. Fortunately, the House did not pass such an exemption and it was dropped. But until there is sufficient public understanding of the hazards that exist in all workplaces, it is going to be a continuing battle to maintain the strength of the Act.

Let me say one more thing. There are a lot of people in this room who have information that needs to be disseminated much more broadly. That would bring a greater understanding of the need for this Act.

MS. MAUREEN J. O'BERG: I am from Du Pont Company and I have some particular concerns about the policy regarding the conditions of workers being exposed to known human teratogens and experimental teratogens. I wondered how many workers had actually been excluded from jobs because of the policy, and how many have signed this statement to which he referred, indicating acknowledgment of the possibility of a teratogenic problem? And, also, when was your policy adopted, please?

DR. GORDON: The answer is that I don't think anyone has been excluded as a result of the possibility of being exposed to a known human teratogen. None that I know of.

Your second question is how many people have signed a statement? I have really been talking about guidelines that are in the process of being evolved. In actuality, all of our employees are required to be present at a safety presentation wherein they are given plant rules, safeguards and the possibility of hazards. There they are given a booklet in which there is a page they are required to sign, which states that they have, indeed, been present at the presentation and that they have been given the booklet. In order to get these signatures, the Safety Department people who make the presentations actually have to do the work they are supposed to do. Now, insofar as signing that they have been informed about known human teratogens, to the best of my knowledge, we've had no exposures at this time.

DR. TURSHEN: I am from the Institute of Policy Studies in Washington. I would like to ask a question of Mr. Edmonds. Would you please clarify the residential and occupational histories with the cases of birth defects in Painesville, Ohio. I understand from Dr. Infante's study that there are three polyvinyl chloride plants in northeastern Ohio, which are quite close together, and that many people live in one town and work in another. Did CDC investigate whether the parents of the birth defects cases who do not work in Painesville, in fact, work in some other PVC plant, and has he plotted not just the residential histories of people but their occupational histories in relation to the three plants?

MR. EDMONDS: Yes, we did both. In both studies, we plotted the location of work and compared the cases and controls, and saw no difference. Does that answer the question? We did look at the work. There was no difference. As for the first part of the question, the people did not work in any polyvinyl plant in Painesville or in any of the surrounding cities.

MS. KATHY HUNNINEN: I am with Tennessee OSHA. One area I think we should probably look at a little bit more in depth is the behavioral effects that have been touched on, particularly in the presentation of mercury. Some of the evidence of animal studies show that because of the immaturity of developing nerve cells even in the late stages of pregnancy, the fetus is very susceptible to subtle behavioral effects that might not even show up until five or ten years later.

And I have a question for Dr. Corbett. I saw a reference to a study on synergistic effects that might occur from radiation and anesthesia. It seems such an obvious thing to look at, because women who work around anesthetics are often exposed to radiation. This was mentioned earlier by Dr. Carnow. And I was wondering if anybody has studied this, or if you plan on looking at it, and if so, what would be some of the mechanisms that might be an effect of these conditions.

DR. CORBETT: I am not aware of any studies that have been performed

on the synergistic effect of radiation and the anesthetics. It is something we probably should look into. To my knowledge, however, these studies have not been done.

MS. JOYCE LEADER: I work with the McGraw Hill Newsletter that deals with chemicals. My question is for Sylvia Krekel. I am interested in what sorts of documentation you might have concerning the extent to which the administrative practices you mentioned are now being used. That is, removal from a job, or having to sign something in specific instances, or documentation. Do you know of any? And also do you know of any legal action that might have been brought as a result of birth defects that are occupationally related?

MS. KREKEL: I don't have any statistics on this. I think our legislative director is here. He may know of specific instances where administrative action has been taken. Tony Mazzocchi, do you want to answer that?

MR. MAZZOCCHI: I think the question and the answer raises a more fundamental question. The fact is, we have taken no action, simply because we don't know what to do. We don't know what the facts are. There isn't a single worker, including those at Dow, who knows what the hell he works with in the first place. He is never given a choice about what he may be confronted with. Let the worker know what he is subjected to. Choices will grow out of awareness.

No industry has allowed us to look at monitoring data. The oil and petro-chemical industry has decided as a result of collective bargaining to allow us to receive mortality data only going back to 1969 and 1970. I'm not a scientist, but I ask those of you who are scientists, what good will that data do workers in the petro-chemical industry today?

So I think the only answer to the question is that we don't know. We're not suggesting that people be removed from the workplace. Ms. Krekel announced very forcefully that our position is to make the workplace safe for everyone. We don't want to be confronted with Hobson's choice because workers have to eat every day.

For workers to voluntarily remove themselves from the workplace subjects them to the other peril of not being able to feed their families. We can give you many specific instances of that type of choice. Rather than correct the situation, allow the person to quit. We aren't being told what is carcinogenic, or what is teratogenic. We are learning after the fact.

And those of us who have the responsibility to convey the facts to the workers are walking into the workplace gagged, or with our hands tied behind us. We're not allowed to receive the type of information that will allow us to carry out our legal responsibility.

Forget the morality part. I wouldn't dirty the conversation with morality, when it comes to occupational health.

We're not even allowed to pursue the legal obligation of saying that in order to properly represent people in the workplace, we must know what affects them. There isn't a single industry in this country which is submitting that data to us. And I think that any conference such as this should stand for the right to know, and for the right of individuals to be able to make decisions based on knowledge.

I think the type of paternalism that I heard from the Dow representative is the very thing we take issue with. We know what is good for you. We will tell you when something is wrong. Well, we are reading a lot of death certificates out of Dow and other companies which tell us that something is very wrong.

DR. FINKLEA: We wouldn't want Tony to leave the federal government out of the picture. He's been maybe a little too kind to us. In recent hearings by one of the House Committees on the issue he's describing -- namely, the responsibility of the employer to know the exposures his employees may be undertaking, and the right of the employee to know what he or she is being exposed to -- it was pointed out that we've had requirements for federal contracting activities and General Services Administration and Department of Defense and other agencies to make sure that the workers in companies that receive these contracts do, indeed, have this information. And we haven't done our job.

I think there's a lot of progress that needs to be made in these areas. I know Harold is on the grill up here, and I wouldn't want him to be on there by himself. Harold, did you want to offer a comment or two?

DR. GORDON: What can I say?

DR. FINKLEA: You could say "amen".

MS. LEIZA ZADEL: I speak as a nurse and as a woman of childbearing age. As a nurse, I work at Children's Hospital, in Boston, on an orthopedic ward. I work with many children who have the birth defects we have been talking about all day.

As a woman and a worker, I speak as someone who would like to have children some day, and I'm concerned about my own environmental and occupational exposure. Now we have heard all day long about the excellent studies that have been performed. But I think we have been a little bit too polite and a little bit too theoretical. I think experimentation is excellent. But while experimentation is going on, we cannot forget that women are still working and still bearing children. We need to talk more specifically about what are we going to do now for the women who are bearing children now.

DR. FINKLEA: If I might answer that, or give you some information. I think the Department of Health, Education and Welfare and the Department of Labor are trying to move to meet this, along with the people in Labor and our friends in industry. I might mention that all the recommended health standards that we are forwarding from HEW to the Department of Labor have addressed whatever information that is available in these areas for about the last nine months. It's a late beginning, but it is a beginning.

Both the Appropriations Committees and the Congress and the public health services have recommended accelerated gathering of information and testing of materials so we can amend previous health standards that are supposedly now enforced in the workplace.

The Department of Labor is trying to address this issue in its standards dealing with metals. We'll be addressing it in the recommended standards for some of the halogenated solvents you've heard about today, for pesticides and in a criteria document that will recommend controls for anesthetic gases.

So I think we are trying to move. I think there is a lag time. Government is very ponderous. And we can't assure the complete degree of protection people might desire. But I think our goal is to maintain equal job opportunity and the least possible risk to public health, and we're doing our best to try to get there. I wish we were getting there faster.

MR. CHRIS WISE: I am with the U. S. Chamber of Commerce. I'd like to address my question to Peggy Taylor. What are the chances of the On-site Consultation, or S. 3182, coming to the Senate floor? And if it doesn't come to the floor, is there any specific reason?

MS. TAYLOR: The Chamber is well aware that we are holding oversight hearings and legislative hearings to consider 3182 along with other bills. The Senate Labor Committee is in the process of holding legislative hearings on S. 3182, and any other bills that are before the Committee. I really can't predict until the hearing record is complete how the Committee will act on the bill, or whether it will be put out to the floor.

UNIDENTIFIED PERSON: I am with the Benzoid Company. We are concerned with the exposure of chemicals to both male and female workers. My question is how do we treat the female worker who is pregnant and exposed to heavy physical labor?

DR. FINKLEA: Perhaps I could offer a comment, and then maybe Hal Gordon would like to comment also. The American Occupational Medical Association and the American College of Obstetrics and Gynecology is addressing the issue you mentioned, and should have a report out in the next few months. They are going to try to have guidelines that would address some aspects of physical activity during normal pregnancy. I don't think there is any plan for

any federal regulatory effort in this area at this time. And it's my understanding that the advice given varies with the industrial establishment at the present time. So, it's a matter of medical judgment, with relation to particular jobs.

DR. GORDON: In the past, I think we've over-protected women, both before pregnancy and during pregnancy. In my company and in most installations, this is left entirely up to the female employee who is pregnant, and her attending physician. If he says that she can work and if we're sure he knows what her job entails and what the problems are, we allow the matter to be decided between him and her.

MR. DAVID ERICKSON: I am from the CDC in Atlanta. A question for Larry Edmonds. Were most of the seventeen counties which have VC polymerization facilities east of the Mississippi?

MR. EDMONDS: Most of them were. That's where the majority of the counties in which we monitored birth defects were also.

QUESTION: Wouldn't it be surprising, considering the east-west gradient in central nervous system defect rates, if there were not higher rates in the east than in the west? And would you comment on the birth defect rates in the period that preceded the lowering of vinyl chloride emissions?

MR. EDMONDS: Okay. Some additional environmental data that I did not cover in the talk relates to vinyl chloride emissions provided by the plant in South Charleston. An interesting point is that the levels of vinyl chloride emission began to drop in 1974.

From 1967 to 1973, vinyl chloride emissions ranged from 235 to 270 pounds per hour. In 1974, the level went down to 180. In 1975, it was 76 pounds per hour.

If you remember, we talked about the rates in Kanawha County birth defects. The rates of birth defects began to decline before the vinyl chloride emission rates declined. This preceded the decline in VCM emissions by more than a year.

MS. MADELYN GOLDMAN: I am working in a tube factory. I do a lot of work with asbestos. About a month after I was working in that factory, I began to read about asbestos and I realized it was dangerous. And yet nobody has said anything to me.

Now my plant considers itself an enlightened plant. It has industrial hygienists, it has a health and safety officer; some of the same things that the man from Dow Chemical was talking about. And to me that is just a smoke screen for really what happens and what workers have to deal with.

For example, we brought in OSHA to check on the asbestos.

Before that, the plant was doing some work toward improving some of the asbestos dust levels. I thought things were really going to improve once we got OSHA in, but then we found that the asbestos dust levels were for the most part below or within the permissible levels.

So the company doesn't have to do a thing now. We just continue to be exposed. And everybody knows that the asbestos standard as set by the federal government at this point is not a safe standard. People in that plant have been exposed to asbestos dust for 35 years, and, of course, the regulations are not going to include or cover anything that happened to those people.

I have to agree with Mr. Mazzocchi that the only way it appears to me that we're going to be able to change any of these conditions is when the workers themselves have some control over the regulations and the health and safety of the plants.

DR. INFANTE: I want to respond to the gentleman from CDC who just made the comment that he's not too concerned that four out of seventeen communities had significant excess of central nervous system anomalies because most of the communities were east of the Mississippi. I think it's our responsibility to find out -- if that is, indeed, the case -- why there is an excess east of the Mississippi and not just accept high levels as the norm, and discount the observation.

MR. EDMONDS: Dr. Infante, we are not discounting these levels. At the present time, these levels are not high. They are what are expected. However, we will monitor these counties and watch them.

MR. WALTER LEAR: I am from the Pennsylvania Department of Health. I want to follow up a little bit on the comment of the nurse from Boston, and also on the comment of the worker about what should be done now.

For a number of years, people in the health field who have social commitments and progressive political views have become increasingly concerned about occupational health problems. For example, the Medical Committee for Human Rights several years ago established an occupational health project. And now, throughout the country and in Philadelphia, we have an occupational safety and health project, which is a coalition of workers and progressive health workers.

I believe that this kind of citizen action, which uses professional knowledge and organizational skill, is our best hope for making immediate goals on these very serious problems.

SESSION III

OPEN FORUM FOR WORKING MEN AND WOMEN-- WHAT ARE YOUR CONCERNS?

MODERATORS:

Dr. Gloria C. Gordon
Research Associate
Department of Psychology
St. Louis University

Ms. Andrea Hricko
Health Coordinator
Labor Occupational Health Program
University of California--Berkeley

DR. GORDON: I am welcoming you on behalf of the Task Force on Occupational Health and Safety with the Coalition of Labor Union Women to this open forum for working women and men. I am a psychologist concerned with occupational health and well-being. I belong to the Office of Professional Employees International Union, Local 13 of St. Louis, Missouri, and I belong to the St. Louis Chapter of the Coalition of Labor Union Women.

Our purpose this evening is to encourage the direct dialogue between our sisters and brothers, who face daily job health hazards, and experts in the field of occupational health, who are here with us. We all have questions to ask one another and information to give one another, to raise our knowledge and raise our consciousness about job health problems.

To kick off the discussion, we will hear from several rank and file women workers. We realize that we may be starting out with a one-sided presentation, but we hope that by giving the workers a running start, so to speak, we will achieve a well-balanced dialogue in this session.

For those of you who may not know about CLUW, it is a membership organization of women who belong to labor unions. It was founded two years ago in March, 1974, and now has a membership of about 5,000 persons in chapters all over the country. The goals of CLUW include encouraging women to participate more actively in their unions, working for equal rights on the job, pressing for legislation on women's issues such as day care, and helping to organize more women workers.

CLUW has appointed a Task Force on Occupational Health and Safety within the past couple of weeks. About six or eight participants in this conference are among those who have just been appointed to the task force, including Andrea Hricko and myself.

The task force hopes to help our chapters develop active programs concerning job health. Many of us believe that working women, armed with good information, may well take on a large part of the struggle for job health in the workplace just as women have traditionally been the health care providers and decision makers for the family.

I would like to turn the program over to Andrea Hricko, who will make further introductions and moderate the discussion.

Andrea is a staff member of the Labor Occupational Health Program at the University of California at Berkeley. She is co-author of a new handbook for women workers entitled "Working for Your Life", a women's guide to job health hazards. She belongs to the American Federation of Teachers Labor Education, Local 189, and to the San Francisco chapter of CLUW.

MS. HRICKO: First, this evening we are going to have a panel discussion by the group of women workers, who are seated at the podium. We will then turn to any other workers who are in the audience, who would like to identify themselves, and just let us know the types of work that they do. In other words, as the discussion goes on this evening, we would like to have people from the audience talk about hazards that they are facing at their own jobs so that we can discuss these hazards with scientists, government, industry people, who are also represented here.

On the far left is Jeanne Reilly. Jeanne is a member of the International Federation of Professional and Technical Engineers, Local Number 57, in Niagara Falls, New York. She works in a laboratory of a chemical plant.

Seated to her right is Leiza Zadel. Leiza is from Boston, Massachusetts, and is a nurse who works in a pediatrics ward of a hospital that is not organized.

To her right is Myrtis Frazier. Myrtis is with UAW Local Number 659 in Flint, Michigan. She works in a plastics factory.

Karen Osborne is next. Karen is a member of UAW Local Number 696 in Dayton, Ohio. She works in an auto parts manufacturing plant.

Mary McDaniel comes out of UE Local 1012 in Ontario, California. She is General Vice President of United Electrical Workers and works in an electrical manufacturing plant.

Sheri Stephen is a member of UAW Local 659 in Flint, Michigan, and works in an auto frame and stamping plant.

MS. STEPHEN: We do a lot of arc welding at the plant I work in. We make frames for trucks, Cadillacs, and small and large Chevro-

lets. Until recently we didn't have any ventilation at all in the welding area. Now we have hood-type deals. However, we need more of them. They are not adequate. People are passing out because of fumes from welding.

We also have an area where we spray zinc coating on the inside of the fenders. It runs through a small room probably 20 feet by 10 feet.

We have had people pass out in there from dizziness and people with headaches. The company says it is not harmful to us in any way. This situation doesn't only affect people at the ends of the lines, but people who work to the side. When the pipefitters go in to clean the area, they leave the doors open. Windows are frequently broken out and we have a problem with getting them repaired. There is also oil on the floors.

MS. OSBORNE: Our plant has a lot of machining and a lot of oil, and the oil lays on the floor. A janitor attempts to mop it up, but the cleaning fluids he uses are not adequate to pick up the oil deposits. The situation has become very hazardous. We can't get the company to get the proper equipment to scrub the floors and pick up all this oil that is laying around. If you have any overflow of hydramation fluid you then get a very slippery and slick area. In fact, it is almost impossible to walk in certain areas of the plant.

But my main complaint in our plant is the asbestos. Our plant is one of the largest makers of disc brake pads, and in the areas where they mix and form and cure and grind the asbestos, there is inadequate ventilation. They have an overhead ventilation system that supposedly picks up loose particles in the air over the machinery, but when this system breaks down, the guys have to keep right on working. The company doesn't care. They want their production out. The guy standing there, he has a respirator on. It is about 140 degrees, and all this asbestos is floating around in the air, and you can see a haze over the whole department where this work is done, and anybody walking through the area gets asbestos particles on their clothing.

The guys or the women who work in the department have no provisions for coveralls or any sort of shop-supplied uniform. So these people who are working in this asbestos take their clothes home or wear them home, where their families are also exposed to the asbestos dust. And that is pretty much the problem. OSHA hasn't seemed to have done much about it. They have come in and they have fined the company, but we haven't seen any improvements at all.

MS. HRICKO: It appears that asbestos is still a very common problem in the workplace. I think, Mary, you told us about some asbestos problems at your plant?

MS. MC DANIEL: In the Ontario plant they used asbestos for a total of about 40 years. One day, all of a sudden, the company sends out a couple of health specialists, and they discontinue the use of the cord that they were putting on the irons. We make clothing irons in my plant.

So we became suspicious. We sent off a sample to find out exactly what was in this cord, and we discovered it was asbestos. So we started checking into asbestos. It is a very scary thing, especially for workers in the shop, and especially when you are working with it without knowledge. I worked for the General Electric Company for 18 and a half years, and year after year asbestos fuzz would be floating over the plant. You would have a cup of coffee. The white fuzz, you would just wipe it to the side, and you drink the coffee. The people in my plant are paying for that now. Our union has done many in-depth studies. We are trying to determine the amount of damage that the asbestos has done.

We have also uncovered a variety of other damages that the employees have suffered due to other chemicals in the plant, such as degreasing fluids, acids, and plating fluids. The variety of problems that have now surfaced makes our plant almost seem like a bomb.

And the only reason we know about all this is because we started checking out asbestos, and we opened up a whole new world of industrial injury and damage that was done to the employees, not only our plant, but I am sure in most of the industrial manufacturing plants in this country.

It seems like a stone wall when you really try to do something. I know it is very discouraging, as an officer of the local, to try to negotiate something with the company, when you can say, "Well, the damage is done." But what happens to the people who have been severely disabled?

And when you try to use the laws, you find they are very insufficient. You find out that Workmens' Compensation coverage isn't quite as good as you thought it might be, because there is no guarantee there that you might have lifetime medical coverage.

And I think this is a major problem that we people in the plants need to address ourselves to, and that we need to collectively, all of us participating in this conference, try to work out.

MS. HRICKO: It may seem incredible to think that workers are working every day with chemicals or substances, but with no knowledge of their chemical structure. Perhaps Jeanne can explain to us what problems she comes across in her laboratory in terms of really understanding what the chemical substances are.

MS. REILLY: I work at the Hooker Chemical Corporation in Niagara

Falls, which is a wholly-owned subsidiary of Occidental. We are a basic chemical industry plant. We don't make any products that are sold directly to consumers; our products are sold to other chemical industries.

In our laboratory, we test all incoming material that is used in the manufacturing process. We test all intermediary sampling for process quality control. We also certify any final shipments that go out of the laboratory plant.

There are over 110 chemicals made currently at the Niagara plant complex, which is the largest chemical plant complex in New York State. It covers 365 acres in the city of Niagara Falls, and if you have ever visited there, then you have smelled the Hooker Chemical Corporation.

Hooker has several major product areas. There is the caustic soda with the Hooker-type "S" diaphragm cell. Mercury cell potash is also made there, which is why the EPA constantly monitors for mercury pollution in the upper Niagara River.

We make quite a few chlorinated hydrocarbon compounds. We make C56 -- a trademark name -- which is what they make kepone from. We used to make Mirex. We make many compounds whose chemical formulation the company will not tell to the employees. The company simply tells them it is hazardous. The employees were told that lindane was bad for them. They weren't told that it was extremely poisonous, and that it was absorbed by the skin. The company simply said, "Well, we sell it to pharmaceutical companies." My understanding is that pharmaceutical companies make it to help those poor people who are infected with body lice because it kills the lice. In large amounts it kills the person.

Hooker was fined \$900 this Tuesday by OSHA. And do you know why? Because there were four men killed there, in December, by an explosion in a chlorine residue collecting system. There were 90 people from the surrounding area who were hospitalized with chlorine inhalation, who didn't know what it was. Many people unfortunately never went to the hospitals. They only suffered with it.

The main concern is that although there are simple procedures for preventing exposure to some of the compounds you are handling, they don't tell you, and they don't instruct you.

One has to threaten to go to a third party, either through a union contract, through arbitration, or to one of the federal or state agencies in order to get information. The people in management forget that when they say they can't build a feasible ventilation system in the works laboratory where I am, because the building is old, and because it is difficult to balance with hoods, they make the worker expendable. Everyone at Hooker knows that chlorine will kill you, but the thing that bothers the majority of workers

at Hooker is not the mass chlorine escapes. It is the 15 or 20-minute light gases that you get when a fan goes down over one of the cells, and a waft of chlorine floats across the area, and, because of the ventilation system in the laboratory, is picked up and brought into the laboratory. We act somewhat as a scrubber for some of the other areas in the plant.

And they tell you, "Well, it is not going to hurt you because it is only 15 minutes." But your eyes hurt. You can taste it. And you might have a headache the next day. A lot of the people find that they are extremely irritable because of the chemicals they are working with. And no one tells you if they know of any synergistic effects. No one tells you that if you are working with carbon tetrachloride you shouldn't have been out drinking the night before.

And the failure of management to instruct the employee, and then to turn around and say that the employee is too dumb is just the failure of supposedly intelligent people to use their intelligence.

MS. HRICKO: Thank you, Jeanne. Myrtis, perhaps you can explain whether there are similar kinds of problems in your plant in terms of obtaining information about the kinds of chemicals or substances with which you work.

MS. FRAZIER: In my plant, we make plastic grills for cars and trucks. I had a problem that I took to management. I had a respiratory problem with fiberglass. I had to take parts out of a machine. After they came out, I had to stack them, trim them, and grind up the scrap that was left over. In doing this, I inhaled some of the fumes and some of the particles, and I could hardly breathe.

I was told that the man before me didn't have the problem I had; that he hadn't had any kind of breaking out or any type of a rash; and that because I was a woman I was supposed to be more durable. I was supposed to set a good example. That's a quote, unquote, from my supervisor.

Someone has to listen because no one comes into those shops and sees the condition we have to endure. We send representatives to Washington. We have lobbyists. We have legislators. But no one represents us. I shouldn't be here because there is a safety representative who works full-time in my shop. He should be here.

My main point is to let you know that we do have problems, and that we need your help. Don't forget us because we are there. We are behind you, but we need your help.

MS. HRICKO: Thank you Myrtis. As we learn more and more, we are

finding out that it is not just industrial jobs that cause health hazards and health problems. Leiza, would you tell us some of the hazards of your job working in a pediatrics ward of a hospital?

MS. ZADEL: The problems that nurses face are the same problems that anyone working on a hospital floor faces. They are partly problems of radiation.

Supposedly, when an X-ray machine is being used in a hospital room, everyone who has to stay in the room with the patient should wear a lead apron. It doesn't always happen. Sometimes the portable X-rays are done in the hall, and there is no way to shield people walking up and down the hall. So dietary people, house-keeping people, all the nursing staff, nurses aides, R.N.'s, the whole crew -- everyone who happens to be around when the machine is up there is exposed to radiation.

Now the machines are supposedly safe, but you don't really know how much to trust them. I do know that there is a certain radiation hazard for people who work in hospitals. There are also problems associated with shift work; problems of changing back and forth from night to day. I know there are studies done that show this is really hard on your system, and that you can't just continually change back and forth every two weeks to a different shift and maintain any kind of physical and mental capacity. A lot of people have a great deal of difficulty making the change back and forth every couple of weeks or so or however often you are shifted around.

MS. HRICKO: Thank you. Now I would like to turn the discussion over to the audience. I hope we will have a dialogue back and forth between some of the union staff people in the audience, and the industry people, the government people, and the scientists.

Some of these women have asked for help on some of their problems. They are exposed to asbestos, to fiberglass, to various kinds of hazards in hospitals. They are exposed to radiation, zinc, a variety of chemicals, some of which they have the names of, some of which they don't.

Do any of the people in the audience have any ideas on how these women can get help? Do they have any ideas on where these women can turn in terms of solving these problems in their workplace?

MR. WODKA: One of the things I would like to hear more about is the experience you have had with OSHA, either the federal OSHA or the state OSHA. It hasn't come up much at all today. The federal law is supposed to protect us from all of this stuff. So I would like to hear more from the women, who are on the panel, of their experience with OSHA, whether they have gone around with an OSHA inspector, what he has done, what he hasn't done, where OSHA has failed to cite the company, and so on.

MS. HRICKO: Jeanne explained what the fine was at her plant. Would you like to explain what the inspection was like and whether someone from your plant accompanied the inspector?

MS. REILLY: Well, primarily OSHA comes in whenever there is a fatality on the job. It catches their eye. They came to Hooker in late November because there was an apprentice welder killed on the job. He happened to have a piece of angle iron caught on a flywheel on a compressor, and he lost the top half of his head. OSHA came in and investigated the Niagara plant, and they said that in this particular case it was the employee's fault. He failed to lock out the machine with his lock as all the maintenance men are supposed to do. So they fined Hooker for failure to enforce their own safety procedures.

Two weeks later we had the explosion at Hooker where the four people were killed, and the chlorine was spread over the city. OSHA people came in and conducted another major inspection of the plant. They found out that they didn't know what caused the explosion, but they did find out that, once again, Hooker had failed to follow a standard industry procedure for checking pressure releases on containers of chlorine under high pressure. This happened in a stationary tank car that was attached to the building through piping processes. Chlorine was in there, plus several unidentified chemicals, and they don't know what combination of chemicals was in that tank car at that particular time.

They probably never will know what caused the explosion, but they do know that, prior to that time, they had had a lot of complaints from the men who use the 53rd Street locker room, because chlorine was constantly escaping. It wasn't that much chlorine, but on occasion the shift couldn't get in to change out of their work clothes, and they went home because the locker room was filled with chlorine.

From what I have seen, when OSHA comes in, the first thing they look for is the employee's fault. Now, granted, I suppose many times it is, but employees are people, and people have a habit of doing dumb things at times. But what is the point of disciplining an employee, or citing and saying, "This employee made a mistake," without ever getting rid of the factors that led up to the employee making that mistake or that error. I can't see what OSHA accomplishes when they simply cite a company for failure to follow its own procedure. There must be something better than a \$900 fine for four people dead.

MS. HRICKO: Karen, can you explain whether the OSHA inspection at your plant was prompted by a fatality or an employee complaint or whether it was a random inspection?

MS. OSBORNE: Would you believe I really don't know? I know that OSHA has come in several times, and that the company has been fined several times. I guess it is a big joke to the company because

they certainly aren't taking anything serious. You can go into the area that was pointed out in the citation, and you will find exactly the same problem that was there when OSHA came through and fined them.

MS. HRICKO: We turn to the audience now. Are there representatives here from OSHA, who would like to respond to some of the problems that these women have raised about OSHA inspections?

MS. KATHY HUNNINEN: I am a health inspector for the state of Tennessee. I shouldn't be standing up here saying what I am getting ready to say except that I don't disagree with anything that was said on the stage tonight. I came out of the United Paperworkers and was at the CLUW founding convention, and am a very strong supporter of CLUW.

I have a lot of frustrations as an inspector for the state of Tennessee. I have seen almost all of the experiences you all have related. I have been in plants where these same kind of things are going on. I know that citations are being issued. I know the penalty process and I know that fines mean nothing.

I have been in situations where there are serious hazards, like lead poisonings, which have not been given serious citations. But I have to say to the unions that we have a job to do in training and teaching. Some of the unions represented here today know that there is such a thing as occupational health, and some of them have no idea what occupational health is, or what an OSHA inspector is, or what he is going to look for, or what he is going to do. So let's have a little bit of self-criticism here because unions have a job to do in educating their members about what is going to happen when an OSHA inspector comes around. When a citation is issued, what are the unions going to do about following up on them? Let me tell you, you are going to get walked over. You are just going to get walked over until you sit down and go over those citations and review them and be a part of the whole process, because let me tell you, industry is doing it.

What is happening is that industry is contesting. Industry people are tying up the whole court process. They are not having to come into compliance. They get off in these little meetings, and they make decisions with no employee representatives there at all. Lots of times they make decisions about how citations are going to be settled in lieu of fines and of coming into compliance. Or else they are given a long period of time to come into compliance.

MS. HRICKO: Are there any representatives here from NIOSH, who would like to speak to some of the health hazards that these women have raised?

DR. JOSEPH K. WAGONER: We in the Federal Government also have many problems. I think one has to recognize that the system we work in

is not the most conducive to open, free, liberal, or very active speaking to the issues. I would like to give an example. In 1974, the government financed and published an investigation indicating that there were major problems in operating rooms in the United States. Yet here we sit in Washington, D. C., two and a half years later, and we don't have a standard for operating room exposures.

Dr. Corbett referred to the fact that only about 40 percent of our operating rooms are being scavenged at the present time -- two and a half years after knowledge of the hazard. So it seems to me that the voluntary route of resolving these health issues may well be a complete failure, and also that the cumbersome machinery of the Federal Government has imposed upon those of you who work in operating rooms two and a half years of additional hazards. Shouldn't people in the operating rooms stand up and express their moral indignity as to why things are not being done? I don't think we in the Federal Government can assume all of the burden.

With regard to the issuance of standards, I have said it before and I will say it again. Occupational standards are only as good as the paper they are written on unless they are being enforced.

If we look at the history of the Occupational Safety and Health Act, we find that four standards have been promulgated. Each one of them have come by way of a petition, either from the labor movement or from consumer interest groups, for an emergency temporary standard. Four standards in six years, that is a pretty poor track record.

I want to encourage a greater dialogue between employees and people in NIOSH, and I will step forward and offer myself as one whom you can contact to get some help in resolving these issues, because reliance on the standard route has proven to be inadequate. So I extend my commitment to you, and I would appreciate your commitment also. There has got to be a better way.

MS. HRICKO: Frank Wallick has his hand up. He is editor of the UAW Washington report.

MR. WALLICK: We have three UAW members up here and we feel that we have a very democratic and open union. We are very proud of what we negotiated in 1973. In most of our large plants -- this is not necessarily true in some of the smaller plants -- we have full-time health and safety people who are paid by the company, and who represent the union and the workers on health and safety questions.

Our particular union has a full-time professional staff in addition to the health and safety reps on the shop floor. One of them happens to be here. He is a member of the Council of the Society for Occupational Environmental Health. I think it would be very helpful for him to explain how a large and powerful union can

try to deal with these problems, because we don't pretend to think that we have solved every health and safety problem.

I worked here in Washington for many years and didn't even know what health and safety was. Suddenly, one day, lightning struck and I have become a fanatic. This is part of the problem we have in union bureaucracies. We tend to concentrate on wages and hours, and working conditions are problems that are very difficult to get excited about or know what to do about. There is also a great deal of fatalism among people. But I would like Dan MacLeod, if he will, to explain how he responds to a problem, how these problems are initiated, and what he sees as the results of our 1973 negotiations.

MS. HRICKO: Dan MacLeod is with the UAW Safety Office in Detroit.

MR. MAC LEOD: One of the reasons workers have organized into unions throughout history has been to improve working conditions. That is one of the reasons workers still today organize into unions. You can look at the history of the American labor movements and see how certain conditions led to greater efforts toward organization.

You can look at the Triangle Fire, in 1911, when female workers were locked into a garment sweatshop, and the whole place burned down, and they couldn't get out. That created an issue that caused people to organize.

You can look at the Mineworkers' Union, and you'll see similar sorts of things. But the severest problem has come, as you all know, with the new technology, with the chemicals and different processes that may look clean or at least cleaner than they have in the past. I think the union movement has to respond to these new conditions.

Now, one of the things that the UAW has done is create a staff of people with technical training. I, myself, have training in industrial hygiene. My job consists for the most part of doing plant inspections, sometimes through a complaint from a local union that cannot resolve a problem at its own level, and that needs some help. Another way we operate is by selecting target industries, or high hazard industries, for our efforts. So far these have included the foundry industry, in which approximately 100,000 UAW members work, and the lead battery industry.

Just last week, I was in a small plant in Indiana which makes mercury relay switches. If you looked in that plant, it would probably appear cleaner than this room. It has a nice shiny floor, good lighting, and fine equipment. However, there are little pools of mercury everywhere in the plant, and people working very close to that mercury.

When I inspected this plant, I took measurements with a mercury

vapor meter and found that air levels were at about the existing permissible level and also exceeding the existing permissible level.

Now the company didn't know what was going on although they had their own mercury vapor meter. For years the company people had been doing air sampling in that plant, but they were making a mistake in the way they were taking the measurements. The women were leaning over their machines, working just inches away from open mercury, but the company had decided that a good spot to do the sampling was on a bench 20 feet behind them.

Now, that is all right for an area sample, but it is not a breathing zone sample, which is what we need to take. So all those years of data, which the company could point to in their logbook, and inform their workers about by posting it on a bulletin board, did not reflect what those workers were breathing.

Now I talked to some of the people in the plant, and they told me about some of the symptoms they were having. To begin with, they were all missing teeth. Their teeth were falling out and they had problems with their gums. Now that is something which is very consistent with what you would expect from people exposed to mercury. I am no doctor. I am not about to diagnose anybody's problems, but I can tell you they were referring to symptoms that are consistent with what you would expect from overexposure to mercury.

They were also talking about nervous system problems. They were irritable and anxious, and they were on tranquilizers to keep them from becoming ornery and mean. Now, again, those symptoms are what you would expect from people working around mercury. They also talked about thyroid problems, which in some cases have been related to mercury.

Well, through the union we will be able to solve that problem. I mean the engineering controls are not enormous. They can very easily be fixed. The union will be able to provide a program of education and make sure that those controls will be maintained. Further sampling will be done. We will be able to get medical exams for those people. Through the union, you can do it. You can't do anything without the union. There is no other mechanism for improving those conditions.

MS. HRICKO: We have two other union representatives who would like to say something. First, Steve Wodka from the Oil, Chemical and Atomic Workers; then Matt Amberg from IUE.

MR. WODKA: The thing I do for the union is work on health and safety problems and on coordinating our union's efforts in regard to OSHA. I would like to share with you tonight some tactics that work to get plants cleaned up, and to correct some very serious health hazards in the United States.

First of all, there isn't much you can do in a plant that does not have a union. Is it possible for a worker who doesn't have a union to have protection once he starts to speak out about problems? Now every union contract recognizes the union for the purpose of wages, hours, and working conditions. This is very important because we need very sophisticated health and safety language in our contracts in order for conditions to be improved. But a union can't bargain over working conditions unless it knows what those working conditions are.

So all around the country, we have instituted what we call a model grievance or a model opening letter when we begin contract negotiations. Essentially, we ask the company everything. We want to know the common generic name because we have found that you can't get anywhere until the workers know what it is they are working with. In most plants, the most toxic substances are identified by code names or brand names in order to keep the workers in the dark. So that is the first thing you ask for, what are you working with.

Second of all, you ask what are the levels of exposure. A lot of companies have done a lot of monitoring and they have never told our people what the results are. They have kept these results to themselves. And they have also brought in the Workmens' Compensation insurance carrier, who have taken measurements and given the report to the company, but never to our people.

A third thing is to find out what has been the morbidity and mortality experience of the people who have worked in that plant. Every company that has a union contract most likely has a medical insurance plan and a life insurance plan, and the companies collect these records year after year after year. They know who is getting sick. They know what people are getting sick from. They know what the mortality rates are. And any union is entitled to that information. You cannot represent your people unless you have this basic information, and we have yet to be turned down by an arbitrator or by the National Labor Relations Board in every single case where we have requested it.

The companies know that under the National Labor Relations Act, under the Collective Bargaining Agreement, they cannot withhold this information from a union. That is the first place to start, find out what the company knows. When you get that information, then you can start approaching the situation logically. You can go to outside groups, technical people, scientific and medical people, who have the expertise and bring them in and show them these records.

Now one of the things we have been able to do is force the company to allow us to bring in independent people to take readings in the plant in order to find out how bad the levels are because nine times out of ten, the companies lie, they falsify records, and they never tell you what you are really being exposed to. So

if you have an inkling that something is bad, bring someone in from the outside.

I am not talking about the government right now. I am talking about people from universities, some of whom are here in this room tonight, who are willing to work with unions at very reasonable fees and to come into your plants and take readings and whatever.

The same thing goes for the medical end. The most important thing is to determine the medical condition of the people who are working in your plant, and find out who is suffering from disease because many people need medical help right away.

Let me just talk for a minute about OSHA. Now how do you deal with OSHA? Well, there are certain basic things that you do when the OSHA inspector comes into your plant.

First of all, you have the right -- whether it is a state OSHA or federal OSHA or NIOSH inspection -- to be with that government inspector from the minute he crosses the gate. Now this is very important because if you are not with him when he crosses the gate, he is going to sit in the front office and drink coffee and have doughnuts in the morning. In the meantime, the operations of the plant are being cut back or the plant is being cleaned up. So naturally, when the inspector finally arrives there, he isn't going to see conditions that are representative of what they really are.

So you make it clear when you file a complaint that when the inspector crosses the gate, he calls you. Put your phone number in the plant right on the complaint, so he picks you up right away.

The second thing to remember is that the inspector is there for your benefit, not for the company's benefit. You take that inspector around. I have seen too many times when we have filed complaints and our people have let the company lead the inspector around. Well, you have the right to lead that inspector around by the nose, take him around any place you want to, and let him see or talk to any people you think he ought to talk to.

Another thing is this procedure at the end called a closing conference. Under OSHA's stupid rules, if management objects to you sitting in on the closing conference, you can't sit in on it. But you have the right to a separate closing conference, and these conferences are very important because that is when the inspector relates what he has seen and the violations he has seen. That is when he will relate to you, if it is a separate conference, what management has said in terms of how long it will take to abate. And that is your chance to get in there and say, well, that is a crock of you know what, it is not going to take them five months to clean that up. It is only going to take them two weeks. That is your chance to serve as a check on the company.

Now you also have the right to get copies of the citations when they are issued from the OSHA area office. If you have a big plant, you will have a comprehensive OSHA inspection. You can't go running all around the plant trying to pick up where all the citations have been posted because they are supposed to be posted near where the violation occurred. So you should demand your own complete copy and then sit down, review those citations, and compare them with what you complained about. You have to do this right away because there are only 15 days from the point when the citation is issued for you to contest the abatement date.

Now if you are dissatisfied with the citations, there is an appeal type of procedure. It isn't very good, and I am the first to admit it, and you are going to need backup from your international union to go to the OSHA regional director. But there is one thing about these regional directors I have found; a lot of these guys are career-oriented, and they don't like bad publicity.

We were able to get the OSHA regional director for the Dallas region removed. His name was John Barto and he was in charge of the states of Texas, Louisiana, New Mexico, and Oklahoma. There were eight fatalities just among OCAW plants in the first three years of the Act's operation among major oil companies, and not one citation was ever issued. We had enough of that and we went public with it. Our locals fed the information to us. We went public with it, and Barto got dumped.

There was an OSHA area director in Columbus, Ohio, who was doing the same kind of stuff. He was putting out a quota system. He was saying to his inspectors that they could only give out so many citations to any company no matter how many violations there were. He was also withdrawn from his job.

Lastly, another point that is very important. Companies are contesting citations left and right. They are contesting those citations that you have really strived for, the ones that finally deal with a bad situation such as severe asbestos exposure. And when you contest a citation under OSHA, there is no abatement. All abatement is stayed until the case is finally decided. Well, in any federal or state OSHA program, employees have the right to get involved by electing party status. We elect party status in every case that our local unions are involved in.

When you elect party status, you can prevent the kind of thing that often happens right before a trial, when the company goes up to the OSHA attorney, and says, "Now, you have got 100 citations here against us and it is going to take you five weeks to litigate this. Wouldn't you like to drop half of these and we will withdraw our action?" And nine times out of ten, the OSHA attorneys will drop them. However, if you have elected party status, they can't drop anything unless they have the approval of the employees. So it pays to get involved and it pays to follow a number of steps

like this.

UNIDENTIFIED PERSON: I want to make a short statement. I want to say that something has really been bothering me here. At this conference, it seems to me that women have not been seen as human beings. They have been seen as reproducers.

I think there are a number of major problems this conference is not talking about. We are not talking about why are we in this lousy bargaining position? Why are NIOSH and OSHA not doing their job? We are not talking about problems peculiar to women, such as sex-stereotyped jobs.

I would like to respond to the women in the panel because I think they are very brave women to put up with the kinds of conditions they have described. I would like to respond personally and to say that I feel empathy for you. Maybe if we feel some empathy, we can start talking about the problems that are really making working conditions in this country such a mess.

MS. HRICKO: Would any of the women at the table like to respond to that?

MS. FRAZIER: Yes. I came here not knowledgeable of the things that were going on in our great big country, but just curious to know how you scholars and well-educated, well-bred people thought.

I am sitting here and I am ready to cry because we live in America, and hey, that is beautiful, but I am going home, and I am going to fight like hell to replace some of the people who came here, and spoke of things that they don't have any feelings about, and who only gave us statistics, figures, and dates.

That lady, whoever you are, thank you. I am not speaking for myself only, but for the thousands of other women who are facing the same problems I am.

MS. FRANCINE WARTMAN: I am with the Yale Medical School and I would like to respond to the last two women who spoke. I feel both as a woman and as someone who has had diversified working experience that I can be very emphathetic, but I think that emotionalism, while it is appropriate, does not take the place of scientific information, which is what management responds to in part. Management will certainly respond to political pressure and to monetary factors, but they will not respond purely to the emotional aspects, which seem to be very appealing to so many people here. I think you have to keep in mind the balance between the two.

MR. MATT AMBERG: I am a member of the Newspaper Guild. I work for the electrical workers' IUE. I am a member of CLUW. I happen to be the person who drafts the IUE health and safety bulletin.

I want to talk about something that one of our speakers was talking about today. The gentleman from Dow Chemical was telling us about their wonderful program for safeguarding the health of workers at Dow. He was talking about how they are briefing the workers on the hazards that they may face. He also talked about their product stewardship, their concern for the downstream effect, he called it, of the product. I got hold of him later and asked him why they don't put on the barrels and drums containing the chemicals they make, the trade name, the scientific name, and also in plain English that anyone can read, what this chemical can do to you, and what kind of precautions you have to take.

He indicated to me that this would be quite a problem. So, I said I was grateful for the fact that his company was lobbying real hard for big appropriations for OSHA, for NIOSH, for the National Cancer Institute, and also that they were lobbying real hard for the Toxic Substances Control Act. And he said, "You know we are not doing that." I knew it and I want you to know it too.

MR. VERNON ROSE: I am from NIOSH and I am the guy who is responsible for putting out the criteria documents. Before that, I spent a year over at the Department of Labor on assignment and got deeply involved in putting out the 14-carcinogen standards, which was really kind of a landmark activity as far as standards production was concerned under OSHA.

I would like to make one point. Joe Wagoner said very correctly that standards are not the only thing that are going to get the problem solved, but I would like to point out that in my experience and my belief that standards are where we have got to start at.

Whether we are organized or not organized, whether we are female or male, I still believe that the basic thing that transmits research into effective action in the workplace is standards that are based on good data and that are designed to protect the health and safety of the worker.

To me the most important activity we can get involved in is making sure that we get effective standards and then apply them through compliance, education, and joint negotiations between employers and employees.

DR. HARRIET HARDY: I think the last few speakers need to be scolded. They have taken away from the great contributions made by our friends on the panel, whom we should listen to. These people have really done the evening's job and they have done it very, very beautifully, and I scold my masculine friends for their interruptions.

MS. HRICKO: In closing I would like to say that it has become quite clear that occupational health and safety problems of all

workers have generally been ignored in this country, and certainly that the problems of women workers have been given virtually no attention.

What has become clear, particularly in this discussion, is that workers need to be informed. They need to be informed about the chemical substances they are working with. They need to know about the health hazards and the health effects of the chemicals that they are working with. They need to know what kind of scientific studies are being done, and they need to know what these studies are in lay language. It doesn't do them any good to know that something is mutagenic, teratogenic, or carcinogenic if they don't know what those words mean. It is essential for the scientists to put information out in lay language so the workers can understand them.

Workers need to understand how to use OSHA, how to use the grievance procedures, and how to use the collective bargaining. They need to know, as Steve Wodka pointed out, how to get publicity if these other methods that they are trying fail.

Workers need scientists. They need NIOSH. They need OSHA to explain to them how to use the Act. Certainly workers aren't going to learn this information from their employers. That is quite clear.

It is only when workers are informed that they will be able to fight for changes. Knowledge, basically, is power. When these women have facts about the health hazards on their jobs, they will be able to fight for changes that will protect them, their husbands, their co-workers, and all of their future children.

SESSION IV

HEALTH RISKS ASSOCIATED WITH JOB PLACEMENT PATTERNS RELATED TO SEX

MODERATOR: Dr. David Wegman
Assistant Professor of Occupational Medicine
Harvard School of Public Health

Yesterday we talked about the issues that have to do with women as childbearers; today we are going to go into the area of job placement discrimination and we are going to discuss the aspects of discrimination that have affected the health of women in the workplace. Dr. Shirley Conibear is going to set the stage for this discussion with a broad overview.

WOMEN AS A HIGH-RISK POPULATION

Dr. Shirley Conibear
Consultant

University of Illinois School of Public Health

The question of whether or not working women experience greater disease and disability than their male co-workers has great social and economic importance as women move into the work force in greater numbers, work for longer periods of time, and hold jobs of greater and greater variety.

After World War II, women were virtually eliminated from mining, construction, transportation, and all types of basic industry. Now they are re-entering these occupations and are experiencing heavy exposure to a variety of toxic materials, as well as working in physically stressful environments. They are also working without the benefit, questionable as it was, of the so-called protective legislation for women.

Many questions remain unanswered. Do working women need special protection in the workplace? Are all women at an increased risk, or only women with certain exposures, or only fertile women, or only pregnant women? There are many questions, little data, and even fewer clearer answers.

First, it is necessary to define what is meant by a high-risk population. Two types of high risk can be identified. The first type will be called a situational or environmental high-risk population. It consists of a group of workers who are or have been exposed to a stressor known to be harmful, but have not yet developed evidence of the harmful health effects. In this case, the cause of the high-risk effect is the exposure of an unprotected or insufficiently protected worker to a toxic agent. Thus it is the environment which creates the high risk and not anything inherent in the group itself. An example of an environmental high-risk group is workers who have been exposed to high levels of asbestos and thus are at high risk of developing mesothelioma.

It is important to identify high-risk groups of this type so that further exposure can be prevented. Multiple exposure to other types of toxic materials which may have an additive or synergistic effect must also be prevented. Appropriate medical care and close monitoring and follow-up are also necessary.

The second type will be called an intrinsic high-risk population and consists of a group of workers who are more likely than the general population of workers to experience harmful health effects when exposed to an equivalent amount of chemical or physical stressor. The health effect may be of the same type but differ in magnitude when compared with what occurs in the general population, or it may be of an altogether different nature.

This definition includes the toxicologic entities of hypersensitivity, hyperreactivity, individual variation, and allergy. The causes are multiple and all exist within the exposed individual. They include such entities as genetic deficiencies, personal habits, medication, life style, nutritional status, chronic or acute diseases, age, or multiple exposures. Examples of intrinsic high-risk groups include heavy drinkers who are exposed to solvents, and those with kidney disease who are exposed to lead. The approach to protection for this type of high-risk group is similar to that of the environmental high-risk group. The object is to prevent harmful health effects. The methods include setting and achieving environmental standards which take high-risk groups into consideration, providing extra protection such as respirators or other equipment for workers at high risk, and careful environmental and biologic monitoring of the high-risk individual. The high-risk worker must be informed of the nature of his or her situation, warned about the danger of adding additional risk factors such as smoking or drinking, and advised to eliminate or control any risk factors that he or she can. If the combination of exposure and risk factor is potentially life-threatening or severely disabling and the exposure cannot be reduced to a safe level, it may be necessary to remove the worker from the exposure. However, this should be the exception and not the rule.

Women working in industries or types of jobs that, before Title VII of the Civil Rights Act, were known as women's jobs may constitute environmental high-risk groups by virtue of their specific exposures, but not because they, as individuals, are female.

The main issue currently being debated is whether or not working women constitute an intrinsic high-risk group because of certain qualities inherent in being a female in our society today. Many different qualities have been offered as reasons for classifying working women as a high-risk population. This paper will discuss some of the more popular ones.

Some argue that women in general lack the physical strength, stamina, or dexterity to work at many jobs. If placed in these jobs, they might harm themselves by over-exertion, cause accidents endangering themselves and others, or place an additional burden on their male co-workers by asking them for help.

Traditionally, women have been viewed by our society as physically weak and frail. Yet during World War II, women were able to move into all types of jobs without apparent difficulty. One can cite many examples of women who routinely perform strenuous tasks without any problem. Thus, many of the objections would seem to be colored by social prejudice and have very little real data to back them up.

There are some data which suggest that the strength of the

average woman is approximately two-thirds that of the average man. Obviously, then, some women are stronger, whatever that means, than some men. Also obvious is the fact that jobs vary in the amount and type of strength and stamina required to perform them. It therefore makes sense to talk about fitting the strength of the worker to the requirements of the job through ergonomic evaluation rather than to make generalizations about men and women.

Another argument for placing women in an intrinsic high-risk category is that women have a "special susceptibility" to certain toxic substances, lead and benzene being good examples. It is postulated that this special susceptibility may be due to increased absorption, greater storage capacity or differences in organ systems. Whatever the mechanism, it is argued that the biochemical and physiologic makeup of women is significantly different from that of men and predisposes women to harmful health effects.

There is very little scientific data to support this, and again one must be careful not to attribute apparent differences to biochemical mechanisms until social conditions such as poor nutritional status or fatigue have been ruled out. Excluding child-bearing, men and women are more alike than different in terms of biochemical and physiologic processes. Medical science has not found it to be necessary to develop a separate pharmacology or laboratory norms for males and females. The hypothesis of special susceptibility because of sex is an interesting one but so far largely without basis.

Some substances are known to cause genetic defects, and it is felt that if women are allowed to work with a variety of toxins, genetic defects will increase greatly. This is a very important and critical question. But we must not focus only on exposed women workers but be equally concerned with the exposed men. Men and women exposed to a mutagenic agent certainly fit into a situational high-risk group. More detailed study is needed to know whether men and women differ significantly in their response to mutagenic agents.

However, one might postulate that males are more susceptible to mutagens since sperm cells are always rapidly dividing and this is when many mutations occur. Also, the testes are more exposed than the ovaries to hazards such as high temperature which can affect fertility and ionizing radiation which can cause mutations.

Women's life style may differ significantly from men's in certain aspects. Often working women still have primary responsibility for housekeeping and childcare. Thus a working woman's day may be four to six hours longer than a man's, and if she has a sick child or an infant, her working "day" may extend into the night. If she works a night shift, she often still gets up to cook the meals for her family. This places an additional stress

on working women. Here again, there are no hard data to show that these factors justify placing women in the intrinsic or situational high-risk group. However, jobs in which women are required to rotate shifts may pose problems. When average number of days off for illness for men and women are compared, there is little or no difference, depending on which study is quoted. Data on accident rates are difficult to interpret because the women workers are frequently very new on the job and lack experience and skill when compared with men.

Again, in terms of life style, women as a group smoke and drink alcohol in smaller quantities than their male counterparts. This probably decreases their risk of occupational diseases. Many women are using long-term contraception such as birth control pills or IUDs, and this may constitute an intrinsic high risk.

The situation of the pregnant worker deserves special consideration. Marriage no longer means leaving the work force. Women also tend to stay on the job during most of the nine months of their pregnancies, many times out of economic necessity. This means that the fetus is being exposed to a host of toxic materials and possibly stressful situations. There has been no systematic attempt to identify which chemicals cross the placenta, in what concentrations, or what their teratogenic potential is. However, there are a few examples such as prenatal exposure to low levels of radiation, or to therapeutic drugs such as DES, or to thalidomide, which suggest that the fetus may be harmed by concentrations of chemicals that are well within the safe range for adults. The developing fetus has tremendous nutritional needs and is exquisitely sensitive to any lack of necessary nutrients or blockage of their use. Any disruption in cell division, differentiation, or migration is irreversible and is magnified thousands of times in the adult. If the exposure is extremely damaging and occurs early in gestation, the fetus may be too severely damaged to survive, and a miscarriage occurs. Or, the change may be more subtle and consist of such disorders as fewer brain cells, resulting in lower IQ's, or nonspecific damage to the central nervous system resulting in such conditions as hyperactivity. In utero exposure may result in childhood cancers, or genetic damage may occur in the fetus's germ cells, resulting in genetic diseases which manifest themselves several generations later.

At present, the available data are only suggestive of many of the previously mentioned effects. However, the potential harm is so great that the warnings cannot be ignored. There can be no question that the fetus constitutes an intrinsically high-risk population and that adequate protection must be provided.

This brings us again to the problem of how to protect an intrinsic high-risk population. In the case of working women, it is necessary to digress for a moment and consider two important pieces of legislation which have a bearing on this problem. The

Occupational Safety and Health Act states that all workers have the right to a safe and healthful workplace. Title VII of the Civil Rights Act says that women have the right to work at any job, and cannot be excluded on the basis of sex.

Therefore, protection cannot include the blanket exclusion of working women from any industry or job type. Women have been underpaid and kept in low skill jobs with little chance for advancement, and they have been used as a reserve labor force to be moved in and out of the job market as needed. Our society has decided that this is morally wrong and has made such discrimination illegal. To then proceed to use women's reproductive function as an excuse to re-instate discriminatory practice is inexcusable.

In order to protect the pregnant woman and fetus, we must re-examine all of the existing standards in terms of what constitutes a safe exposure level for the pregnant woman. Intensive epidemiology and toxicologic research must be started immediately to further define which substances are mutagenic, teratogenic and carcinogenic for the fetus, and what occupational levels represent an acceptable social risk.

An education program must be begun to inform workers, employers, and health care providers of the hazards. Every effort must be made to reduce or eliminate exposure to known teratogens, carcinogens, and mutagens. Only in this way can working women and their children be assured of a safe work environment.

BIBLIOGRAPHY

1. Equality of Opportunity and Treatment for Women Workers, Report VIII, ILO, Geneva, 1975.
2. Hunt, Vilma R., Occupational Health Problems of Pregnant Women, A Report and Recommendations for the Office of the Secretary, Department of Health, Education and Welfare, Order No. SA-5304-75, April 30, 1975.
3. Revision of the Maternity Protection Convention, 1919 (No. 3), ILO, Geneva, 1952.
4. Rieke, F.E., Thirty-Two Million Women at Work - How Different Are They ?, J. Occup. Med. 15:729-732, Sept., 1973.
5. The War and Women's Employment, Studies and Reports, new series No. 1, ILO, Geneva, 1946.
6. Wegman, David H. (M.D.), Occupational Health Hazards of Women, paper presented at American Association for the Advancement of Science, Annual Meeting, January 28, 1975.

HAZARDS RELATED TO PERSONAL PHYSICAL STRENGTHS

Dr. Don Chaffin
Professor and Director
Occupational Health and Safety Engineering Program
University of Michigan

It is no secret that we all vary considerably in our physical capabilities. The question is what does that mean in terms of occupational health and safety. Does such a difference mean that new rules, policies, and standards are necessary to protect those who are weaker, be they men or women? If so, how much can we afford to redesign jobs, as was mentioned by Dr. Conibear, in order to accommodate all the working population on all of the jobs? Or can we justify, in some way, certain personnel or functional medical tests to protect those who might be at high risk due to low physical capabilities?

If we are to attempt to redesign jobs by standards or some other incentives, we could be setting standards that would prohibit, for instance, lifting more than 25 pounds in close to the body, or even 10 pounds in more awkward positions. That may be a good moral goal, but as a consumer and taxpayer, I have certain questions to ask about that as a way to proceed.

How about determining, then, who is at high risk of injury and illness before they are placed on jobs requiring high levels of physical exertion? This is the attack I would like to discuss with you today.

I am going to talk about strength testing of men and women. I believe that whenever we are talking about functional tests, we have to keep in mind three criteria. One is that the test needs to be repeatable, which means that if it is readministered to a person, the results will come out the same. Secondly, we must have a test that is easily administered and safe. And, thirdly, it certainly must measure an attribute of the individual which is important to the problem of concern, in this case, personal risk.

I think personal strength testing satisfies the first two points. If done in a standard way, it is repeatable. If done in a controlled way, it can also be safe. We have tested over 2,000 people in eight different plant situations and to my knowledge nobody has been injured in the strength testing.

But the third criteria is the one we must look at and consider today. That is, does it measure an attribute which is important? As Dr. Conibear mentioned, it must be an attribute that relates to personal risk.

How does one prove that it might indicate personal risk? Well, first of all, one must build a rationale for such testing

and, secondly, one must prove it with data. We must go into the plants, instigate the testing, look at the situations facing people in the plants, and gather the medical data.

The rationale I would propose here is fairly simple and straightforward. People who habitually perform high strength-requiring activities are the types of people who can tolerate and will tolerate, the physical stresses on their bodies. In fact, stresses help them to adapt to those activities. In other words, people develop both the physical and the psychomotor capabilities to safely handle heavy loads. That adaptation, though, many times is a slow process, and it also depends on certain inherent capabilities of the muscles and the skeletal system.

Now we don't know a great deal about the capabilities of the tissues involved--the tissues that are being stressed in the body when one picks up a load--but we are starting to understand some of them. And what I would like to discuss with you is the way the problem is attacked.

We call this field of study "occupational biomechanics," and it is gradually shedding some light on what happens to tissues when put under load. In this field, we treat the body as a set of links, which grossly correspond to the skeleton. We can determine as a load is lifted what kind of forces are developed at the various joints of these links by applying well-known principles of physics.

When picking up a load, the resulting force acting on the low back becomes one order of concern. If the load is held away from the body because of leverage, it creates a high compressive load on the lower lumbar spine. That compressive load can become quite high.

Before going further though, I must point out one more physiological concept. When anybody picks up a load, the abdominal muscles also contract. There is a reflex that contracts those muscles. The abdominal pressure built up as one picks up a load can be quite high. I just point this out as an indication of further concern for women. Research is definitely needed, I think, to indicate the degree of concern in terms of personal risk to the pregnant woman, who at this point has a different biomechanical structure in the abdomen.

Now let's go on with what happens when we pick up a load.

Let's take an example: 100 pounds held at forearms' length in front of the body generates 1400 to 1500 pounds of compressive force acting on the lower lumbar spine. Don't let anybody convince you that if you pick up 100 pounds, the only load acting on the spine is the 100 pounds plus your body weight. The principle of leverage here indicates that that is not true at all. There is

a very high compressive force that is developed within the spine when lifting.

The question is, is such a high spinal force damaging? A number of studies of cadaver spines indicate, indeed, that compression forces of a magnitude above 400 pounds can be damaging to the spinal column of some people, particularly older women. But such data are not sufficient for selection by themselves. So our question comes back to who is going to have a spine which is susceptible to damaging forces? Unfortunately, that is a question which we must look at only epidemiologically. We cannot put pressure transducers into everybody's spine and measure those forces and their tolerances.

Since the compression forces act on the spinal disks, and in this case primarily the lower lumbar disk, we find out that the high compression forces act in a way that depends greatly, in terms of injury, on whether there is a large pressure-bearing area. One reason I mention this is that certainly anatomical studies have confirmed that on an average the female spinal compression capability and force-bearing area is smaller by about 15 to 20% than that of the average male. But, again, we are dealing with averages that indicate a matter of general risk. There is certainly a large percentage of female spines that are larger than male spines, and I am going to continue on this point when we move to the epidemiological approach which is next.

Since we can't go very far with biomechanics except to indicate that we should be concerned, we have to deal with the real world, the real data, and go out and look at the situations that people have in the plant. We at the University of Michigan have been doing this for the last ten years. We start such studies by going out and taking photographs of people lifting loads. We have done this now on over 1600 jobs in different plants, and we determine what the physical requirement is on those jobs in a very systematic way.

We have what we call a lifting strength rating methodology. It simply uses a measure of that load being lifted by the individual on a job and divides it by what we think a very large, strong male could lift in the same position. We could have used any percentage of the population, male or female, as the denominator of the ratio.

What we end up with is a rating system for a job which, when we compare it to injury and illness data, gives us some interesting statistics that I will get to in a minute. First, let me add that we have done this on a number of jobs. We have developed a distribution of numbers of jobs that we found in five of the study plants to be populated by men, and here we have the situation as of three years ago for women. The results of such a comparison show that there were not very many women performing high lifting

strength-requiring jobs.

Now getting to the injury and illness data, if we follow people medically on jobs that are rated to have various lifting requirements, we begin to see some interesting results. Using low back pain incident rate data for men and women on jobs classified by their lifting strength ratings, we found no difference in the low back incident rate between men and women on jobs that were populated by both men and women. Unfortunately, we did not, as I indicated before, find any women who were even attempting to perform jobs or who were allowed to perform jobs that required a great deal of strength. This was as of three years ago. So at this point we must conclude that the women who were on the jobs that we were studying did not on the average have a higher incident rate of low back pain than the men on those jobs.

The question becomes, however, what about their individual tolerances to such stress? Does strength as a human attribute of an individual indicate the level of personal risk to the individual when performing a physical job?

If you systematically measure strengths of men and women, you quickly conclude that there is a very large overlap between their lifting capabilities. So again talking about averages doesn't help us at all in assigning personal risk of injury.

What we have done then is to classify people in terms of their lifting capabilities and their strengths, rather than whether they are male or female. To do this a strength tester is set up to simulate what is actually required on the job, and the person then performs a number of simulated lifts. These tests are administered in the medical departments of the various plants.

If we now look again at the low back incident rate data, those people who were relatively weak--meaning that the weight they were lifting on their job was greater than what they demonstrated they could do in the systematic test lift--had three times the incident rate of low back pain than those people who demonstrated the capability to lift what was required on their jobs. The point is that strength is the attribute that discriminates here, not sex.

Now just to go one step further with this. What we are talking about is matching the job with the capability of the person; in other words the strength of the person and the physical job requirement. What we would like to do is have people well matched. What unfortunately happens today is that we might have a person who is fairly weak, male or female, being placed on jobs that, in fact, require quite a bit of physical exertion.

In one ongoing study of people who have been mismatched, the severity of musculoskeletal problems was found to be much higher

for those who have been overstressed. This means that the weak person on a job requiring a great deal of strength had much more severe musculoskeletal problems in general than people who were better matched.

The conclusion from all this, I believe, is that strength testing can be effective in detecting the physical attributes of employees which are related to a person's personal risk when performing manual handling of loads. I also believe that by strength testing, for placement purposes, we can achieve a significant effect on reducing the risk of injury not only to women, but also to weaker men who may be placed on jobs requiring a high degree of physical exertion.

HEALTH PROBLEMS IN THE AIRLINE INDUSTRIES

Sunny K. Wofford
Health and Safety Project Coordinator
Association of Flight Attendants

Good morning, ladies and gentlemen. I would like to discuss the health problems facing today's flight attendants. The Association of Flight Attendants, representing 18,000 flight attendants throughout the United States, who are employed by 19 major airlines, has been devoted to occupant safety aboard commercial aircraft for over a decade.

As you may know, the first flight attendant was a graduate nurse named Ellen Church Marshall. The flight attendant's duties in those days consisted of various activities we would find amusing, such as checking floor bolts on the wicker seats, warning passengers not to throw cigar butts out the windows, and dusting the window sills. These flight attendants found themselves sitting in lavatories, closets, aisleways, entrances and galley areas. They were not afforded adequate protection needed to survive impact forces and to carry out duties which they had to perform during emergencies. Not only was their own personal safety often dangerously overlooked and compromised, but their health was also. This is a result of a continued adherence to outdated regulations that were initially promulgated in the interest of the passenger and flight deck crew.

Unlike those first flight attendants, today's flight attendant's duties and responsibilities require the highest degree of training, coordination and communication. Today's flight attendant serves aboard jets which carry over 300 passengers and which travel over 600 miles per hour, often through several time zones; yet few regulations have been revised to encompass the changing working environment of the flight attendant.

Within the last five years, we of the Association have fought long and hard for changes in the regulations to include occupational safety considerations for flight attendants. Regulations have been or are being changed in the areas of flight attendants' garment flammability rules, safer seat belts, and better constructed galley areas. In recent years, problems related to occupational health have become apparent to the flight attendant union. Additionally, there is and has been an increasing concern by airline flight attendants that flight duty coupled with ancillary ground responsibilities induces health deficiencies with long-term effects.

Problems that are due to the newer, expanding, and more demanding duties of the jet age were not experienced by flight attendants in the early years of flying. One very important factor which contributes to the concern among flight attendants

is that today's flight attendant's average tenure is seven years. Flight attendants are reporting health and safety problems to the Association of Flight Attendants. We have corresponded with the airlines to gather information about these flight attendants in conjunction with a health project, but a majority of the airlines did not respond to our letters, and a few airlines stated they simply were not interested in such a project.

Due to the growing concern, the AFA last year conducted a primary health survey which was sent out in the Union news magazine. We wanted to establish if there were in fact problems, and what specific areas they encompassed. The surveys were computerized and analyzed by Georgetown University in Washington, D.C., and we have recently received the final analysis. Many respondents attached personal notes and letters to their questionnaires to express in detail their problems and appreciation for the investigation and interest of the Association. The responses sometimes varied but often suggested important relationships and indicated that many flight attendants are concerned about health and safety problems. Responses were received from flight attendants who had been on the job from four months to twenty-eight years. Based on this study, we plan to do a more scientific and qualitative study with an outside group doing an independent evaluation.

Before I discuss our specific health problems, I would like to discuss our academic problems. Flight attendants are not certificated by the FAA and are not required to undergo yearly physicals, such as the flight deck crew members. Many companies only require an entrance physical. Keep in mind that the average tenure of today's flight attendant is seven years. In corresponding with the airlines the Association of Flight Attendants requested records of injuries and illnesses of flight attendants; however the airlines are not required by the Federal Government to retain records of files on flight attendants' injuries or health difficulties.

In fact, we were informed that no one state or federal agency has jurisdiction over flight attendants. No studies are known to have been initiated by the airlines or by the Federal Government concerning the injury and illness rate of commercial flight attendants. Although the Federal Aviation Agency finally claimed, in 1975, to have jurisdiction over all aspects of flight on commercial aircraft, the Agency has no specific regulations which cover the reporting or documentation of the health and safety of the flight attendant. Flight attendants today await the action that should go hand in hand with FAA's assumption of responsibility for flight attendant health and safety. In short, the Association of Flight Attendants is desperately in need of some means of evaluating the health of its members in order to provide the necessary advice and assistance to them.

The ultimate purpose of the health study is to determine the

nature and extent of flight health problems as a basis for counseling flight attendants and instituting corrective action as necessary. The reported injury rate of flight attendants increased alarmingly over the past three years. In addition, the deterioration of certain health aspects have become more prevalent. This is seen in the increased number of reports and inquiries the AFA has received. These reports are submitted on a voluntary basis either by individuals or by AFA safety representatives. Some of the health problems that have been reported on an unsolicited and unstructured basis by flight attendants range from bladder problems to ulcers. However, over the past decade, despite the growth of aerospace medicine, a relatively small number of studies have been conducted on the psychological and physiological effects of flight duty on commercial flight attendants.

The most outstanding effect of flying has been found to be tiredness and eventual hazardous fatigue. Factors contributing to fatigue are cumulative sleep deficiencies, time changes, physical and mental workload, levels of temperature, humidity, oxygen content, noise, personal discomforts, problems with food and liquid intake, upset circadian rhythm, tension work stress, emotional disturbances and preoccupation. Cumulative sleep deficiency occurs quite frequently in today's jet age. I'm sure that those of you who travel frequently know what "jet-lag" is. In addition, many flight attendants find it difficult to fall asleep. Sixty-five percent of those polled encountered problems of staying asleep. Hotel accommodations for flight attendants are often uncomfortable and noisy, which contributes to sleep deficiencies. Eventually sleep deficiency has a proven effect on the performance and mental state of the flight attendant. Although it has been proven that time zone changes disrupt the circadian rhythm of the body, little is known of the effects of these disruptions other than sleep deprivation and impaired performance. Headaches, tiredness, dulling of alertness, and digestive upsets due to circadian rhythm disruption could have an effect on reaction time and decision making in and out of an emergency situation. In the recent survey analyzed by Georgetown University, 97% of the 1,110 flight attendants who were polled indicated that fatigue contributes to unsafe practices. Body cycles subject to circadian rhythm disruption include the sleep-wakefulness cycle, the heart rate, body temperature, blood pressure, the activities of the liver, kidneys and other glands or organs of the body, as well as the menstrual cycle and performance cycle.

Changes in cabin temperature and humidity have produced problems with dry skin, skin disorders, and mental fatigue. The noise levels along with pressurization lend to mental and physical fatigue. Mental stress is present as a result of the risk element involved in flying, continual passenger contact, the ever present obligation of putting one's best self forward and the regulated working schedule of the flight attendant. Due to these stresses, recovery from flight duty may require from 12 to 48 hours in order

to return to a normal schedule.

Another area of concern is the female menstrual cycle. There seem to be conflicting opinions on this subject, but one thing everyone agrees on is that changes do occur. Some of these changes include length of the cycle, increased swelling of ankles and legs, increased bruising of extremities due to oxygen hunger, abdominal cramps (dysmenorrhea,) hypoglycemia, irritability, and weight fluctuation.

Today's flight attendant has no restriction on age, race, sex or marital status. Flight attendants today have families to support and life styles to contend with. These, together with the health problems I have discussed, play an important part in the total overall picture. Results of the survey show that over 600 respondents checked "yes" when asked if social or domestic problems affected their performance. Sixty-seven percent of the total number of flight attendants questioned responded that they were affected by social and domestic difficulties and that they became depressed "once in awhile" on duty. Sixteen percent suffered occasional depression, and twenty-eight percent experienced no depression.

In the survey, the relationship between the maximum number of hours a respondent could work before tiring and the person's mental condition while flying was one of the strongest examined. As the number of hours a flight attendant could work increased the portion who felt relaxed increased as well. Of those who indicated they could work from four to six hours before exhaustion, twenty-seven percent felt relaxed on duty. Of those who indicated they could work 12 to 14 hours before exhaustion, seventy-five percent felt relaxed in the air.

Often a flight attendant's day will begin at 6 a.m. and end at 6 p.m., sometimes with as many as 13 takeoffs and landings. Flight time duration for flight attendants ranges from 15 minutes to 10 hours. Often the flight attendant is not allotted sufficient time during the duty day to eat a nutritional meal. Quite frequently, flight attendants will eat snacks throughout the day and upon arrival at their final destination may go to bed without eating a full meal at all. Many times restaurants are closed between arrival and departure from their layover hotels.

Of course, there are many things to take into consideration when discussing flight attendant health and safety. Study of applicants for flight attendant positions may be one important factor in the entire picture. What type of person becomes and stays a flight attendant? What are their social habits; are they night or day people; do they want the job because they think it's fun?

We have also been getting many inquiries concerning alcoholism

and pregnancy. There are most certainly conflicting opinions on both subjects. Why are flight attendants turning to alcoholic beverages to relax? Are the pressures greater than we realize? During pregnancy should the flight attendant continue to fly after her second trimester? Are there hazards that have not been studied which may harm mother and child? We are asking ourselves these and many more questions. This is why we feel the flight attendant should have a scientific and qualitative study done on their behalf. We at the Association of Flight Attendants want to better our occupational and environmental health and safety, and we are working actively towards this end.

RESPIRATORY DISEASE PREVALENCE IN BEAUTICIANS AND ITS RELATIONSHIP TO AEROSOL SPRAYS

Dr. Alan Palmer

Support Services Branch

Division of Surveillance, Hazard Evaluations and Field Studies
National Institute for Occupational Safety and Health

A comparative survey was undertaken in Utah to determine if practicing female cosmetologists had an increased prevalence of pulmonary dysfunctions. The survey was prompted by an increasing number of physician case reports indicating that subjects exposed to aerosol hairsprays became ill with a disease known as pulmonary thesaurosis. The paucity of any survey evidence in light of these continuing reports prompted this medical survey to be undertaken to determine if a highly exposed group of people exhibited an increased prevalence of any type of pulmonary disease.

A probability sample of 262 student cosmetologists and 213 graduate cosmetologists from all regions of Utah were medically tested and compared to a non-occupationally exposed control group of 569 people matched by age, smoking histories and region. The medical tests consisted of a posterior-anterior and lateral chest x-ray, the forced expiratory spirogram pulmonary function test, and a medical questionnaire (BMRC). The questionnaire not only elicited symptoms of respiratory disease but occupational history, smoking and allergy history, and quantitated home and work aerosol usage. A 50% systematized subsample received three other tests of respiratory function, namely the closing volume test, the single breath carbon monoxide diffusion test and a sputum cytology test. The major disease categories sought were signs of sarcoidosis (this disease is indistinguishable from thesaurosis), chronic respiratory disease (obstructive and restrictive), and abnormal lung cell pathology.

Analysis of the data demonstrated that cosmetologists have more early chronic obstructive lung disease than the control group. This observation was confirmed through an increased prevalence of borderline and abnormal chronic respiratory disease symptoms, as determined by the questionnaire, supported by increased prevalence of abnormal closing volume values and spirometric terminal flow rate data. (62% of the experimental group had CV and TFR abnormalities with questionnaire findings.)

The combined average prevalence of borderline and abnormal questionnaire categories in the control group (36.9%) compared well with that observed in the general population by Discher, in which a 36.8% prevalence was demonstrated. The 46.7% prevalence seen in the cosmetology industry is notably higher.

Spirometry measurements, between cosmetology groups for measures of large airway patency, i.e., $FEV_{0.5}$ and $FEV_{1.0}$, were not

significantly different ($p > 0.05$), although student cosmetologists exhibited a depressed PF ($p < 0.05$). Other measures of large airway patency as demonstrated by the FEF_{25%} and FEF_{50%} showed that salon employees were significantly better than other groups ($p < 0.05$). The FEF_{75%}, a measure medial airflow, showed that employees of small salons had significantly reduced function ($p < 0.05$). Student cosmetologists demonstrated significantly greater measures of terminal airflow i.e., FEF_{75-100%} ($p < 0.05$). Controls also showed a FEF_{75-100%} nearly equal in magnitude to that of the students.

These findings are important since they demonstrate a continuum of respiratory pathology in the cosmetologist. Depressed values of PF and FEF_{25%} in students probably represent increased airway resistance in the larger airways, perhaps due to the contact of aerosol components on the cilia-lined airways and their subsequent efforts to cleanse the airway causing bronchospasm. Such bronchospasm may be related to the increased prevalence of abnormal closing volumes seen in this group, since bronchospasm can be readily transmitted to the smaller airways by vagal stimulation. Those student cosmetologists who chose to remain in the industry because of a lack of sensitivity to increases in airway resistance or who chose to ignore the subsequent discomfort became hardened (adjusted) and the temporary bronchospasm disappeared with prolonged time in the industry.

Depression of terminal flow rates, i.e., FEF_{75-100%}, in graduate beauticians indicates that due to their extended experience in the cosmetology industry, they have insidious increases of airway resistance in their peripheral airways, a finding consistent with early subclinical obstructive lung disease. This observation cannot be explained by smoking since smoking adjusted data confirm this finding.

Component measurements of the forced expiratory spiogram, when plotted against the number of years worked as a cosmetologist, reveal a specific pattern of impairment with time. There is a statistically significant deterioration of function of measures of medial airflow, i.e., FEF_{75%}, and terminal airflow FEF_{75-100%} ($p < 0.05$).

Experimental studies by Zuskin and Bouhuys (1974) showed depressions of the FEF_{50%} and FEF_{75%} in men and women after acute exposures to hairspray preparations. These flow rate reductions (22% depressions) were short lasting (10-60 minutes) and were elicited by exposures lasting for 20 seconds. Data from this survey generally supported their findings in that increased airway resistance was seen in the small airways, i.e., FEF_{75%} and FEF_{75-100%}; however, the magnitude of change seen was only 10%. These differences may be attributable to the sensitivity of the testing techniques employed by each group. The data collected by Zuskin was obtained using a partial expiratory flow volume curve.

Supporting evidence was sought for the flow rate findings by measurement of the component measurements of the closing volume test. Although none of the data analysis showed any significant differences ($p > 0.05$), the unidirectional trends of the data offer some confirmatory evidence of early lung disease.

Cosmetologists also demonstrated an increased prevalence of sputum atypia as compared to the control group. Although the sputum findings have no known pathologic significance, epidemiologically they do indicate that cosmetologists as a group are definitely removed from normality and moving towards more abnormal categories. Whether or not these changes are a consequence of increased chronic respiratory disease prevalence or reflect early changes such as those found in carcinoma of the lung is not known. The two year follow-up study now in progress will permit this question to be more fully answered. It was noted that graduate cosmetologists demonstrated a higher prevalence of atypia than students, indicating that time in the industry was an important variable ($p < 0.05$).

The major category of disease sought in this survey was that of pulmonary thesaurosis. Since this disease falls within the broad category of restrictive lung disease, a combination of tests were used to more precisely determine its presence. These tests included x-ray evidence of sarcoidosis, a reduced vital capacity, and a reduced diffusion capacity. The presence of an abnormality in any one or more of these tests classified the examinee as a reactor. Prevalence of sarcoid-like disease was demonstrated in graduate cosmetologists to be 1.6 times greater than that seen in controls ($p < 0.05$). Student cosmetologists' rates were not significantly different from the controls. Unfortunately, due to the complex problem of differential diagnosis of thesaurosis, it was recognized that those cases identified as having the sarcoid-thesaurosis syndrome may indeed have some other type of granuloma restrictive disease. (A follow-up study is now in progress whereby each person identified as positive will be comprehensively evaluated in a university medical center pulmonary laboratory). A subsequent report will be issued upon conclusion of this research.

Cosmetologists working in small salons were considered to be a group more likely to have increased prevalence of disease due to the general lack of ventilation in those places of business. In fact, small salons demonstrated higher environmental concentrations of particulates, the highest prevalence of chronic respiratory disease, and the highest prevalence of atypical sputum specimens. This finding was strengthened by the fact that cosmetologists working in small salons smoked only half as much as their peers in large salons or colleges.

A total of 146 examinees (14% of the total study group) reported symptoms that correlated with aerosol use, i.e., wheeze, sputum, cough, and phlegm. In cosmetologists phlegm production

correlated significantly with hairspray use ($R +0.8$) ($p < 0.05$), also with aerosol breath fresheners ($R +0.84$) ($p < 0.05$). Symptom prevalence by group showed that cosmetologists have the highest amount (84%), followed by student cosmetologists (13%) and controls (3%).

Thirty-nine percent of the specific brands of hairspray encountered were shown to be related with chronic respiratory disease (19/49). No brands showed significant relationships to sarcoid symptoms. No significant associations were observed between brand and sputum atypia.

The theory that cosmetologists with a history of allergic disorders might be the group at highest risk of developing the pulmonary thesaurosis syndrome gains little support from this survey. Although allergic cosmetologists have a higher prevalence of sarcoid symptoms and atypical sputum cells, the differences are not statistically significant. However, symptoms of chronic respiratory disease are significantly increased in allergic cosmetologists and controls ($p < 0.05$).

In conclusion this survey demonstrated:

1. Female cosmetologists are at increased risk of developing chronic respiratory disease and atypical sputum cytology which may progress toward more severe changes.
2. The thesaurosis-sarcoid syndrome was demonstrated in 22.5% of the graduate cosmetologists whereas students and controls were not significantly different from each other (12% and 14% respectively).
3. Time in the industry is an important variable in the development of respiratory disease, graduate cosmetologists showing more dysfunction than student cosmetologists. Self-selection of reactor student and graduate cosmetologist is evident, yet offers a degree of health protection, since most of the disease seen is early to moderately advanced as opposed to late non-reversible disease.
4. Those examinees with a history of allergic disorders demonstrated more chronic respiratory disease than their non-allergic peers.
5. Because of marginal ventilation systems found in small salons, the highest concentration of environmental particulate was found in these units. Cosmetologists working in small salons showed increased prevalence of chronic respiratory disease and atypical sputum assays.
6. In addition to receiving more hairspray exposure, cosmetologists were shown to use significantly more beauty aerosols and

household aerosols than their control group. Relationships were shown between environmental particulate concentration, chronic respiratory disease, and the sarcoidosis syndrome.

WOMEN IN THE TEXTILE INDUSTRY

Dr. Kaye H. Kilburn
Department of Medicine
University of Missouri Medical Center

I concluded about five years ago that one of the few things we didn't have to be worried about in textiles was a male-female difference in response. We examined North Carolina textile workers in terms of such factors as males versus females, blacks versus whites, and the non-cigarette smoker versus the cigarette smoker. We examined the effects of age and the age gradient; the cumulative effects of exposure to cotton material over a period of time; and special susceptibilities, particularly those of the asthmatic versus the non-asthmatic worker. We also examined the all-important comparison between natural and synthetic fibers.

I would now like to survey with you some of the health problems in the textile industry, and then look at what is being done in the way of protection, and, finally, share with you the data which leads me to the conclusion that there is no evidence for male-female differences, special susceptibilities, or special resistances in textiles.

Ramazzini, author of the first textbook on occupational medicine, in 1705 (1) wrote that "a foul and poisonous dust flies out from these materials," and "by degrees brings on asthmatic troubles" among flax and hemp carders. Thus the whole problem of textile industry work-related disease is probably as old as the preparation of clothing from vegetable fibers.

There are several sets of health problems in textile workers. First, anyone who has been in a textile mill is aware of the tremendous noise level. The noise level in the spinning areas is around 93 decibels, and in some weaving rooms it peaks at 115 decibels, which, as you know, is well above the acceptable noise level for an eight-hour workday. In fact, peak levels are above the tolerable level. Therefore, it is not surprising that several types of patent headache powders were developed in the textile towns because headache attributed by the workers to noise has been a problem for many years.

The second problem is, of course, byssinosis--the Monday morning chest tightness, shortness of breath, and cough associated with return to the environment of the workplace after an absence of 24 hours or more. This is clearly attributable to the inhalation of respirable dust.

However, there are a number of other respiratory diseases including mill or card room fever, which is characterized by an illness on the first day in the mill consisting of fever, a feeling of illness, and often chills. It may or may not be

accompanied by shortness of breath. (2) Probably related to mill fever is mattress-makers' cough, which is thought to result from the inhalation of fungal spores in cotton stained by the action of various of the *Aspergillus* species of fungi that color the cotton. And there is also weavers' cough, which is attributed to the inhalation of various tamarines and starches used to size fabrics.

Most important of all is chronic bronchitis, which is characterized by chronic cough and the production of sputum with shortness of breath that leads to disability and finally to retirement. So when you study a group of people in the textile industry, you are studying a population that has survived these disorders, a survivorship, if you will.

In addition, there is the "smoke box" that sets prominently in many textile mills. It is called the smoke box because, in order to reduce the fire hazard in a dusty area, a wooden or metal box is the approved special location for workers to smoke cigarettes. This device produces a large amount of secondary smoke inhalation for the person who is smoking within its enclosure.

Other problems in the textile industry that worry both management and the workers are absenteeism, and a large turnover. About 20% of the work force changes six or seven times a year. Finally, there is the susceptibility to accidents and fires for reasons that are easily understood. Weave rooms are particularly at risk because the friction of the apparatus and the presence of tinder, both in terms of fiber and of dust, produce fire hazards. Thus, it is a rare textile mill that gets through a month without at least one fire on a weaving frame.

The cotton plant has a pretty flower. Its hairy seeds which develop in the boll beneath the flower are the source of much of what we used to wear before we decided to invest in petroleum-based, inflammable synthetics such as polyesters. Beneath and partially surrounding the boll of the cotton plant where the seeds are generated are the bracts. The hairs on the seeds, which are composed of cellulose fiber, are made into apparel, padding, and paper. The bract is the source of most of the dust. In the process of harvesting and of ginning, which is removing the seeds from the cotton, fragments of the bract are incorporated with the fiber. It goes to the mill and is cleaned out of the cotton by various processes, which generate the cotton trash into the air as dust.

Raw cotton comes through a cleansing operation, and goes to carding, where it is combed and straightened. It is then drawn out into a fiber. That fiber is further stretched and twisted, finally ending on the spinning frame. It then goes through operations that make it a tighter and tighter thread. Finally, it is ready for either weaving or knitting.

When synthetic fiber is mixed with cotton, it is done in an early operation called blending. In the preparation room about 90% of the workers are male. They "break" cotton from the bale and feed it into the picking machine, which is the first cleansing operation. Blending is the next operation in the preparation area. A carding room contains about 100 carding engines. They convert the laps from the picking or blending operation into clean slivers. The lap from the picker is fed between a continuous belt of steel fingers, which "card" the cotton against a large rotating drum that is covered with similar steel fingers. Again, this is predominantly a male work area. It is the highest dust generation area because of the nature of the operation and the speed with which these machines run--20 to 30 pounds of cotton per hour.

The stationary ring spinning frames, which make cotton thread, are largely manned by women. Although some spinning frame tenders or doffers are males, the workers who stay in this job are basically housewives. With mobile spinning frames the workers stand still, and the spinning frames move past them on a conveyor belt. The spinning tender's job is to repair or put back together threads that break or separate during spinning. Whichever operation it is, the workers either stand still as these machines that need to be tended come by, or they move up and down a long line--it may be 60 to 80 feet in length--checking the thread that is running from the bobbin underneath to the one on top as it is being twisted and spun. Because pay is by output, the pace is fast, breaks infrequent, and fatigue considerable for the spinners. So not only is there a lot of noise and a lot of dust, but there is need for a lot of quick movement.

Now this is not a new problem, as is shown by data that came from Britain and that simulated the studies conducted by Richard Shilling, who fathered modern interest in byssinosis. Let's consider the mortality experience of strippers and grinders. Incidentally, they are not burlesque stars. They are the people who repair carding engines. They strip them, clean them, and sharpen them by stripping and grinding. They have a higher death rate than spinners and weavers, and are much above the standardized rates for males in Britain. They also have a higher sickness rate from bronchitis. In fact, bronchitis rates are highest in strippers and grinders, who are the most heavily exposed to dust. Notice that this data dates from 1920, the time when in the United States the problems of textile workers were attributed by management to the hangovers which they had from drinking Saturday night and all day Sunday, before coming back to work on Monday. But notice that compared to other cohorts--printers and transportation workers--they had a higher bronchitis rate beginning at about age 35. And, finally, textile worker cohorts in Britain's Lancashire mills had reduced ventilatory function similar to what Alan Palmer talked about with the cosmetologists. There was a fall-off of flow rates with age. Between the ages of 40 and 60, there were steeper curves of descent for byssinotic textile workers than for

non-byssinotic workers and controls.

Our early studies in 1970 (3) showed that byssinotics who have occasional symptoms, those who have symptoms every day, and those workers who are asymptomatic during the work shift from 7:00 to 4:00 all had gradual reductions in expiratory flow during their eight hours of textile dust exposure. The thing to appreciate is that even those who don't have symptoms have a reduction in expiratory airflow, which is a convenient function to measure.

This study convinced us that byssinosis was not an allergic or an immunologic disease, but a disease to which everyone was susceptible although a person's susceptibility might vary as human susceptibility does to almost everything. We found that function diminished daily during the work shift, although the decrease was greatest on Monday because recovery was more complete then. (3) In the flax industry exactly the same daily decrease is seen in the Irish workers studied by Carey. (4)

What then is the relation between the respirable dust and byssinosis prevalence? The data for men and women together shows that as the respirable dust level increases, the prevalence of byssinosis in the population goes up in a linear fashion. (5) It goes up much more steeply for current smokers, as our previous speaker alluded to, than it does for those who have never smoked. So there was clear evidence that the most important difference between the people employed in textiles was not sex or color, but was whether they were current cigarette smokers or had never smoked at all.

Examination of similar data for decrease in function (forced expiratory flow) during the workday as a percentage of initial function, when plotted against levels of respirable dust, showed a linear relationship of greater reductions in function as dust levels increased. Thus whether we consider symptoms or functional loss information, the results are the same.

Now, where are women in the textile industry? In particular, where are they in preparation areas, including carding and drawing, yarn-making, spinning, slashing and weaving. Women are predominant in the spinning area. They are rare in the preparation area. And the sex ratio is almost balanced in the weaving rooms.

If one looks at the prevalence of byssinosis in women in mixed cotton and blend mills, there is a cigarette smoking effect that is very definite. Also, as dust levels increase from zero to 0.5 milligrams per cubic meter, to .09 to 0.1 and to .19, there is a definite gradient of byssinotic symptoms with increasing dust level. This correlation doesn't look much different in the weaving areas except there are usually fewer symptoms in weaving at high dust levels that are disproportionate because weavers are exposed to starch sizing which is inert unless it has molds growing in it.

Cigarette smoke exposure and cotton dust exposure are additive in producing chronic bronchitis in both men and women and in the under and over 40 age groups. (6) Thus non-smoking cotton workers have, if they are males, about a 20% incidence of chronic bronchitis. The wool workers who smoke have about 13% and the cotton workers who smoke get up to about 25%.

The situation with females is even more regular. The non-smoking cotton workers have an 8% incidence of bronchitis; the cigarette-smoking wool workers have an incidence of about 12%, and the smoking cotton workers have 26% for a clear additive effect. Why should the female gradient of effect be more distinct than the male? We think the reason is that women smoke about seven cigarettes less a day than men, and that their dust exposure is about four-tenths of a milligram per cubic meter less than men. So in the males we see that a maximal exposure response is achieved with cigarette smoke or cotton alone, and we cannot see a clear additive effect because at any given time 25% of the male population, who either smoke or work in cotton, has chronic bronchitis. Some of them are ill enough to retire.

If we examine length of employment with the decrease in pulmonary function in males, there is a more rapid decrease in those exposed to cotton dust, who have symptoms, versus those without symptoms who show little decrease. Women have a very similar curve with a couple of peculiar things; a decrease and an up ordinate at the end. This is clearly a matter of survivorship in small numbers who have had 40 years of continuous employment in cotton mills.

The lesson to take away from this is that even in the group who remain in employment, there is a decrease in function with time, and that women who are probably less exposed show less effect than men.

We can summarize this another way by looking at the relative risk of disability based on age and adjusted for byssinosis and bronchitis in the cotton mills without incorporating smoke exposure. In this case, you have got about, we observe, a four-fold risk above that expected in non-smoking synthetic and wool workers. And there is a clearly additive effect of cotton dust exposure and smoking.

In summary, the evidence is strong that cotton dust is a health problem. We are still waiting for the adoption of a new cotton dust standard. The data was originally submitted to NIOSH in November of 1971, almost five years ago. It is now in the process still of being argued as to what should be the level of exposure to which workers can be safely employed in industry.

What has been done in terms of protection? Environmental modification, which is probably the best solution, has not been

fully utilized, and it is clear that by better dust control in the entire mill, better air handling, there can be a considerable reduction in the incidence of byssinosis and bronchitis.

It is a different problem in noise. It is practically impossible to make a quiet cotton mill even if you make everything out of nylon, that is, all the machinery out of nylon, gears and so on. So earplugs have been considered to be an effective way of reducing noise exposure where they have been adopted and utilized. Where this policy has been practiced, it has provided a good hearing conservation program. Apparently part of the reason why the worker wears earplugs is he doesn't get a headache, and that is an immediate take-home benefit. So he preserves his hearing by avoiding the headache.

Unfortunately, individual protection for dust is practically impossible to achieve. Any kind of respirator interferes with carrying these large laps of cotton, which weigh around 80 pounds, from the picking engine to the carding engine. The choice then is clearly for atmospheric controls not respirators. The other solution, which is placement by medical examination, is obviously discriminatory. Our evidence would suggest that even the worker who doesn't react violently to dust exposure is still getting a cumulative effect, which will inevitably produce disease if he stays in industry for 40 years.

The other societal response to this has been to increase Workmens' Compensation and to make byssinosis compensable. So far as I know, the only state which has achieved this in any degree is the State of North Carolina where they now are pegging the Workmens' Compensation in the textile industry to the average daily wage scale in industry in North Carolina, not on something like \$40 a week for 80 weeks, which it originally was.

In conclusion, I think we need to consider the food, fiber, and fodder industries, and to realize that so far as health-related problems occur in textiles, there doesn't seem to be a sex difference. It is much more important what age you are, whether you smoke, how long you have been in industry, and whether you are asthmatic than whether you are male or female.

REFERENCES

1. Ramazzini, B., "Diseases of Workers," New York: Hafner Publishing Co., 1964, p. 257.
2. Harris, T.R., Merchant, J.A., Kilburn, K.H. and Hamilton, J.D., Byssinosis (and Respiratory Diseases of Cotton Mill Workers), J. Occup. Med. 14:199-206, 1972.
3. Merchant, J.A., Halprin, G.M., Hudson, A.R., Kilburn, K.H., McKenzie, W.N. Jr., Bermanzohn, P., Hurst, D.J., Hamilton, J.D. and Germino, V.H. Jr., Evaluation before and after exposure, the pattern of physiological response to cotton dust, Ann. N.Y. Acad. Sci. 221:38-43, 1974.
4. Carey, G.C.R., Elwood, P.C., McAulay, I.R., Merrett, J.D. and Pemberton, J., Byssinosis in Flax Workers of Northern Ireland, Belfast H.M.S.O., 1965.
5. Merchant, J.A., Lumsden, J.C., Kilburn, K.H., O'Fallon, W.M., Ujda, J.R., Germino, V.H. and Hamilton, J.D., Dose response studies in cotton textile workers, J. Occup. Med. 15:222-230, 1973.
6. Merchant, J.A., Lumsden, J.C., Kilburn, K.H., O'Fallon, W.M., Ujda, J.R., Germino, V.H. and Hamilton, J.D., An industrial study of the biological effects of cotton dust and cigarette smoke exposure, J. Occup. Med. 15:212-221, 1973.

RADIATION EXPOSURE AND PROTECTION

Vilma R. Hunt

Associate Professor of Environmental Health
Pennsylvania State University

It seems appropriate in a panel such as this for one to examine the process of risk estimation for ionizing radiation and other hazards to which the general population and specific groups of workers are exposed. It is the estimation of risk which in part influences the setting of standards for allowable exposure to hazardous conditions.

As we look at radiation exposure and protection today, particularly in the workplace, the practices and constraints are quite different from those we find associated with other hazards. It would be interesting to know how historians would explain these differences. If my claim has any validity--that the philosophy of protection of the worker from industrial hazards is far from being a unitary concept--can futurists predict with any accuracy the eventual impact of this new phenomenon of radiation on the environment, including the work environment? Will the impact be much different from that of the old familiar dangers and the newer suspected ones?

One reads the biography of Marie Curie and of her death from the effects of radiation when she was 67, forty years after discoveries in the physics and chemistry of radioactivity. Her biographer does not record the effect on her of the medical reports of the mid-twenties describing the prevalence of leukemia and osteogenic sarcoma in the young women who were radium dial painters in New Jersey, Connecticut and Illinois. The reading of the newspaper accounts today of effects of kepone, vinyl chloride, MBK, etc. provides far more restrained accounts than those describing the morale, working conditions and pathologic effects of radium and mesothorium on the radium dial painters, many of them young women in their teens. It was not until the Manhattan Project, under which the atomic bomb was developed, that detail epidemiologic and radiochemical studies were made of the occupational experience of these women. The need in the early Forties was to establish safe working limits for exposure to radioactive isotopes, unfortunately including Polonium-210. I say unfortunately, because Polonium-210 was the first radioactive isotope identified and named by Marie Curie in honor of her native land, Poland - and it was used to trigger the atomic bombs over Nagasaki and Hiroshima.

To my knowledge, the men and women who were exposed to excessive polonium contamination in the Forties under the Manhattan Project were never followed up, although estimates of body burden were made and the health physics experience contributed to subsequent radiation protection reports in the early Fifties. But in the Sixties came the realization that the human and experimental

animal data upon which standards for public protection might be based applied primarily to high exposure doses (i.e., 100 rads and above) when information was really needed on exposures of 0.1 to 0.5 rads. A working assumption developed among organizations concerned with radiation protection that there is a proportional relation between radiation dose and the biological effect, and that the effect would be considered to be independent of the dose rate in the lower ranges of exposure. The implication is that there is no threshold, i.e. any radiation exposure may have a finite possibility of being causally associated with carcinogenic, developmental and/or genetic effects. There is strong evidence to indicate that these conditions are too restrictive, but for a working assumption they should provide a considerable safety margin.

The evidence is quite clear from observations on both human and animal populations that genetic and developmental effects and several kinds of cancer can be produced by high doses of ionizing radiation. As the dose diminishes the number of individuals affected also decreases. And in both epidemiologic studies and laboratory experiments we find that the dose reaches a point below which there are so few identifiable cases that they cannot be differentiated from the background noise.

I have chosen to rapidly scan our experience with ionizing exposure and protection for the past 80 years because the very first damaging effects, in the form of radiation burns, were recognized within months of Roentgen's x-ray apparatus coming into use in the 1890's. Cancer ensued for many, as a German memorial monument attests, commemorating the sacrifice of scientists who died as a result of their investigations of radioactivity, Marie Curie being one of them. Such a selective and limited review is instructive I believe, in any comparison we make with the standard setting procedures for other physical and chemical agents.

It brings us to this past year when the National Council on Radiation Protection and Measurements (a non-profit corporation chartered by Congress) published a report, "Review of the Current State of Radiation Protection Philosophy," which analyzed the reports published since 1970 by the International Commission on Radiological Protection and the National Academy of Sciences of the United States. This is the continuation of a process of re-evaluation which has gone on for more than 30 years.

Whatever one's judgment of the particular standards set and the efficiency and vigor of enforcement of the standards, the process of standard-setting during the 80-odd years since the introduction of ionizing radiation into the workplace bears comparison with what we have been hearing at this conference.

The current guiding principle of the NCRP, which undergoes continual review and which has most strongly influenced the

setting of numerical radiation protection guides or dose limits for occupational exposure, is that the "lowest practicable radiation level" is the concept basic to the establishment of radiation standards. In addition, the assumption is made that radiation health hazards do not have a dose threshold.

In other words, numerical radiation protection guides or dose limits for the exposure of radiation workers are provided only as upper limits, with the expectation that all exposures will be kept to a practicable minimum, far below what is allowable.

As a working philosophy, how does that apply to the fertile woman? Although the larger proportion of fertile women who work in a radiation environment are hospital workers, more women are entering the nuclear industry and we have new occupations appearing, e.g. airline baggage inspection.

The appendix to the National Radiation Commission Regulatory Guide 8.13 applies to workers employed in facilities licensed under the U.S. Atomic Energy Act, and stems from a proposed amendment to section 19.12, 10CFR Part 19. That would require NRC licensees to include instructions to all workers, information about the biological risks to embryos or fetuses exposed to ionizing radiation, and, in addition, to advise women employed in jobs involving radiation exposure that the intent is to minimize exposure to and possible adverse effects on embryos or fetuses. The proposed amendment also states that licensees should make particular efforts to keep the radiation exposure of an embryo or fetus to the very lowest practicable level during the entire gestation period.

This recent concern arises from a recommendation made several years ago by the National Council on Radiation Protection (NCRP) that during the entire gestation period the maximum permissible dose equivalent to the fetus from occupational exposure of the expectant mother should not exceed 0.5 REM, i.e. one-tenth the maximum permissible dose of 5 REM allowed the worker. The comment that went with the recommendation reads in part as follows: "The need to minimize exposure of the embryo and fetus is paramount." It becomes the controlling factor in the occupational exposure of fertile women. In effect, this implies that such women should be employed only in situations where the annual dose accumulation is unlikely to exceed 2 or 3 REMs and is acquired at a more or less steady rate. In such cases, the probability of the dose to a fetus exceeding 0.5 REM before a pregnancy is recognized as negligible. Once a pregnancy is known, the actual approximate dose can be reviewed to see if work can be continued within the framework of the limit set above. The method of application of the recommendation is speculative and needs to be tested for practicality in a wide range of occupational circumstances. For conceptual purposes the chosen dose limit essentially functions to treat the unborn child as a member of the public involuntarily brought into

controlled areas. The NCRP recommends vigorous efforts to keep exposure of an embryo or fetus to the very lowest practicable level.

Rather than pursue the pros and cons of this approach to protection of the fetus right now, I want to pick up the thread of discourse I began with, namely that the extent of the danger which can result from radiation exposure is acknowledged, and that practical means of avoiding exposure to the fetus are being developed. In addition, the central principle of keeping exposure as low as practicable is being emphasized, even if that exposure is already well below the maximum permissible dose.

Now I want to identify some aspects of the radiation experience which might be useful in other occupational settings. What are the current deficiencies in knowledge and practice? Let me consider practice first. Although individual monitoring for radiation exposure has been a regular procedure for many years, it is only very recently that hospitals have started to become more responsible in their checking of exposure records. They are still irresponsible in their lack of instructional programs for employees regarding occupational hazards. The NIOSH study on hospital occupational health services showed that in the hospitals reporting 64% of the small hospitals, 40% of medium-sized hospitals, and 30% of large hospitals had no routine in-service training programs for the control of radiation exposure. These would be institutions which were generally not NRC licensees, but which are now under the jurisdiction of OSHA. Less than 2% of the more than 5,000 hospitals queried replied that pregnancy received any emphasis in their safety and health education programs. Better work practices are going to have to be demanded of health professionals as is currently expected of NRC licensees. Speaking of the hospital setting, it is ironic that there are epidemiologic studies of the longevity, morbidity and mortality of radiologists (usually excluding the few who were female) that have been going on for 20 years--but that there is nary a one on x-ray technicians, nuclear medicine technologists or nurses dealing with radiation therapy. Who knows what their reproductive experience has been these last 30 or 40 years? And how much more efficiently and expeditiously improved exposure standards could have been introduced if these studies had been available?

Now what is the state of our knowledge? The research of the past 60 years shows that there is biological susceptibility in certain population groups to radiation which is seen as a predisposition to the development of cancer. However, the influences that make for differences in susceptibility in humans are little known though the evidence for a relationship with impairment of immune reactions is strong, stronger perhaps than the evidence relating childhood leukemia to radiation exposure in utero at low doses of a few RADs.

The futurist view is that we will move toward a biological and epidemiological understanding of cancer susceptibility, including particular subgroups in the workplace. The real practical usefulness of "low as practicable" should become apparent more quickly and directly as more hazardous substances for which "zero exposure" is necessary come to be identified.

In addition to considerations of potential carcinogenicity, hazardous substances are now being tested for mutagenicity. The experience with ionizing radiation goes back to Herman Muller's *Drosophila* experiments of 50 years ago. But estimates of genetic risks in human populations are still based primarily on experimental animal data and the assumptions of the linear hypothesis I mentioned earlier.

Some attempts have been made to examine spermatogenesis under different conditions of radiation exposure--men accidentally exposed and astronauts. A few years ago I thought it would be interesting to measure the extent of radioisotope concentration in human semen and for the purpose obtained over 100 samples of whole semen. Sperm separated from seminal fluid were analyzed for Polonium-210, a naturally occurring radioactive alpha-emitting element which is inhaled in tobacco smoke. The concentrations of Polonium were higher in smokers. Most interestingly, however, Polonium was not in the seminal fluid, but was concentrated in the sperm which had been centrifuged down. It seems reasonable to examine the concentration of other toxic substances in human semen and male gonadal tissue. Reports on the effects of lead on spermatogenesis have recently been made though with no measurements of lead concentrations in the semen or testes. It seems only reasonable that the sperm which fertilizes the egg should undergo some scrutiny.

Another tissue which has been amenable to examination for radiation effects is the placenta. The placental tissue itself is most radio-resistant and it appears that it cannot be sufficiently damaged itself by ionizing radiation so as to be a factor in the development of congenital abnormalities. It is quite difficult to extrapolate placental effects observed in animals to women, but in a study I published about ten years ago I showed that the mouse placenta acted as a barrier to Polonium-210. Other radioisotopes pass more freely (e.g. iodine-131) and in general we are a fair way along in identifying the radiosensitivity of the placenta. However, we now need to identify the critical stages of placental growth, the sensitivity of its various layers and extend our knowledge of the physiology of maternal-fetal exchange.

When we come to evaluate most toxic substances currently in industrial use, we have little to guide us. Does the placenta have detoxifying capacities that offer some degree of protection to the fetus? What is the rate of transfer of a substance in an early pregnancy as opposed to later stages? There is little

reported on the gross or histological features of placental senescence under different conditions of pregnancy. Is the placenta equally resistant to the effects of vibration, or chlorinated hydrocarbons, or mercury as it is to radiation?

For the tens of hundreds of years of exposure to lead, and mercury, much of the memory has been lost and the experience ignored. It could have been put to better use. The continuing experience of women in the workplace has provided the information many times in the past, but discontinuously. The experience with less than 100 years of exposure to ionizing radiation is still far from satisfactory and sometimes far from rational. I only hope that for the older hazards and the new we are now on the right track to realistic evaluation of the hazard, a rational estimate of the risks, and full protection for all those who enter the workplace.

MERCURY: A HEALTH HAZARD ASSOCIATED
WITH EMPLOYMENT OF WOMEN AS DENTAL ASSISTANTS

Meier Schneider, P.E., M.S.
Medical Services Division, Personnel Department
Los Angeles, California

Hector P. Blejer, M.D., D.I.H.
National Institute for Occupational Safety and Health
Occupational Health Program
City of Hope Medical Center
Duarte, California

ABSTRACT

An occupational health survey of 19 dental offices employing a total of 303 dental workers, 177 of them women, was conducted to determine sources of uncontrolled exposure of dental workers to mercury vapor. An additional objective was to assess those job-function worker groups at greatest health risk from occupational handling of and exposure to mercury. Data obtained from wipe, air and urine samples were used to evaluate the degree of the risk due to absorption of mercury vapor for the cohorts studied. As determined by urinary mercury excretion, the group showing the greatest risk of exposure to mercury vapor was that of the 107 dental assistants, all female and most of them young, who prepare mercury amalgams for filling tooth cavities. Further appropriate clinical and other epidemiologic studies appear indicated to determine the extent of chronic toxic effects, including untoward reproductive and genetic changes, among such mercury-exposed young female workers and their progeny.

INTRODUCTION

Approximately 125,000 non-military employees work as chair-side dental assistants in the U.S.A. Most of these workers are young females with the majority in their late teens or twenties. One of the main tasks of the chairside dental assistant is the repetitive preparation of the mercury-silver amalgam material used

This study was part of a project sponsored jointly by the Department of Health Science, California State University, Northridge, and the Occupational Health Section, California State Department of Health.

The opinions expressed herein are those of the authors and not necessarily those of their former or current affiliations.

to restore decayed teeth. For this purpose dentists in the U.S.A. use more than 200,000 pounds of liquid mercury metal every year, which accounts for almost 50% of all the mercury metal used yearly in this country. (1)

Knowledge of the toxicity of mercury in humans dates to ancient Greece. In its elemental liquid state the metal exerts an appreciable vapor pressure at room temperature and consequently vaporizes readily into the ambient, breathable air. Thus the principal type of occupational poisoning by inorganic mercury is produced by chronic inhalation exposure to the vapor. (2-6)

The major organ site of the toxic effects of chronic exposure to mercury vapor is the central nervous system, although the kidney is the critical organ for body accumulation and retention of the absorbed mercury. (7,8)

Classical signs of chronic mercury vapor poisoning include inflammation of the gums and of the mouth accompanied by excessive salivation, metallic taste, marked emotional instability, or erethism, and tremor. Kidney damage, or nephrosis, can occur from chronic mercury absorption. High levels of mercury exposure can produce acute respiratory system effects such as pneumonitis, bronchitis, chest pains, undue shortness of breath and coughing. In the absence of tremor, the onset of symptoms from chronic exposure is insidious since the affected individual may ignore them or attribute them to other causes. (7) A notable example of this lack of any indication of chronic mercury poisoning is a reported fatality of a female dental assistant with a 20-year history of work with dental amalgams containing about 40% mercury. Her death was due to acute renal failure, although apparently there had been no indication of chronic mercury poisoning until she became suddenly ill with vomiting, passing of dark urine, pain in the right flank and swelling of the face and legs. The manner in which her death occurred is considered uncommon for those exposed to mercury vapor. (6)

Metallic mercury injected intraperitoneally into rats has invariably produced sarcomas at the site of injection, i.e., where there was direct tissue contact with the injected metal. However, despite the presence of serious absorptive toxic systemic effects among the test animals, no tumors have been produced elsewhere than at the injection sites. (7)

The present study was begun shortly after publication of reports stating that the suicide rate among dentists is greater than that among other professionals. Because inhalation of mercury vapor is well known for its effect on the central nervous system with subsequent marked alteration of behavior patterns, many dentists in California became concerned and asked the Occupational Health Program of the California State Health Department to evaluate the extent, mode and level of mercury contamination in their

offices. By then a few such evaluations in dental offices had been reported in the literature, but with notable exceptions these reports provided few environmental data on work practices involving mercury handling in dental offices. One exception was the study reported in 1972 which was conducted in Alberta, Canada. (9) Because of the aforementioned concern and the considerable variations in climatic conditions and dental practices which can occur between countries and within a country, it was decided to conduct in Southern California a study aimed primarily at measuring the degree of absorption of mercury vapor among dental office personnel.

METHODS

Initially, the California Dental Association was contacted and appraised of the purpose of the study. The Association gave its support to the survey and publicized it beforehand in its newsletter. Secondly, several forms were developed specifically for use in the survey. These included the general survey data collection form; a self-administered questionnaire; consent form; instructions to the examinee on how to collect the urine specimen; and follow-up letters sent to those study participants whose urine mercury levels were found to be within our protocol's pre-determined normal limits. A different notification letter was sent to those whose urine mercury levels were considered to be above normal. Also, the personal physician designated by the study participant received a letter to aid, where necessary, in the interpretation of the participant's urinary mercury value.

The majority of the 19 dental offices surveyed practiced general dentistry. A few restricted their practice to children's dentistry, or pedodontics, a specialty which uses significantly larger quantities of amalgam than do general dentists.

Urinary mercury was determined from eight-hour samples collected during the work shift. In some cases spot samples were collected where it was impractical to collect a full work-day's voidings. Wipe samples were taken to provide an indication of the extent and amount of surface contamination. Airborne mercury vapor levels were determined with a direct reading "Mercury Sniffer," J/W Bachrach, Model MV-2. Additionally, airborne mercury particulate in the form of finely divided amalgam together with any vapor present was sampled by collecting, absorbing, scrubbing the dental office air through solution of potassium permanganate and sulfuric acid. This solution was subsequently analyzed in the laboratory for total airborne mercury.

The time-weighted average eight-hour work-day concentration recommended by the National Institute for Occupational Safety and Health (7) and also the Threshold Limit Value (TLV) (10) for airborne inorganic mercury are 0.05 milligrams per cubic meter of air (mg/M^3). The current U.S. federal occupational health

(OSHA) standard is 0.1 mg/M^3 as an acceptable ceiling. No acceptable levels for surface contamination have been established.

Study participants were classified according to functions performed in the handling of mercury in the dental practice. So as to facilitate the statistical treatment of the data, urinary concentrations were separated into two arbitrary classes: $30 \text{ } \mu\text{g}$ or less of mercury per liter of urine ($\mu\text{g/l}$), and greater than $30 \text{ } \mu\text{g/l}$. Because wide fluctuations are found in the hourly as well as the daily urinary excretion of mercury by humans, urinary mercury values greater than $30 \text{ } \mu\text{g/l}$ were considered to be definitely increased in that they indicated a three-fold increase over the commonly used upper normal limit of $10 \text{ } \mu\text{g/l}$ per 24-hour volume of urine for non-occupationally exposed people. (11-13)

The p values obtained from the statistical treatment of the urine mercury data are based on the Chi square test with one degree of freedom and the application of the Yate's correction for continuity.

FINDINGS

A total of 303 persons worked in the 19 dental offices surveyed. As shown in Table 1, there were 101 dentists, 107 dental assistants, 13 dental hygienists, nine dental x-ray technicians, 20 dental laboratory technicians, 47 receptionist/clerical personnel and six maintenance staff. Of the 303 workers, 177 were female, which included one dentist and all the dental assistants, hygienists and x-ray technicians as well as the receptionists and office employees. Table 2 shows that the ages of the dental assistants who regularly handled mercury ranged from 18 to 56 years, with a mean of 26.6 years and the largest number in the 18 through 24-year age group.

The range of ambient air mercury concentrations in 17 of the 19 offices surveyed is detailed in Table 3.

The wide variation in surface contamination in the surveyed dental offices is evident in Table 4, where each value represents the average of about six individual wipes.

Urinary mercury levels for the dental office functional groups are summarized in Table 5. A rank ordering of functional groups according to risk of absorbing mercury reveals that dental assistants who handle amalgam have the greatest risk and that non-operatory personnel have the least risk. This is based on the percentage of individuals in each functional group who exhibited urinary mercury levels greater than $30 \text{ } \mu\text{g/l}$.

Table 6 compares urinary mercury levels by functional groups. A significant difference in urinary mercury levels was found when dental operatory personnel were compared against dental

non-operatory personnel; and between dental assistants who handle mercury amalgams and those who do not. No significant difference was found between dentists handling and not handling amalgams, or between all dentists as a group and dental assistants who handle amalgams -- i.e., these assistants and all dentists face a significantly elevated risk ($p < 0.01$) of mercury vapor absorption. Moreover, on a rank order basis, the assistants' risk is the greater.

DISCUSSION

Every dental office surveyed during this study was contaminated with mercury. The level of contamination was governed by a large number of variables such as the number of amalgam restorations prepared per day; the method of mixing amalgam; the care in the handling of mercury and dental amalgam; the type of floor covering and chair covering; housekeeping procedures; and the type of ventilation.

Dental office employees whose functions involve the full-time use of the dental operatory where amalgam is used were more exposed to mercury vapor than those whose primary duties are conducted outside of the dental operatory.

Close observation of procedures in the dental operatory revealed that the chairside dental assistant is momentarily but frequently subjected to concentrations of mercury vapor several times in excess of the current OSHA standard and the excursion allowed by the TLV. This occurs as the plastic capsule containing the amalgam is opened following removal of the capsule from the amalgam mixing machine. Other sources of exposure to mercury vapor by the dental assistant are waste mercury accumulated in the amalgam preparation area, and the squeezing of excess mercury from prepared amalgams.

Given (a) the history of intermittent, brief but severe exposure to mercury vapor; (b) the results of biological monitoring of increased absorption of mercury as shown in urinary mercury concentrations; (c) the long biological half-life of mercury in the human body; and (d) the severe toxic chronic effects of mercury, it is evident that chairside dental assistants constitute an occupational group at greatly increased risk of developing chronic mercury poisoning. This, however, has not been reported previously, possibly because it has not been studied among these workers. The apparent lack of chronic mercurialism among dental assistants in California, the U.S.A. and elsewhere could be or have been due to non-occupational factors such as the transitory nature of this type of employment. Since most of these female workers are quite young, marriage, pregnancy and other sociopersonal factors contribute to a high turnover rate. Thus, this short employment duration would act as a "safety valve" and limit the extent of mercury absorption.

To the authors' knowledge, no epidemiologic or clinical studies have been reported on the chronic toxic effects of such intermittent severe exposures to and absorption of inorganic mercury vapor among dental office personnel and, in particular, among the more severely exposed dental assistants. Moreover, the Minimata tragedy vividly demonstrated the teratogenic nature of organic mercury. (8) Transplacental migration of both organic and inorganic mercury occurs in the pregnant woman who is exposed to and absorbs this element. (14) Moreover, elemental mercury is converted in part by the body to organic mercury, such as methyl mercury. Consequently, appropriate studies of the offspring of female dental assistants, in particular, and of dental personnel of both sexes, including dentists, appear warranted. So do appropriate studies of menstruation, fertility, reproduction, chromosomal intactness, and any other chronic toxic effects which may indicate genetic damage.

CONCLUSIONS

Every dental office surveyed during this study was contaminated with elemental mercury due to poor work practices. This liquid metal, which vaporizes readily at room temperature, produced intermittent, short but gross air mercury concentrations with resultant inhalation exposure to and systemic absorption of the inhaled mercury vapor.

Proper work practices in the handling of elemental mercury in dental offices are mandatory. Such industrial hygiene practice includes education, storage, disposal, ventilation, respiratory protection, use of nonporous work surfaces, and is easily compatible with good dental practice. (15)

Biological monitoring of mercury excreted in the urine of dental office personnel reveals that the functional group at greatest risk of mercury absorption is that of dental assistants, who in this study were mostly all young women.

No significant difference in excreted urinary mercury concentrations was found for dentists who handled mercury as compared with dentists who did not.

Based on the cited findings and on the widespread use of mercury by chairside dental assistants and other dental operator personnel, appropriate studies of chronic damage or dysfunction--such as renal, psychological, menstrual, reproductive, and genetic effects--among this group, and of birth defects among their progeny, appear highly warranted.

REFERENCES AND BIBLIOGRAPHY

1. Mineral facts and problems, 1970 edition. Bureau of Mines Bulletin 650. U.S. Dept. of the Interior, U.S. Government Printing Office, Washington, D.C., 1970.
2. Bidstrup, P. Toxicity of mercury and its compounds. Elsevier monograph 34. Elsevier Publishing Co., New York, 1964.
3. Goldwater, J.J. Occupational exposure to mercury. Harben Lectures 1964. J. Roy. Inst. Public Health 27:279, 1964.
4. Maths, H.B., et al. Maximum allowable concentrations of mercury compounds. Report of an international committee. Arch. Environ. Health 19:891, 1969.
5. Hazards of Mercury. Special report to the Secretary's Pesticide Advisory Committee, U.S. Department of Health, Education and Welfare, 1970. Environ. Res. 4:1, 1971.
6. Cook, T.A. and Yates, P.O. Fatal mercury intoxication in a dental surgery assistant. Br. Dent. J. 127:553, 1969.
7. Criteria for a recommended standard....occupational exposure to inorganic mercury. U.S. Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, Publication No. HSM 73:11024, U.S. Government Printing Office, Washington, D.C., 1973.
8. Bingham, E. Metals seminar keynote address. In Xintaras, et al., eds. Behavioral Toxicology. U.S. Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, Publication No. (NIOSH) 74-126. U.S. Government Printing Office, Washington, D.C., 1974.
9. Buchwald, H. Exposure of dental workers to mercury. Am. Ind. Hyg. Assoc. J. 33:492, 1972.
10. American Conference of Governmental Industrial Hygienists. TLV's--Threshold limit values for chemical substances and physical agents in the workroom environment, with intended changes for 1976. American Conference Governmental Industrial Hygienists, Cincinnati, Ohio, 1976.
11. Jacobs, M.B., Ladd, A.C., and Goldwater, L.J. Absorption and excretion of mercury in man. VI. Significance of mercury in urine. Arch. Environ. Health 9:454, 1964.
12. Molyneux, M.D. Observations on the excretion rate and concentration of mercury in urine. Ann. Occup. Hyg. 9:95, 1966.

13. State of California Department of Public Health, Bureau of Occupational Health. Medical supervision of employees in mercury mines and mills -- Technical bulletin for physicians. California State Department of Public Health, Berkeley, 1967.
14. Tejning, S. Mercury levels in blood corpuscles and in plasma in "normal" mothers and their new-born children. JOM. 15:656, 1973.
15. Schneider, M. An environmental study of mercury contamination in dental offices. JADA, 89:1092, 1974.

TABLE 1

Subjects Studied in 19 Dental Offices
by Functional Group and Sex

<u>Functional Group</u>	<u>N</u>	<u>Female</u>	<u>Male</u>
Dentists	101	1	100
Dental Assistants	107	107	0
Dental Hygienists	13	13	0
Receptionists/Clerical	47	47	0
X-ray Technicians	9	9	0
Lab Technicians	20	0	20
Maintenance	6	0	6
	<hr/>	<hr/>	<hr/>
TOTAL	303	177	126

TABLE 2

Age Distribution of Female Dental
Assistants Handling Mercury

<u>Ages</u>	<u>Number</u>
18 - 24	38
25 - 39	22
40 - 56	7
57+	0
	<hr/>
SUBTOTAL	67*
Not stated	40
	<hr/>
TOTAL	107

*Mean Age = 26.6 years

TABLE 3

Average Ambient Air Concentrations of
Mercury Vapor in 17 Dental Offices Surveyed

Air Concentrations, mgHg/M ³		
<u>Average Ambient</u>	<u>Range</u>	<u>TLV</u>
0.03	0.00-0.16	0.05

TABLE 4

Surface Contamination by Mercury
in 16 Dental Offices Surveyed

<u>Office No.</u>	<u>Average Surface Contamination, ug Hg/100 cm² Surface</u>
1	24
2	557
3	2
4	171
5	13
6	93
7	6
8	247
9	41
10	37
11	2
12	8
13	2
14	21
15	43
16	8

TABLE 5
Urinary Mercury Levels for Functional
Groups in Dental Offices

		Urine mercury levels, ug Hg/liter			
<u>Group</u>	<u>Number</u>	<u>Range</u>	<u>%>30</u>	<u>Mean</u>	<u>SE</u>
Dentists					
Handling mercury	76	2-110	26.3	24.7	20.1
Not handling mercury	25	2-60	20.0	19.3	17.4
Dental Assistants					
Handling mercury	74	2-300	40.6	37.6	44.5
Not handling mercury	33	2-92	12.1	16.8	19.6
Other personnel (non-operatory)	95	2-224	11.1	18.3	29.3

TABLE 6

Comparison of Urinary Mercury Levels
Among Dental Office Personnel

<u>Group</u>	<u>Number</u>		<u>Total</u>	<u>Statistical Significance (p)</u>
	<u>ug Hg/liter</u> <u><30</u>	<u>>30</u>		
Operatory	132	56	188)	
)--	<0.01
Nonoperatory	85	10	95)	
Dentists				
Handling mercury	56	20	76)	
)--	Not significant
Not handling mercury	20	5	25)	
Dental Assistants				
Handling mercury	44	30	74)	
)--	<0.01
Not handling mercury	29	4	33)	
Dentists handling mercury	56	20	76)	
)--	Not significant
Dental Assistants				
handling mercury	44	30	74)	
All Dentists	76	25	101)	
)--	Not significant
Dental Assistants				
handling mercury	44	30	74)	

DISCUSSION

Dr. Marcus B. Bond, Corporate Medical Director
American Telephone & Telegraph Co.

Dr. Janette Sherman, Detroit, Michigan

DR. BOND: Certainly the first speaker, Dr. Conibear, made a point that should be repeatedly emphasized. When we physicians evaluate persons for work and make recommendations about them, we should do it on an individual basis and not as to whether they are males or females.

One other thing that I believe was said was that women and men have about the same amount of sickness absence. That is not true in Bell System. Our women have quite a bit more sickness absence, but this has always been true, and it does not appear to be related to type of job nor, I believe, even to health status.

Dr. Chaffin talked about biomechanics. I think this is a very logical and scientific way to approach the fact that some people, male or female, doing strenuous physical jobs are going to have problems. He did point out something that has been indicated by other studies; women on the average are not as strong physically as men.

Ms. Wofford talked about the flight attendants and described a number of projects that they are pursuing. I have not seen any studies of workers in this area. I don't know whether they have serious problems, but the studies should be pursued.

I wasn't too clear from Dr. Palmer's remarks about the actual comparison of smokers in the cosmetologists and in the controls he studied. It also wasn't too clear to me what was studied when he monitored the working environment, whether he was just studying particulates, or whether he was studying polyvinyl pyrrolidone, or whatever else might be in that area. There are, of course, a lot of chemicals and substances used in the cosmetology industry.

Dr. Sherman certainly made some stimulating remarks, but one thing bothered me just a little bit. She indicated that sometimes employees might be suspicious of physicians representing employers. There are a number of honest doctors in industry, too, and I wanted to point that out, that we are conscientious and careful. In our company, I am as conscientious in dealing with employees as I was when I was in private practice before that. It is a policy in our company that when we examine an employee in regard to work, we tell that employee whatever we find. And if we make some recommendations that might limit their assignment, we explain the reasons to them and talk to them as much as they want about it.

Let me make some comments about my own experience. For the

past few years the Bell System has been placing a greater than usual number of women in jobs that have traditionally been filled by men. Women request these semi-skilled and skilled craft jobs because the pay is better than in the jobs traditionally favored by females, such as clerks, secretaries, telephone operators, tellers and similar jobs. Women have always had the opportunity to compete for these jobs, but we now have an Affirmative Action Program to actively assist women.

The non-traditional (for women) jobs here referred to include Central Office Switchroom Craft Workers, Telephone Installers, Telephone Repairmen, Cable Splicers, and Linemen. The Switchroom workers are skilled, but the job gradually has become light in terms of physical demands. The other four positions are outside plant craft jobs that can be described as "strenuous or potentially hazardous." The term "strenuous" is used in a common sense way and not rigidly defined. "Potentially hazardous" refers to possibilities of physical trauma more than exposure to dangerous conditions or substances in the workplace. These jobs all require a high degree of alertness and balance, plus accurate use of special senses--vision and hearing, for example. They also require driving a truck, working on rung ladders, in manholes, and outdoors in various types of weather.

We have had a very modest degree of success in placing women in these jobs. We are disappointed in that many women fail to pass the one week or more training course that has always been required of all outside craft workers. This is primarily for safety purposes. It is simply too risky to assign anyone--male or female--to climb telephone poles, work on ladders and in manholes, use brace and bit as well as electric drills and wooden extension ladders, etc., without training. Even after a high fail rate, a sizeable number of women quit after a week or a month on the job, stating that they don't like the work for a variety of reasons, including fear of working on poles and ladders. In addition, we find our women craft workers have a higher frequency of on-job accidental injuries than do male employees. The difference in frequency rates is great enough so that we will increase our efforts to prevent accidents and will conduct a detailed study to see if we can learn why there is a high rate of accidents in females.

As a physician specializing in occupational medicine, I think I can do a pre-placement examination of high quality. All of our employees receive a pre-placement examination prior to assignments to jobs that are strenuous or potentially hazardous. It appears this has not been adequate to identify many of those who will have accidents. And it has not identified many of those who are unable to do a satisfactory job performance due to inadequate strength, or stature, or coordination, or balance. Of course, it is unrealistic to think that satisfactory completion of a pre-placement examination will prevent all accidents and insure a suitable degree of job performance. But we have always believed our efforts

are helpful in both these areas. Therefore, a high accident rate and a high failure rate in training as well as on the job bothers us. Are we doing pre-placement examinations as well as we should? Are our recommendations about job placement proper, or could we improve? Should we be doing additional tests or examinations in order to screen out those most likely to have accidents?

We don't know the answers to these questions. We plan to study this subject in order to learn more about it. We don't think anyone else knows the answers at this time either.

DR. SHERMAN: I am an internist in private practice in the Detroit area, and I am not employed by either a company, a union, or the government. So I think I have a rather independent point of view.

In the course of my practice I have seen 2,000 people from various and sundry industries. One of the things that I see is that where women are in traditional "male" occupations, they have the same diseases that the men have. For example, women who have worked for 30 years in a foundry as core makers with a high exposure to sand and chemicals have the exact same diseases as men.

In new industries where people are working in plastics, most of the jobs fall to the women. I have seen a number of cases of irreversible lung disease in women with as few years exposure as three, who are working with some of the heat-setting resins.

I came to the meeting last night to hear Andrea Hricko and the very articulate women who spoke about their job situations, and one thing appalled me. I looked up on the stage and two or three of the women were smoking. I have to say to you that if you want to be taken seriously about cleaning up the work situation, you better stop smoking because they are incompatible. In every single case I have ever testified on, the smoking history was brought out, and if a person did smoke it was used against them as far as making any kind of an occupational lung claim.

I would like to share with you a few ideas on how to choose a doctor for work-associated diseases or environmental-associated diseases. Try to pick a doctor who will listen to you, and one who will examine you from head to foot and not just look in your ears and listen to your heart through your blouse or your shirt.

You have a right and an obligation to know your medical history, to know all your medical records, and to know your medicines. It is your body and you are paying the bill, so don't walk out with a prescription for Brand "X" pink pills without knowing what they are.

Now there is a different situation when it comes to physicians who are paid by the company. Most of the time you are not

told your physical findings, your medical findings, or the results of any lead tests if you are working in a battery plant. You are not told the results of your medical tests. You may be given pills for back strain or for headache, and you may have no knowledge of what they are. I would suggest to you to make sure that you do.

I would also like to point out something I didn't find out until quite recently. Physicians who are paid by industry are not under the usual malpractice rules. So if you see a physician in the plant who fails to tell you that you have a cancer so you can do something about it, or who treats you incorrectly, you cannot sue that physician under the malpractice rules of your state. You can only recover what is available through Workmen's Compensation. This is something that clearly needs to be attended to as far as your contract talks.

Another suggestion I would make is to know all the chemicals that you come in contact with. Dr. Gordon from the Dow Chemical Company yesterday said that we not only have to consider occupational exposure, but also household exposure. He is absolutely correct.

Your Saran Wrap, your pesticides, your hairsprays and deodorants, etc., all contain chemicals. I challenge you to go home and make a list of all the chemicals you are using, all the over-the-counter drugs you are using, all the food products you are using, and try and find out what chemicals they contain. You are going to have quite a chore. I would also suggest that you not ignore the inert ingredients. If you recall, many paints were packaged with a vinyl chloride propellant. In many household products, the inert ingredient is freon, which is a cardiotoxic drug.

Now to move on to the workplace. You are going to have even greater difficulty in finding out what chemicals you work with. I think this is probably top on the list of bargaining demands that must be made by the worker and that must be agreed to by the corporations. You should be given definitive information about and the generic names of the chemicals you are working with.

Most women are not represented by unions. So people who are represented by unions must make sure that the laws cover everybody in the workplace. We must have national laws concerning disclosure of information and disclosure of chemicals that are used in all products. We are indeed our sister's keepers and don't forget it. Once we get the women liberated, we can then work on the men.

We have heard over and over again for at least a year and a half that 80% of all cancers are occupation and environmentally caused. Even if we are wrong by a 50% margin, this is a God-awful amount of disease that is preventable. I don't know any of the data specifically on birth defects, but if the teratogens are like the carcinogens and the mutagens, then many birth defects

have also got to be preventable.

It costs a minimum of \$6,000 a month to be in a hospital. You can do an awful lot of cleaning up for that kind of money. And if you are talking about the birth defects, you have a life-long cost of care for that person, as well as loss of that person to productive society.

Most physicians have taken the Hippocratic Oath. One of the things the Hippocratic Oath says is, "If you can do no good, at least do no harm." I think we have to err on the side of conservatism. Where there is a question of genetic risk and carcinogenesis, I believe we have to get those chemicals out of the workplace, out of consumer products and away from people.

QUESTIONS, ANSWERS, COMMENTS

MR. HARRY SLATON: I am a consultant in industrial hygiene. Yesterday the question came up whether the government had any official regulations or pronouncements on women who were pregnant. I don't know of anything in the Federal Register, but this is a little booklet that is approved by Office of Child Development Number 73-17, called "Prenatal Care." It happens to have been written by my wife, Marian Slaton. I would like to quote from it for the record.

"What things should I know if I work outside my home? It is best for you not to work or play to the point of getting tired. Your physician might contact your employer to help you find a less tiring job. There are some things you might be able to do on the job which will keep you from getting too tired.

"If you have a job where you stand most of the day, during the rest period sit down and put your feet up. If you have a job where you sit in one position, if possible get up and walk around.

"You may be working at a job where certain chemicals are dangerous to you when you are pregnant. When you know you are pregnant, tell your nurse or supervisor so that you can be transferred to a safe area if this is necessary.

"You should not be working at a job that requires heavy lifting or moving. If things are going well during your pregnancy and you feel up to it, the chances are that you can work during your entire pregnancy.

"Your doctor will tell you when and if you should stop working."

I would also like to make a comment on Mr. Schneider's presentation. When I was the State Industrial Hygienist in

Nebraska, I had reason to visit dentists' offices with a mercury testing device, and wherever I went the mercury was ubiquitous. It was on top of shelves. It was underneath the cupboards. It was in corners. It was all over the place.

DR. JEANNE STELLMAN: I am now with the University of Pennsylvania. I was formerly Assistant to the President of the Oil, Chemical and Atomic Workers. I would like to make a comment on the perspective of yesterday and today. Yesterday our entire perspective was devoted to considering women as the vessels for future generations and today we have itemized women into particular professions.

First, my comment is that the vast majority of women in the workplace have already had their children. Women generally have completed childbearing by the age of 30 and still have another 35 years to go.

Second, as a mother of children, I wonder why we are so busy preserving them until the age of 18, only to go and dump them into the workplace with all the hazardous substances.

Occupational health is occupational health is occupational health. I don't think that babies are any more valuable than adults. The occupational health problems of women are no different than occupational health problems of men except when they are pregnant. I would like to say that the problems of the pregnant woman are not very much different from the problems of the 50-year-old man who has had cardiovascular disease, and who also needs special temporary disability or permanent disability which will still allow him to work.

When we allow women to be picked out, the next group that will be picked out will be the 50-year-old men with heart disease, and the next group that will be picked out will be cigarette smokers, which has been suggested by people in the asbestos industry. And soon we will fragment the work force in such a way that we'll have a group of super workers, who can withstand any conditions that we place them in.

The papers that were presented today were interesting, but the fact is, the basic problems that women have are (a) they work 80 hours a week when men work 50. That is why they are tired, not necessarily so much because of their jobs; (b) they work in jobs in which there is stress, and in dead-end jobs. For example, they are put into the health occupations, not as physicians, but as people who have to lift patients and carry bottles of urine.

Women are segregated. They are discriminated against. And they face dead-end jobs in dead-end careers. And if we are

interested in children and the care of children in our society, then let's start providing childcare facilities for women who have to work, and let's start providing the means for women to fulfill their social obligations as mothers and childbearers. That is what we should be talking about, not simply whether a few isolated substances will pass through the placenta and end up in a few occupational diseases. Not that I am denigrating the work that was done; I am just denigrating the perspective of placing women in the category of always being pregnant until proven otherwise.

MR. ERIC FRUMIN: I am with the Department of Occupational Safety and Health of the Amalgamated Clothing and Textile Workers' Union. I want to respond to what Dr. Kilburn has said about women in the textile industry with two short comments and one question.

First, for everybody's information, you can control noise in the textile industry. It is not nearly impossible as he said, and earplugs are not effective.

Secondly, Dr. Kilburn did not discuss or mention the widespread use of chemicals in the textile industry, which are presenting serious and as yet unexplored hazards in this country. These chemicals are quite similar to those used in the graphic arts industry, which have been demonstrated to have severe toxic effects on workers, male and female.

As far as cotton dust is concerned, I would like to ask Dr. Kilburn what he thinks has been the effect of the time that it has taken for the American medical establishment and the American government to give official recognition to the existence of cotton dust as a hazard in this country. I would also like to ask him what has been the actual experience of cotton mill workers who try to get compensation, which he says is provided for them legally.

DR. KILBURN: Well, as we accumulate more occupational exposure problems, and as one sees the period of time it takes to put in motion the governmental operation to regulate, one gets actively discouraged. I think we are falling behind. We haven't even gotten to the point that we can put TLV's on the 409 things that TLV's are needed on. And we haven't translated that into occupational inspection. So that I think we are well behind and that we are losing ground.

In the interests of trying to make my presentation brief, I certainly did leave out consideration of chemicals. They probably are important in fractional parts of the textile industry. I was mainly talking about the making of cloth and not about permapress, which is a tremendous problem. All of you people who like permapress clothing should remember that the workers who are putting the formaldehyde in the permapress are about five times as likely to get chronic bronchitis as those who are making the cotton fiber or weaving it. So it is just like the examples we have seen in

other situations. Every time you start an investigation, you unearth a whole new group of hazard factors and it is important to try and put these in some kind of perspective.

MS. KATHY HUNNINEN: It has been mentioned several times that employees encounter difficulty in getting safety information. I would like to remind you that lots of times OSHA inspectors can't get that information either from the companies.

I have a comment for Dr. Chaffin. Studies of women employees in European countries, where there are better physical education training programs for young girls in school, show that they have higher strength capabilities than the average for women here in the United States. American women haven't had the chance to develop their bodies like men have, and that should be considered.

And, Dr. Hunt, there are some studies that show teratogenic effects of non-ionizing radiation with microwaves. I don't know if it is a potential problem or not, but it is probably something that should be looked at.

DR. CHAFFIN: I couldn't agree more with your comment on the training effects here on strength. I would stress that it is a primary factor and that we should have policies which would not only test a person for strength capability when going on a job requiring strength, but that would also help to facilitate the strength capability of a person if the person still wants to pursue that job. I agree wholeheartedly that there is a lot of potential for such training to protect a person.

DR. HUNT: Yes, I would certainly agree that we need more information about radiation other than ionizing radiation. I chose to concentrate in the ionizing area because that has been where my experience has been. Anybody who tries to move into the area of non-ionizing radiation is going to find even more difficulty, I think, in getting the appropriate information.

MS. JAN SHUMBERG: I am from Yale University. I have a couple of comments on Mr. Schneider's presentation. Unless I misunderstood or misheard some of the numbers you presented, you said that the concentrations of mercury which were commonly found in your control populations were on the order of 2 to 4 micrograms per liter with upper limits around 10 micrograms per liter, and yet you used 30 micrograms per liter as the cutoff point between normal and abnormal in your occupationally exposed group. It seems to me that more than 30 micrograms per liter is very high. I would also hope that some studies and further analyses are going to be made on the people exposed to over 10 micrograms but less than 30, because it seems to me that those are also very high levels compared to true normal levels in unexposed people.

Also, one other comment on some of the methodology that was

presented. It seems to me that it is a fairly common practice in all kinds of epidemiological studies to send some information to subjects on whether or not their test results were normal or abnormal, but basically to leave it up to the subjects' private physicians to explain to them the extent of what the abnormal findings mean. I think it is about time that all of us doing these kinds of studies reconsider whether this practice really is acceptable. Maybe we should take upon ourselves the responsibility to explain directly to people what these findings mean because I don't think it is really being done.

MR. SCHNEIDER: I appreciate your last comment, but unfortunately we were under some legal constraints, and that was the reason we did what we did.

Insofar as choosing the 30 micrograms as our limit for what we would consider to be increased absorption, and under 30 micrograms as not indicating increased absorption, that was purely arbitrary. It tends to be 10% of 300 micrograms per liter of urine at which you may see symptoms of poisoning. It just was coincidental that we took 10% of that particular value. It has no real meaning except for ease of handling the data.

DR. TURSHEN: I am with the Institute of Policy Studies. This is a comment I would like members of the panel to respond to. There is a tendency, both in the medical field generally, and reflected in this conference yesterday and today, to blame the victim and to place the responsibility for accidents or illness on the worker instead of faulting the industry for providing working conditions that cause injury or ill health.

It is true that smoking and excessive alcohol consumption are detrimental to health, but that issue is separate from the problem of creating a safe and healthy working environment. If the victim-blaming philosophy were taken to its absurd conclusion, it would be that workers should not have sexual relations because they might produce deformed babies.

DR. HUNT: I would like to comment on that because I think that society's acceptance of tobacco use, which may well be one of the most serious sins that we can place at the door of the tobacco industry, is the camouflage that allowed us for a long period of time to ignore the problems in the workplace because for so long the blame was placed on cigarette smoking.

I might add that the work concerning smoking and Polonium-210 in tobacco smoke is work that we were doing back in 1962. We were saying then that if the time ever came when we could separate the tobacco industry and the problems in the workplace, life would be much easier. But every time you pick up a copy of "Ms" or a copy of "Women's Sports" and see the extent to which the tobacco industry is advertising to women, you can see how much more they are

camouflaging.

DR. KILBURN: I agree that we need to keep a perspective and I would challenge anyone in the room to show that with very few exceptions we have a total health effect from any occupational exposure to equal that brought about by the personal choice to smoke cigarettes over one's lifetime. I think we really have got to concentrate on priorities, and that is the number one priority for pulmonary disease in the world.

DR. KENNETH BRIDBORD: I am from NIOSH, and I would like to take note of another potential problem for flight attendants. It comes back to the question of smoking and side-stream cigarette smoke. NIOSH was involved in a study a number of years ago of benzo(a)-pyrene levels inside aircraft. This was before the smoking-non-smoking sections came into being, but the levels still looked more like the top of a coke oven than they did the ambient atmosphere, and I am not being facetious either.

These levels were quite high and would lead one to suspect that a long-term study of the possible increased risk of lung cancer among flight attendants would be an appropriate future study. In that regard, one would also be concerned about carbon monoxide. I seriously doubt that the smoking-nonsmoking sections provide any degree of protection for flight attendants.

MS. KAY LUCAS: I am a lawyer with the Department of Labor, and my comments are directed to Dr. Kilburn's paper.

I shared the same sentiment as the gentleman earlier and wondered why Dr. Kilburn had not addressed the use of chemicals in the textile industry.

And, secondly, the statistics on the initial job placement of men and women in the textile industry suggests discriminatory policies and practices. I wonder if Dr. Kilburn has any information himself, or knows of any studies that have been done on either the effect on fertility of those employees or on birth defects of employees in that industry.

DR. KILBURN: I wish there were information. So far as we know--and this is mostly based on the county-wide distribution of disease in North Carolina--there are no effects.

There is one strange effect that I think needs to be followed up. We have concentrated this entire conference on adverse effects but it looks as though there may actually be some protection against lung cancer for workers in the farming and textile industries. There isn't time to go into why, but there is statistical evidence now that there are lower lung cancer rates among textile workers and farm workers than in the general population, even allowing for smoking.

The introduction of chemicals into textiles is increasing. My own personal experience has been amazement at what people are likely to add to most anything to make it less static, less flammable, less this or less that. Some of these things that we test by putting them in the marketplace, which means testing on human individuals, ought to have testing the way we do for food and drugs.

Society needs to do something about this immediately because we essentially are testing these on the user population or the consumer. If that is interference with fair trade, I think we should interfere with it.

MR. JOSEPH GRADEN: I am from the North Carolina Occupational Safety and Health Project. My question is for Dr. Kilburn. You have devoted most of your attention to cotton dust. I wonder if there is any work that has been done with regard to the synthetic fibers, which have been introduced relatively recently, and whether or not there is a potential there for carcinogenicity since the latency period will probably be somewhat down the road.

DR. KILBURN: That is an excellent question and one which is right now worrying, among others, duPont and myself. There is a report that came from Spain in November of granulomatous disease of the sort that was spoken about earlier due to the inhalation of nylon and orlon gratings or dust. This is certainly logical because, as everyone knows, if you want a piece of rope to last ten times as long as rope made of sisal, make it out of nylon. Similarly, if you want to implant something in the human body to last, such as an aortic repair prosthesis, make it of nylon or orlon. So the possibility that this stuff could be ground up and inhaled and could produce disease is logical, and there is, as I say, some evidence of this from Spain. I agree that there is no way, short of lifetime studies, to determine whether these are potential carcinogens.

We now have about 35 years of experience in nylon production, perhaps longer. It would be useful to look at an occupational group who are highly exposed, and see whether they have differing rates. That would be the approach I think would be logical.

MR. JAMES McVAY: I am an industrial hygienist with the American Can Company. I would like to ask Dr. Kilburn about these people who don't come to work on Monday morning and have this initial fever and illness. Is there a causative agent? Is it fungi or bacteria that is causing this?

DR. KILBURN: We think this is the inhalation of endotoxin derived from gram-negative bacteria, which is something that is adapted to in time. What happens to the initial person is this: he either says, "I am so sick that I am not going back to work," and drops out of the industry, or he comes back. He is only

moderately or slightly ill the second day. From then on, as long as he stays exposed, he has nothing in the way of symptoms. This sounds like inhalational endotoxin disease, and although it is not proved, it is our current working hypothesis.

MR. JEFFREY BERGER: I am with the Solicitor's Office in OSHA, Department of Labor. The woman who spoke with regard to the flight attendants pointed out that she was unable to get information as to occupational diseases and injuries. I would like to point out to her that OSHA has recordkeeping and reporting requirements for all workers unless they are covered by another agency. So OSHA would cover your flight attendants, if they are not covered by the FAA.

As a general comment concerning the orientation of this conference, I think it is very useful and informative for everyone to know about what harmful effects are occurring to workers due to chemicals and carcinogens. However, I think that in order to remedy the situation, it is necessary to in tandem do studies of feasible administrative and engineering controls because in order to remedy the situation you just can't shut down all industry. It is also necessary to investigate ways in which it is economically and technologically feasible to correct these harmful effects to workers.

DR. BERTRAM CARNOW: I agree that the problems are overwhelming and that maybe the next conference we have should be called "The Physical and Economic Rip-Off of the Woman Worker," but since we are dealing with some very specific areas, I think we have to address those. I would like to suggest to the person who spoke about women on airlines that one area that is not being dealt with at all is the radiation question. Women on airlines are exposed to increased cosmic rays and they are also exposed to inappropriately packaged radioactive materials. I think it would not be inappropriate for them to wear some kind of monitoring badge.

A friend of mine, who carries around a Geiger counter all the time, says that when he sits on airplanes he frequently moves his seat because some areas of the airplane are more radioactive than others.

In addition, in regard to circadian rhythm, I find in some informal discussions with women airline attendants that they have a lot of gastrointestinal problems. A lot of them have colitis and things like that. There are some data on circadian rhythm problems. Studies have shown that ulcers are much more frequent in people who work on shift rotations than in other people, and that they have considerably more constipation than other people, and other such problems. So I think that when you look at the problems of flight attendants, you should look at all of these questions.

One other comment. There was a statement made that you cannot fault a physician in an industry because he is covered by Workmen's Compensation. This is not universally true. In some states, you can. When irresponsible physicians are acting that way, I think that they should be taken on in those states where they can be taken on.

A final thing. In the study on aerosols, one very interesting point was that small plants seem to be the worst plants of all. That is of concern to all of us because those are the plants that OSHA never gets around to. Those are the plants that are the most dangerous, and most frequently the kinds of plants that women work in because they may be near the house where they live, or they may provide part-time jobs. I think that this is an area that has to be looked at with much greater care.

MS. WOFFORD: First of all, a comment on the radiation levels. There was a study done by the AEC. One hundred flight attendants participated. The calculations from that study were received about November of last year. There was nothing very conclusive about it. We wanted to have wider coverage with a better dosimeter and to do a more intense study on radiation levels for the flight attendant.

Our problem right now is that the cost of badges would be more expensive than what we can handle at the present time. That is why we haven't gone further into the study on radiation.

We do know that the problems do exist, but we cannot state that there are really bad hazards. I don't know if you have heard about the Congressional hearing on radiation. The American College of Radiology and the Society of Nuclear Medicine measured the amount of radiation that pilots were getting in the cockpit. They were getting more radiation from the dials than they were from packages of radioisotopes that were being shipped on the airlines.

As far as constipation and ulcers, we do have very large problems in that area. Pectoral ulcers is one of the main problems we have. And those are all being studied.

MS. JULIE KISIELEWSKI: I am from the Office of the Secretary of the U.S. Federal Women's Program. I haven't heard any mention of the largest occupational group of women. Although we have the cleanest houses and clothes in the world, our housewives probably have lungs full of oven cleaner and bathroom cleanser and detergent. I think this is something that we ought to be looking at. Also, even though people are concerned about women working in the office and carrying around boxes, nobody ever asked me about lugging a 50-pound vacuum cleaner up the stairs, or carrying a 30-pound kid around when I was pregnant. I think our standards are very different for household workers than for office workers.

SESSION V

LEAD AND WOMEN, A UNIQUE PROBLEM?

MODERATOR: Dr. Warren Muir
Senior Staff Member for Environmental Quality
Council on Environmental Quality

REVIEW OF LEAD TOXICITY

Dr. Kenneth Bridbord
National Institute for Occupational Safety and Health

ABSTRACT

This paper presents a general review of lead toxicity considering exposure to lead in the workplace as well as in the general environment. Persons who are exposed to lead on the job are in double jeopardy since the lead exposure while working must be added to the lead entering their bodies from general environmental exposure, which in certain instances may already be excessive. In this regard women workers of childbearing age must be considered to be at triple risk. Among the effects of lead which have been of growing concern to health scientists is its ability to harm a number of organ systems in the body and in particular the central nervous system, the kidneys, and the blood-forming elements. Any recommended standard for lead either in the general environment or in the workplace should consider the special susceptibility of women of childbearing age and particularly the fetus to lead.

I welcome the opportunity to participate in this conference and to present a general review of lead toxicity at this particular session. My experience within government has been such that I have had an opportunity to view the lead problem both from the perspective of protecting the worker, and from the perspective of protecting the general population, especially young children, from the adverse effects of lead. In this regard I believe that a number of concerns expressed about lead exposure in the working population are now impacting our consideration of the situation involving the general population.

The toxicity of lead has been known to man for approximately two thousand years. Pliny the Elder, a Roman senator, warned against the toxicity of lead in the first century, A.D. I am sure that many of you are aware of the speculative opinion that excess exposure to lead may have been a contributing factor in the fall of the Roman Empire. I am obviously not in a position to verify the possibility.

Lead may enter the body from a number of routes. For the general urban population with no unusual source of lead exposure, lead absorbed into the body comes primarily from the diet and from the ambient air. In urban areas approximately one-third of the lead absorbed into the body of an adult comes from inhalation of air contaminated with lead derived primarily from motor vehicles. The relative contribution of automotive-derived lead to lead absorption in children is somewhat less certain but for a number of reasons it is likely that this source contributes even more to lead absorption in children than adults. This is because children, particularly young children, ingest lead-contaminated dirt and dust contaminated in large part by lead fallout from the atmosphere originating from motor vehicle emissions. However, the single most important source of lead for young children is derived from lead paint. Any strategy to control lead in the general environment must consider all sources of exposure, including lead in the diet and in the ambient air.

Persons who are exposed to lead on the job are in double jeopardy, since the lead exposure while working must be added to the lead entering their bodies from general environmental exposure, which in certain instances may already be excessive. The most important route of lead intake for exposed workers is by inhaling air that frequently is contaminated with large quantities of lead often 100 to 1,000 times greater than that commonly found in the ambient atmosphere. In addition, workers may also ingest significant quantities of lead-contaminated dusts on fingers, lips, cigarettes, etc. Lead dust on the clothing of workers has also been reported to cause elevated blood lead levels in children, when contaminated clothing is brought into the home. One special form of lead, organo lead compounds, may also be absorbed into the body directly through skin contact. Organo lead compounds are most commonly used as lead additives in gasoline.

Among the effects of lead which have been of growing concern to health scientists is its ability to harm a number of organ systems in the body -- in particular the central nervous system, the kidneys, and the blood-forming elements. Perhaps most publicized in recent times have been the effects of lead upon the nervous system. The relatively poor capacity of the nervous system to repair itself means that once damage has occurred, as in the case of lead, there is a possibility that full recovery will not take place.

Lead is capable of damaging both the central and the peripheral nervous systems. If exposure to lead is sufficiently great, the central nervous system may be acutely and severely damaged, resulting at times in coma, convulsions, and even death. This condition, often referred to as acute encephalopathy, is most commonly observed in young children. Studies in children have shown that once acute encephalopathy has occurred, there is a high probability of permanent, irreversible damage to the nervous

system. In lead poisoned workers a manifestation of injury to the nervous system, this time to the peripheral nervous system, involves damage to the extensor muscles in the forearm, a condition more commonly known as wrist drop. A common manifestation of lead poisoning in workers is severe abdominal pain, a condition known as lead colic.

In recent years, a great deal of concern has been expressed about the so-called subclinical effects of lead involving the nervous system. A number of studies strongly suggest that permanent damage to the nervous system may have occurred in children who have been only moderately exposed to lead, and in whom no overt symptoms of lead toxicity had appeared. These effects include behavioral problems such as hyperactivity, difficulty in task performance, deficiency in IQ, and nerve conduction deficits. These same concerns are now also beginning to be expressed in the case of workers excessively exposed to lead, who have deficits in nerve conduction, and who may also have decrements in task performance, or increased hostility and aggressiveness, as a result of exposure to lead. These areas of concern are admittedly on the fringes of scientific knowledge, and one can expect considerable new information in this regard in the next several years.

A well-documented effect of lead involves its ability to adversely affect the blood-forming elements. Clinically, this may be expressed as anemia if exposure has been sufficient. It is not uncommon, for example, to have decreases in hemoglobin concentrations among workers exposed to lead. These deficits may not be strikingly evident on an individual basis where blood counts may still, in general, be within or close to accepted limits of normal. However, on a group basis these changes become significant. In general, clinical anemia does not occur until blood lead levels exceed 80 ug/100g although recent studies suggest that mild anemia may occur at blood lead levels in the range of 60 to 80 ug/100g. The effects of lead upon the blood-forming elements may also be detected by biochemical tests. Biochemical abnormalities associated with effects of lead upon hemoglobin synthesis begin to occur at blood lead levels in the range of 30 to 40 ug/100g. The significance of these early biochemical changes has been a point of contention in the setting of standards of lead exposure for the general as well as the occupational population. In my personal view, these biochemical changes are important and should be considered in the standard-setting process.

An important concern involves the ability of lead to adversely affect the kidneys. A number of studies have demonstrated the toxic effects of lead upon this organ. Among exposed workers, lead is known to increase the risk of death from end stage renal disease. Recent studies suggest that adverse effects of lead as measured by biochemical tests may be affecting the kidneys of lead workers. Much more work needs to be directed at this problem, including the development of tests to indicate early kidney

damage before irreversible changes have occurred. In this regard, kidney damage has also been observed among children excessively exposed to lead paint. Finally, studies in experimental animals have shown lead causes kidney cancer, although confirmatory evidence of this in man is, to date, lacking.

The theme of this conference is women and the workplace. So I would certainly be remiss if I failed to voice my very real concern about the effects of lead upon the reproductive process. If workers exposed to lead on the job and in the general environment are at double jeopardy, then women workers of child-bearing age must be considered to be at triple risk.

Among the problems associated with exposure of pregnant women to lead are abortion and injury to the fetus, including damage to the nervous system. This problem has a long history. For example, at the turn of the century women workers in the lead industries were known to have decreased fertility and an increased abortion rate. Ingestion of lead-contaminated whiskey during the first trimester of pregnancy has been observed to cause fetal injury, and increased exposure to lead during pregnancy has been associated with neurologic damage in children born to these mothers. For example, a recent study from England associated an increased rate of mental retardation in children whose mothers during the time of their pregnancy lived in homes containing elevated concentrations of lead in the drinking water.

Numerous studies have shown that the concentration of lead in the mother's blood correlates with the concentration of lead in the blood of her child at birth. This means that lead, a known toxic material, is present in the tissues of the child before birth. Experiments in animals also demonstrate that lead crosses the placenta to reach the unborn infant. Consequently, exposure of the unborn child to lead will reflect the degree of exposure experienced by its mother by all exposure routes.

These facts are important for both women of childbearing age exposed to lead while on the job and to the larger number of women exposed to lesser amounts of lead through the diet and the ambient air. These facts have been of increasing concern to those Federal agencies charged with responsibilities for protecting workers. In a recent proposal to reduce the Federal standard for occupational lead exposure the Occupational Safety and Health Administration cited the problem of lead exposure among pregnant women as one of the reasons for the need to reduce exposure to airborne lead in American industries by a factor of two. The National Institute for Occupational Safety and Health is also concerned about lead exposure among women workers, and is planning additional studies of this problem.

Special risks to the fetus from exposure of women workers to lead, as for example in industries such as smelting and battery

manufacture, have also been of concern to the lead industries. A number of industries have, in fact, refused to place women in jobs where excessive exposure to lead may occur.

In all likelihood these same concerns about lead exposure among women in the lead industry will also impact recommended standards for lead exposure through the diet and the ambient air. As noted above, adults with no occupational lead exposure receive approximately one-third of the lead absorbed into the body from the ambient air and about two-thirds from the diet. In the case of women who reside near heavily traveled roads, or who spend a good deal of time driving in heavy traffic, the contribution of airborne lead to the total amount of lead absorbed into the body may be even greater. This means that women of childbearing age may already be entering the lead industry with blood levels of lead close to or above a level of concern with respect to an unborn fetus. Any recommended standard for lead either in the general environment or in the workplace should consider the special susceptibility of women of childbearing age and particularly the fetus to lead.

References

Baker, E. L. et al. 1977. Lead Poisoning in Children of Lead Workers, New Eng J. Med. 296:260-261.

Beattie, A. D. et. al. 1975. Role of chronic low-level lead exposure in the etiology of mental retardation. Lancet pp. 589-592.

Bogen, D. C. et. al. 1976. General population exposure of stable lead and 210-lead in residents of New York City. Health Physics 30:359-362.

de la Burde, B. and Choate, M. S. 1975. Early asymptomatic lead exposure and development at school age. J. Pediat. 87:638-642.

Gershanik, J. J. et. al. 1974. Blood lead values in pregnant women and their offspring. Am. J. Obstet. Gynecol. 119:508-511.

Lead Disease Among Workers in Secondary Lead Smelters Report to the National Institute of Environmental Health Sciences, May 15, 1976.

Tola, S. et. al. 1973. Parameters indicative of absorption and biological effect in new lead exposure: a prospective study. Brit. J. Ind. Med. 30:134-141.

Wedeen, R. P. et. al. 1975. Occupational Lead Nephropathy. 59:630-641.

THE EFFECTS OF LEAD ON REPRODUCTION

Peter F. Infante
and

Joseph K. Wagoner

Industry-wide Studies Branch

Division of Surveillance, Hazard Evaluations and Field Studies
National Institute for Occupational Safety and Health

INTRODUCTION

Increasing numbers of women are now seeking employment outside the home. Because of this changing employment pattern, Government, Industry and Labor, for the first time, have been forced to focus attention on the health hazards of occupational exposures to women and to the reproductive processes involving exposure to either sex. Because lead is a known abortifacient, women are being transferred out of high lead exposure areas in lieu of industrial engineering control. The alternative to transfer for some companies has been the requirement that women present medical evidence that they cannot bear children in order to maintain employment in areas of sustained high lead exposure. Such approaches ignore possible toxic effects on reproduction through male occupational exposures. Therefore, available evidence is assessed to evaluate the role of lead exposure on the total spectrum of risks associated with reproduction.

For more than a century, lead has been known to affect the reproductive process. Observations among human populations indicate that lead is associated with sterility, spontaneous abortions or miscarriages, stillbirths, birth defects, increased infant mortality, increased prematurity, increased chromosomal aberrations, and abnormal spermatogenesis. Additionally, animal test systems have indicated that lead is associated with (1) impotency (Hilderbrandt, et al., 1973), (2) sterility (Dalldorf and Williams, 1945), (3) teratogenesis (McClain and Becker, 1970), (4) mutagenesis (Varma, et al., 1974), (5) decreased learning ability in the offspring following lead exposure of either parent (Brady, Herrera and Zenick, 1975), and (6) carcinogenesis (Boyland, et al., 1962).

TERATOGENESIS

Laboratory Observations:

Numerous experimental studies have assessed the teratogenic effects of lead in a variety of animal species (Table 1). In 1928, Hammett and Wallace observed growth retardation in the head region of chicks by injecting lead nitrate into the yolk sac of the embryo. Other investigators injected lead into chick embryos and observed harmful effects on the development of head primordium (Catizone and Gray, 1941), the development of anterior meningoceles

and hydrocephalus, (Butt, Pearson and Simonsen, 1952; Karnofsky and Ridgway, 1952), other cerebral anomalies and limb reduction deformities (Gilani, 1973). The addition of 1% lead acetate to the diet of female chickens has induced limb deformities in the hatched chicks (Stowe, et al., 1972).

Formal studies for the passage of lead through the placenta began in the 1930's, with the development of better chemical analytical techniques. In 1938, Morris, et al. studied the trans-placental passage of lead acetate in rats. They observed significant increases in kidney weight and liver size and a decrease in femur weight. Lead also has been shown to cross the placenta rapidly and in significant amounts, even at relatively low maternal blood levels (Carpenter, 1974). In the mouse and rat, lead nitrate has induced cleft palate and hydronephrosis, respectively (McClain and Becker, 1970). In these same species, pup mortality and runting also has been significantly associated with maternal intake of unspecified lead salts (Schroeder and Mitchener, 1971).

In the hamster, lead nitrate, lead chloride and lead acetate have induced tail anomalies (Ferm and Carpenter, 1967). Anomalies induced by lead acetate also have been potentiated into more severe caudal malformations by the addition of cadmium (Ferm, 1969).

A study of the effects of lead metal ingested for two generations by both male and female rats also has been reported. Growth and reproduction in the F₁ and F₂ generations was assessed (Dolldorf and Williams, 1945). They observed normal growth in the first generation. In the second generation, however, (1) a 50% increase in mortality was observed, (2) the surviving young were stunted in growth, and (3) some animals of both sexes became sterile.

Human Observations:

Just as animal data have demonstrated the ability of lead to cross the placenta, so too have observations among humans. As early as 1916, Oliver reported that lead had been found in the liver and other organs of a stillborn child and in the placenta of a mother who was a "white lead worker." More recently, studies have shown a high correlation between maternal and neonatal blood lead levels in humans, $r=0.64$ (Gershanik, Brooks and Little, 1974).

Although the animal data incriminating lead as a teratogen first surfaced in the 1920's, data demonstrating similar effects among humans surfaced much earlier. In 1916, Oliver reviewed data from earlier publications showing adverse effects of lead on reproduction. Included in this review is a study published in 1860 by Constantin Paul, who reported the results of 123 pregnancies where both the father and mother had occupational exposures to lead at an unspecified industry. Of these pregnancies, 52% ended in miscarriage, 4.1% ended in stillbirth, and 3.3% ended in

prematurity. Of the 54 remaining live births, 37% (20/54) died within the first year of life. In a second series of 43 pregnancies, where only female lead exposures (unspecified) were involved, Paul reported that 74.4% ended in miscarriage and 7% ended in stillbirth. Thus, both human and experimental studies have demonstrated transplacental passage and teratogenic effects of maternal exposure to lead.

MUTAGENESIS

Laboratory Observations:

Evidence of the paternal effect of lead intoxicification on perinatal mortality in experimental animals was first demonstrated by Cole and Bachhuber in 1914 (Table 2). They fed a diet containing lead acetate to two strains of male rabbits and mated them with non-exposed females. The results from lead-exposed males only demonstrated: (1) lower birth weights resulting from these pregnancies and (2) higher mortality within the first four days after birth (a two-fold excess) as compared to controls.

A year later, these findings were corroborated in guinea pigs (Weller, 1915). Commercial white lead was given orally to male pigs, which were then mated with lead-free females. The observed effects were: (1) sterility; (2) reduction in birth weight by 20%; (3) increased number of dead in the first week; and (4) offspring of lead-poisoned males remained permanently underweight.

A recent study of reproductive performance from male rats fed 1% lead acetate in their diets by Stowe and Goyer (1971), has demonstrated a significant reduction in pup birth weight, a 15% reduction in the number of pups per litter, and an 18% reduction in survival rate as compared to controls.

In 1973, Hilderbrand, et al. studied the effects of lead acetate ingestion on reproduction in the rat. A significant increase in prostatic weight (to as much as twice the control values) due to hyperplasia resulted from blood lead levels of 30 ug/100 ml. These blood lead levels are only one-half those (60 ug/100 gr whole blood) currently being proposed for biological monitoring of lead workers to provide an adequate margin of safety against adverse health effects (Dunlop, 1975). At the 30 ug/ml blood level, a 70% reduction in testicular weight, impotency and reduced sperm mobility also were observed. (Female rats exhibited ovarian cysts.)

Muro and Goyer (1969) demonstrated a significant increase in gap-break type of chromosomal aberrations by lymphocyte culture from mice fed a diet containing 1% lead acetate. Subsequently, Varma, Joshi and Adeyemi (1974) fed a diet containing 2% lead sub-acetate to mice and demonstrated a dominant lethal mutation effect, i.e. a significant excess of post-implantation fetal deaths resulting from exposures to the male animal only. These

investigators also observed a 50% reduction in male fertility.

Other assessments of potential mutagenic risks of lead also have been made. Gene frequency differences in the fruit fly (*Drosophila melanogaster*) have been significantly correlated with distance from lead smelter operations (Lower, 1975).

Human Observations:

Oliver, in 1916, also summarized data for pregnancy outcome among the wives of males employed as house painters, many of whom suffered from lead colic. Of 467 deliveries, 23% (107/467) were stillborn as compared to 8% for the entire town.

Oliver also reviewed data from Lewin for 32 pregnancies from "healthy" women, who were married to lead workers. The industry was not specified. Of these, 34.4% ended in miscarriage, 3.1% ended in stillbirth. Of live births, 40% died within the first year of life, and only two children lived to adulthood.

The effects of lead on human lymphocyte chromosomes have been studied. As a result of human lymphocytes cultured in vitro in the presence of lead acetate, Beek and Obe (1974) induced archromatic lesions, chromatid breaks and isochromatic breaks well in excess of the control group (Table 3).

From the study of occupational lead exposure, several investigators (Schwanitz, Lehnert and Gebhart, 1970; De Knudt, Leonard and Ivanov, 1973; Forni, Cambiaghi and Secchi, 1976) have concluded that lead is associated with a definite increase in the number of chromatid and chromosomal changes in circulating lymphocytes of workers. Others have reported negative cytogenetic findings (Schmid, et al., 1972; Bauchiner and Schmid, 1972; O'Riordan and Evans, 1974). Although O'Riordan and Evans reported no significant difference between the study group and "inplant" controls, chromosomal anomalies in the study group were significantly greater than general population control, $P < 0.001$. This observation led these investigators to conclude that individual variability in scoring chromosomal gaps might have accounted for these apparently conflicting results. However, "inplant" controls may have been exposed to other mutagens.

Of even greater significance are the findings in 1975 by Lancranjan, et al. who studied the reproductive ability in 150 male workers exposed to lead in a storage battery facility. In workers with moderately increased lead absorption (53 ug/ml of lead in whole blood), a significant decrease in fertile ability was observed. This observation was related to a direct toxic effect on the gonads, which resulted in significant alterations in spermatogenesis--asthenospermia (decreased motility), hypospermia (decreased numbers), and teratospermia (malformed sperm). Even in workers with slightly increased lead absorption (mean of 41 ug/ml

blood), significant differences in asthenospermia and hypospermia were observed. Thus, both human and experimental studies have demonstrated mutagenic effects and reproductive impairment following paternal exposure to lead.

CARCINOGENESIS

Respiratory cancers have been observed in laboratory animals following inhalation exposure to lead oxide mixed with benzo(a)-pyrene, whereas groups given these agents singularly did not develop cancer (Kobayashi and Okamoto, 1974). Kidney cancers have been reported in animals following subcutaneous injection of lead phosphate (Zollinger, 1953; Matthews and Walpole, 1959; Tonz, 1957) and by oral ingestion of lead acetate (Boyland, et al., 1962; Van Esch and Kroes, 1969). The study of Cooper and Gaffey (1975) presents suggestive evidence for excessive human respiratory cancer among lead smelter and battery plant workers.

Cancer mortality experience in Shoshone County, Idaho, where primary lead and zinc smelters are located, is also of interest. During the period 1968-72, the respiratory cancer rate for Shoshone County was the highest recorded in the State (Bax, 1975). An additional report has indicated that end-stage renal disease shows a four-fold excess and kidney cancer shows a two-fold excess for Shoshone County as compared to the State average (Landrigan, 1975). These observations suggest that lead may be carcinogenic as well as mutagenic.

SUMMARY

Evidence based on animal and human studies clearly demonstrates a teratogenic response following maternal exposure to lead. Of equal concern is evidence demonstrating that lead may be mutagenic, or adversely affect reproduction as a result of paternal lead exposure. In light of these findings, must we now transfer male employees from high lead exposure areas, or require proof of their inability to reproduce as has previously been the public health approach for females?

REFERENCES

- Bauchinger, M. & Schmid, E. 1972. Chromosome analyses in cell cultures of the Chinese hamster after treatment with lead acetate. *Mutat. Res.* 14:95-100.
- Bax, J.A. 1975. Letter of August 8, from Idaho State Health Dept. to Ruch, W. on "Selected Causes of death in Shoshone County and the State of Idaho."
- Beek, B. & Obe, G. 1974. Effect of lead acetate on human leukocyte chromosomes in vitro. *Experientia* 30:1006-1007.
- Boyland, E., Dukes, C.E., Grover, P.L. & Mitchley, B.C.V. 1962. The induction of renal tumors by feeding lead acetate to rats. *Brit. J. Cancer* 16:283-288.
- Brady, K., Herrera, Y. & Zenick, H. 1975. Influence of parental lead exposure on subsequent learning ability of offspring. *Pharmacol. Biochem. Behav.* 3:561-565.
- Butt, E.M., Pearson, H.E., & Simonsen, D.G. 1952. Production of meningoceles and cranioschisis in chick embryos with lead nitrate. *Proc. Soc. Exp. Biol. Med.* 79:247-249.
- Carpenter, S.J. 1974. Placental permeability of lead. *Environ. Health Perspect.* 5:129-131.
- Catizone, O. & Gray, P. 1941. Experiments on chemical interference with the early morphogenesis of the chick. II. The effects of lead on the central nervous system. *J. Exp. Zool.* 87:71-83.
- Cole, L.J. & Bachhuber, L.J. 1914. The effects of lead on the germ cells of the male rabbit and fowl as indicated by their progeny. *Proc. Soc. Exp. Biol. Med.* 12:24-29.
- Cooper, W.C. & Gaffey, W.R. 1975. Mortality of lead workers. *J. Occup. Med.* 17:100-107.
- Dalldorf, G. & Williams, R.R. 1945. Impairment of reproduction in rats by ingestion of lead. *Science* 102:668-670.
- De Knuddt, G., Leonard, A. & Ivanov, B. 1973. Chromosome aberrations observed in male workers occupationally exposed to lead. *Environ. Physiol. Biochem.* 3:132-138.
- Dunlop, J.T. 1975. Occupational exposure to lead. *Fed. Register*, (29 CFR Part 1910), Docket No. H-004, 40:45934.
- Ferm, V.H. 1969. The syneratogenic effect of lead and cadmium. *Experientia* 25:56-57.

- Ferm, V.H. & Carpenter, S.J. 1967. Developmental malformations resulting from the administration of lead salts. *Exp. Mol. Pathol.* 7:208-213.
- Forni, A., Cambiaghi, G. & Secchi, G.C. 1976. Initial occupational exposure to lead: chromosome and biochemical findings. *Arch. Environ. Health* 31:73-78.
- Gershanik, J.J., Brooks, G.G. & Little, J.A. 1974. Blood lead values in pregnant women and their offspring. *Am. J. Obstet. and Gynecol.* 119:508-511.
- Gilani, S.H. 1973. Congenital anomalies in lead poisoning. *Obstet. Gynecol.* 41:265-269.
- Hammett, F.S. & Wallace, V.L. 1928. Studies in the biology of metals. VII. The influence of lead on the development of the chick embryo. *J. Exp. Med.* 48:659-665.
- Hilderbrand, D.C., Der, R., Griffin, W.T. & Fahim, M.S. 1973. Effect of lead acetate on reproduction. *Am. J. Obstet. Gynecol.* 115:1058-1065.
- Karnofsky, D.A. & Ridgway, L.P. 1952. Production of injuries to the central nervous system of the chick embryos of lead salts. *J. Pharmacol Exp. Ther.* 104:176-186.
- Kobayashi, N. & Okamoto, T. 1974. Effects of lead oxide on the induction of lung tumors in Syrian hamsters. *J. Natl. Cancer Insti.* 52:1605-1608.
- Lancranjan, I., Popescu, H.I., Gavanescu, O., Klepsch, I. & Serbanescu, M. 1975. Reproductive ability of workmen occupationally exposed to lead. *Arch. Environ. Health* 30:396-401.
- Landrigan, P.J. 1975. Letter of May 28, from Center for Disease Control, Atlanta, to Ashley, J.T. on cancer mortality and renal disease in Idaho counties.
- Lower, W.R. 1975. Gene frequency differences in *Drosophila melanogaster* associated with lead smelting operations. *Mutat. Res.* 31:315.
- Matthews, J.J. & Walpole, A.L. 1958. Tumors in the liver and kidney induced in Wistar rats with 4'-Fluoro-4-Aminodiphenyl. *Brit. J. Cancer* 12:234-241.
- McClain, R.M. & Becker, B.A. 1970. Placental transport and teratogenicity of lead in rats and mice. *Fed. Proc.* 29:347 Abstract 575.

- Morris, H.P., Laug, E.P., Morris, H.J. & Grant, R.L. 1938. The growth and reproduction of rats fed diets containing lead acetate and arsenic trioxide and the lead and arsenic content of newborn and suckling rats. *J. Pharmacol. Exp. Ther.* 64:420-445.
- Muro, L.A. & Goyer, R.A. 1969. Chromosome damage in experimental lead poisoning. *Arch. Path.* 87:660-663.
- Oliver, T. 1916. Diseases of occupation. Ch. VII. Diseases due to metallic poisons, dust, fumes, etc. E.P. Dutton and Co., New York, 3rd ed., pp. 186-202.
- O'Riordan, M.L. & Evans, H.J. 1974. Absence of significant chromosome damage in males occupationally exposed to lead. *Nature (London)* 247:50-53.
- Paul, C. 1860. Etude sur l'intoxication lente par des preparations de plomb; de son influence par de produit de la conception. *Arch. Gen. Med.* 15:513-533.
- Schmid, E., Bauchinger, M., Pietruck, S. & Hall, J. 1972. Cytogenic action of lead in human peripherhal lymphocytes in vitro and in vivo. *Mutat. Res.* 16:401-406.
- Schroeder, H.A. & Mitchener, M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. *Arch. Environ. Health* 23:102-106.
- Schwanitz, G., Lehnert, G. & Gebhart, E. 1970. Chromosome lesions in cases of occupational exposure to lead. *Dtsch. Med. Wochenschr.* 95:1636-1641.
- Stowe, H.D. & Goyer, R.A. 1971. The reproductive ability and progeny of F₁ lead-toxic rats. *Fertil. Steril.* 11:755-760.
- Tonz, O. 1957. Kidney changes in experimental chronic lead poisoning in rats. *Z. Gesamte. Exp. Med.* 128:361-377.
- Van Esch, G.J. & Kroes, R. 1969. The induction of renal tumors by feeding basic lead acetate to mice and hamsters. *Brit. J. Cancer* 23:765-777.
- Varma, M.M., Joshi, S.R. & Adeyemi, A.O. 1974. Mutagenicity and infertility following administration of lead sub-acetate to Swiss male mice. *Experientia* 30:486-487.
- Weller, C.V. 1915. The blastophthoric effect of chronic lead poisoning. *J. Med. Res.* 33:271-293.
- Zollinger, H.U. 1953. Kidney adenomas and carcinomas in rats caused by chronic lead poisoning and their relationships to corresponding human neoplasms. *Virchows Arch.* 323:694-710.

TABLE 1
TERATOGENIC RESPONSE INDUCED BY LEAD

<u>SPECIES</u>	<u>INVESTIGATORS</u>
Chick	Hammet and Wallace (1928)
Chick	Catizone and Gray (1941)
Chick	Butt, Pearson and Simonsen (1952)
Chick	Karnofsky and Ridgway (1952)
Chick	Stowe, Goyer and Cates (1972)
Chick	Gilani (1973)
Hamster	Ferm and Carpenter (1967)
Hamster	Ferm (1969)
Mouse & Rat	McClain and Becker (1970)

TABLE 2

EVIDENCE FOR MUTAGENIC OR REPRODUCTIVE EFFECTS
FROM MALE EXPOSURE TO LEAD

<u>SPECIES</u>	<u>FINDINGS</u>
A. Rabbit	1. Reduction in Birth Weight 2. Higher Postnatal Mortality
B. Guinea Pigs	1. Sterility 2. Reduction in Birth Weight 3. Higher Postnatal Mortality
C. Rat	1. Reduction in Birth Weight 2. Reduction in Litter Size 3. Higher Postnatal Mortality
D. Rat*	1. 2-fold Increase in Prostatic Weight 2. 70% Reduction in Testicular Weight 3. Reduction in Sperm Motility
E. Mice	Chromosome Anomalies
F. Mice	Excess of post-implantation Fetal Deaths
G. Drosophila	Increased Gene Frequency

*Occurred at blood lead levels which are $\frac{1}{2}$ those currently being recommended for biological monitoring of lead workers

TABLE 3
HUMAN EVIDENCE FOR THE MUTAGENICITY
OR REPRODUCTIVE EFFECTS OF LEAD

<u>OBSERVATION IN HUMANS</u>	<u>RESULTS FROM OCCUPATIONAL EXPOSURES</u>
A. Effects on Lymphocytes	Chromosome Changes
B. Effects on Reproductive Ability	Decreased Fertility Due To: <ol style="list-style-type: none"> 1. Asthenospermia 2. Hypospermia 3. Teratospermia
C. Pregnancy Outcome From Male Lead Exposure	<ol style="list-style-type: none"> 1. Increased Miscarriages 2. Increased Stillbirths 3. Increased Postnatal Mortality

JOB PLACEMENT OF WOMEN IN THE LEAD TRADES: AN INDUSTRY POSITION

Dr. Sidney Lerner
College of Medicine
Cincinnati, Ohio

I welcome the opportunity of being with you today to discuss employment of women in the lead industry. My participation in this conference is as an independent physician. In addition to my academic affiliation with the Kettering Laboratory in the Department of Environmental Health at the University of Cincinnati, I am a practicing physician and a consultant in occupational medicine to a number of industries, including those dealing with inorganic lead. I have been called upon to express my views and make recommendations about the health maintenance of all lead workers, including women. The rationale for and recommendations made will be discussed.

Pre-placement assessments should aid in the proper placement of all workers in jobs which they can perform without endangering their own health or that of others. Unfortunately, all too often the examination performed prior to employment has been used to determine who should or should not be hired, rather than to determine proper placement. Too many examination report forms still only give the examining physician one of two options for the final opinion: accept or reject. This is too restrictive. The physician should only recommend to management justified specific medical limitations. Only that medical information specifically needed to properly place the worker should be revealed. Management has the ultimate responsibility for employment with appropriate placement of the worker. The physician should not summarily reject an applicant for a job.

Results of the pre-placement assessment can help provide a data base for future medical care -- both preventive and therapeutic -- for medico-legal issues, and along with medical information subsequently generated can help support current exposure standards or document a need for modifications.

The scope of the pre-placement assessment should be determined by a physician familiar with the physical and psychological requirements of the job. In general, it should include a medical and occupational history along with appropriate physical examinations, laboratory determinations and other studies, as indicated.

Pregnancy and the potential for pregnancy should always be considered when evaluating women applicants. There are a number of physiological changes which occur during pregnancy. These include weight gain, anemia, changes in plasma protein and lipids, cardiac output, and pulmonary function. There may be physical problems associated with balance and posture, increasing the

likelihood of injury or musculoligamentous strain. Special consideration is required in placement of women who may be nursing. A number of potentially toxic agents are excreted in breast milk, including lead.

The proposed standard for occupational exposure to lead requires each employer to offer a medical examination including a complete medical history and physical examination, complete blood count, routine urinalysis and pregnancy test, where appropriate.(1) These examinations are to be performed prior to assignment in areas in which airborne concentrations of lead are at or above the results of periodic biological and environmental monitoring. For reasons which I have stated, and since exposure to lead can be from sources other than lead in air, I feel that all lead workers should be examined prior to employment and periodically, irregardless of the air-lead level. The proposed standard does not specify what will be included in the medical history or physical examination. The reference to "complete" would hopefully assure that enough is covered. On the other hand, much more than needed may be done by a compulsive practitioner. Ultimately, the physician is required to state whether the employee has any detected medical condition which would place that employee at increased risk of material impairment to health from exposure to lead, or would directly or indirectly aggravate any detected medical condition. Furthermore, any recommended limitation upon the employee's exposure to lead, or upon the use of personal protective equipment and respirators, must also be provided by the physician. Once again, specific guidelines on the basis for making these determinations are not included. The decisions of what to do and what interpretations to make are left entirely up to the professional opinion of the physician.

Pregnancy is singled out as a special concern in the background discussion for the proposed standard regarding increased susceptibility of some workers. The initial reference is to the adult female of childbearing age, but the discussion clearly focuses on the fetus and children. It states that the blood lead level in the mother might harm the fetus without producing any clinical symptoms in the mother. There is good evidence that lead absorbed into the bloodstream of a pregnant woman crosses the placental barrier, with the fetal blood lead concentration at birth being the same as that in the mother. One can speculate that if fetal damage were to occur from elevated levels in the mother, the maximal risk may be in the first trimester, when the condition of pregnancy may not be known or be concealed. Children, for a number of reasons which are not clearly understood, may be susceptible to adverse effects from increased lead absorption at lower blood lead levels than adults. Furthermore, children tend to have more frequent effects on their central nervous system than do adults from comparable levels of lead in blood. It is apparent that for these and other reasons the U.S. Public Health Service recommended, in March, 1975, that blood lead levels in children be

kept below 30 ug/100 G blood. (2) The proposed standard could permit fetal blood lead twice this level -- a clearly unacceptable circumstance.

The current and proposed standards for acceptable occupational lead absorption are intended to provide reasonable assurance that generally healthy adults will not suffer material impairment to health from their exposure. The standards are not intended to provide this assurance to individuals having specific medical problems placing them at increased risk. This concept is acknowledged in the proposed standard.

The documentation for threshold limit values, even for exposure of healthy adults, is often quite fragmentary, being based only on animal data or limited and poorly documented human exposures. Data on exposure of the very young or very old, or those with special health problems, is even more limited and many times entirely lacking. In general, we do know that the young human often has a greater sensitivity to toxic exposures than the adult. The most sensitive tissues are generally those which are most immature and undifferentiated as one finds in the developing fetus in the early stages of pregnancy, for example, the first trimester.

The Lead Industries Association has met with representatives of the TLV Committee to bring their attention to the industry's concern for inclusion of the fetal consideration in the setting of TLV's. The objective was to have a notice placed on the list of TLV's, indicating that they are not necessarily applicable to a fertile, pregnant or lactating woman. This should apply to a substantial number of compounds on the TLV list. There was general acknowledgement of the problem but because of its complexity the issue has been side-stepped. All agreed that a more definitive position would have to be taken by the TLV Committee and others in the near future. That meeting was held August 1, 1974, almost two years ago.

The NIOSH criteria for a recommended standard for occupational exposure to inorganic lead does touch on the subject. It states that "thus a biologic standard of 0.08 MG lead per 100 G of whole blood is recommended: It provides a margin of safety in adults but probably not in children." (Emphasis added.) (3)

The proposed standard requires a pregnancy test "where appropriate." It does not define what is meant by "appropriate." This is apparently left up to the physician. Does it mean when the applicant is a woman? Does it mean when she admits to being pregnant or having missed a period?

One large company has a policy, agreed to by labor, which requires the employee to notify the company's authorized physician without delay -- I am not quite sure what that means -- if she misses a menstrual period or has any other reason to believe she

is pregnant. This is not defined.

This approach reminds me of one used in my child's nursery school. Each morning, the nurse would line up all of the children and look at each throat to see if any had obvious infection before letting the child into school. Are we to screen each woman each morning before she comes to work? How early is it necessary to determine when a woman is pregnant? Once in taking a medical history I asked a woman if she was pregnant and if so, how far along. Before answering she glanced at her watch.

More seriously, we must consider if it is adequate to know when pregnancy is established before taking steps to modify exposure. This may not be acceptable for materials which remain in the body after exposure is terminated, such as lead. Furthermore, women may have reason to try to conceal their pregnancy if they feel their job security is at risk. The proposed standard permits the employee to refuse a medical examination or biological monitoring. Exposures could then occur during the early part of pregnancy when we suspect the fetus is most vulnerable. By the time pregnancy is acknowledged or discovered, the damage may have already occurred. Examinations should be required.

Historically, lead has been used as an abortifacient, and there are reports of lead poisoning being associated with abortions and high fetal and neonatal loss and sterility. These reports, with or without control data, led to the widespread enactment by the early 1900's of labor codes forbidding the employment of women in industry involving a lead hazard. Alice Hamilton believed that women were more susceptible to lead and urged the passage of special laws to protect women in the workplace. In 1946, Anna Baetjer, in her classic treatise "Women in Industry: Their Health and Efficiency," concluded that normal women needed no special restrictions but warned that "there is every reason to believe that pregnant women might be adversely affected by exposure to concentrations of toxic substances which would usually be safe for normal women."

Section 4109.12 of the Ohio revised code relating to jobs prohibited for a child under 18 years of age states that "no child under eighteen shall be employed or permitted to work with... lead and its compounds." I would imagine that similar laws exist in other states.

Notwithstanding the past literature, there is little definitive data identifying and quantitating the risk to the fetus from exposure of the mother to levels of lead within what are considered to be occupationally acceptable limits. Much or all of the information we have is based upon the adverse effects of gross overexposure. There is a current need to apply modern epidemiological and toxicological techniques to the study and understand the effects on the fetus from exposure to all toxic agents--not lead

alone.

The Lead Industries Association has attempted to develop a combined effort with other groups to look at the problem generally, but unfortunately no productive cooperative efforts have been possible to date.

Do we have a responsibility, in consideration of what is currently known and not known, to prevent the possibility of a fetus being exposed to concentrations of lead which are acceptable to the adult working mother but which may increase the risk of material impairment to the fetus? The proposed standard, in my opinion, suggests this be done. Furthermore, I believe the same approaches should be used in occupational medicine as are followed in the general practice of medicine. Where insufficient medical information exists to prove safety, especially with regard to fetal exposures, a conservative approach is indicated. For example, the labelling for a large number of drugs carries the warning, "Safety of this drug for use in pregnancy has not yet been established."

Taking all of what I have discussed into consideration, it has been my recommendation to persons responsible for the health of lead workers that fertile, gravid or nursing females not be employed in areas where there will be increased lead exposure, albeit safe for the worker, until such time as adequate information has been developed proving the safety of such exposure to the fetus.

I believe this is a reasonable medical position. I do understand that there may be legal or other issues, but feel these should not influence what medical recommendation is made.

The final decision made by management often must take into account nonmedical factors. The Occupational Safety and Health Act requires the employer to provide a safe and healthful work environment for the worker. It does not specifically deal with the question of safety to the fetus. Whether the fetus is an inseparable part of the worker--within the law--would require a legal interpretation.

Workmen's compensation -- perhaps this should be changed to worker's compensation -- does not, to my knowledge, include coverage of the fetus. The fetus is not considered a worker. The employer does not pay premiums for the fetus, and, as such, an injured fetus is not compensated through this mechanism. An employer may then be liable to unlimited recovery in a civil suit. Negligence must be, of course, proven -- and this is not a requirement under workmen's compensation. A woman cannot bind her unborn child to release from liability. When a fetus becomes a person is open to debate, but it is one, at least after it is born, and may seek redress up to three years after its majority.

This is known as the long-tail effect.

Even if an employer can demonstrate that he has not exposed a pregnant woman to concentrations of a toxic substance such as lead exceeding any existing standard, he is not rendered harmless from any subsequent redress for liability. If, on the other hand, one can show that standards were exceeded, it would be difficult, if not impossible, for an employer to successfully defend himself. A test in the courts may be coming in which the issues regarding protection of the fetus -- whether it be a person or not -- are juxtapositioned against the right to work of all persons. A question that will have to be answered is whether the potential sensitivity of the fetus is of more concern than the rights of the fertile or pregnant woman to her job. I know of no way to predict the eventual outcome. One obvious solution is to impose standards which would protect all men and women equally, including the fetus and persons with special risk. For instance, should individuals with pre-existing kidney problems be permitted to work in areas where they would be exposed to a potential nephrotoxic agent such as lead by lowering the limits so that they could be safely exposed? Or, should we say that these individuals have specific medical problems of their own that preclude their acceptability for work in these exposures? The proposed standard takes the exclusion approach at present, and I believe this is medically reasonable.

It is quite apparent that we would be in a better position if we had more definitive information. Current data does not precisely indicate the risk to the fetus of exposure to lead at varying concentrations above background. Gathering data through human epidemiological studies will be difficult, particularly so long as the number of women in the work force exposed to lead is limited. Women tend to leave work earlier and change exposures more frequently than men, making definitive study difficult. Epidemiological studies should include women as a separate group. Many studies in the past only looked at men.

Recordkeeping remains inadequate. OSHA recordkeeping requirements should make more specific reference to pregnancy or change of job required because of pregnancy.

If, as expected, the fetal effects, if any, from exposure to current acceptable adult levels is a relatively infrequent event, we will have to study large numbers of events before conclusions can be reached. For instance, at a meeting dealing with the susceptibility of the fetus and child to chemical pollutants, Sullivan noted that if the usual frequency of an anomaly is 1:1000 newborn infants, the offspring of 23,000 mothers would have to be studied to detect a doubling in the incidence of the defect. (4) This problem was confirmed in a preliminary investigation into the design of an epidemiological study performed for the International Lead Zinc Research Organization.

Combinations of exposure--interacting environmental chemicals or drugs--may complicate any study. For example, Gardner described cretinism in the Congo caused by a dietary iodine deficiency made worse by eating cassava. A glucoside in cassava produces cyanide, which when metabolized to thiocyanate, depresses iodine uptake by the thyroid.

We must also begin to look at the children of workers, especially if we are to determine mutagenicity. Here we may begin to see effects from toxic exposures, not only to women, but to men as well, as has been reported in recent studies on male anesthesiologists whose wives had increases in abortions and fetal abnormalities similar to those of female anesthetists exposed to operating room gases.

More definitive guidelines are needed for the assessment of the fertile or pregnant women which are consistent with existing regulations. Specific sections of criteria documents and the regulations should be devoted to concerns for criteria of exposure of these women which has heretofore not been the case.

In conclusion, I would emphasize that each applicant for a job, woman or man, be individually medically assessed. Proper placement requires an understanding of the job requirements and health status of the applicant. Medical recommendations should be based on the best available information and must not be compromised by nonmedical considerations.

REFERENCES

1. Occupational exposure to lead. Proposed rulemaking. 40 Federal Register. 45933-48. October 3, 1975.
2. Increased lead absorption and lead poisoning in young children: A statement by the Center for Disease Control, U.S. Department of Health, Education and Welfare, PHS, (March, 1975).
3. Criteria for a recommended standard...occupational exposure to inorganic lead, National Institute for Occupational Safety and Health, p. V-3, 4 (1972).
4. Miller, Robert W.: Susceptibility of the fetus and child to chemical pollutants. Science, Vol. 184, May 17, 1974.

JOB PLACEMENT OF WOMEN IN THE LEAD TRADES: A WORKERS' POSITION

Claudia Prieve
Industrial Hygienist
United Steelworkers of America

The title on the program for this afternoon's session poses the question: "Lead and Women, A Unique Problem?" I would first like to comment on that question. While the peculiar biochemical effects of lead--its insidious interactions with the nervous system, kidney and the human reproductive system, and its ability to be stored for long periods of time in the body--are unique to lead, lead is by no means unique in terms of its consequences for women workers. If anything, perhaps lead has been studied more carefully than other materials in the workplace. But other substances raise a similar dilemma for women. Among them are vinyl chloride, some pesticides, anesthetic gases and other materials which have been mentioned during this conference.

Before I focus on the subject of job placement in the lead industry, I would like to bring to your attention another kind of workplace where more and more women are starting to work. The jobs these women perform are not just hot, heavy and dirty, but expose them, and the men who work there, too, to dozens of known carcinogens, mutagens and teratogens. Workers who spend their careers at these jobs are known to be ten times more likely to die of lung cancer and seven times more susceptible to kidney cancer as a consequence of their work. They fall prey to a host of other ailments as well. Yet no one has begun to look at the potential adverse effects of this exposure to the future children of these workers. I am speaking of coke oven workers.

Tens of thousands of workers in the United States are exposed to the volatile material, the gases and particles, which enter the atmosphere when coal is baked in the coke oven to make coke for the blast furnace. These workers who inhale this air every day are not just exposed to a single substance, such as lead or vinyl chloride, but to thousands of different chemicals which arise during the destructive distillation of coal in coke ovens.

Among these chemicals is benzo(a)pyrene, a compound that has been demonstrated to be a transplacental carcinogen in animals. Nor is benzo(a)pyrene the only such substance in coke oven emissions. Dozens of compounds that are chemically similar to benzo(a)pyrene are formed when coal is coked. And the coke oven worker inhales all of them. Many of these materials have never been tested in animal experiments for their toxic or cancer-producing potential, much less their effects on offspring.

Yet women are taking jobs at the ovens. In some plants as much as 20% of the coke oven workers are now women. Most of these

women start out in the lowest-paid jobs. These are generally the "topside" jobs where exposure to emissions is the heaviest.

What should these women do? How should they be advised? Should they be protected in any special way?

Unfortunately, most of these women are not even aware there is a potential problem. Few coke oven workers are even told of the cancer hazard associated with their jobs, in spite of the fact that the cancer-producing properties of coke oven emissions have been thoroughly documented and are familiar to every steel company and its medical personnel. Hopefully we will soon have a standard that will require coke oven workers to be informed of the hazards to which they are subjected. Thus far OSHA has paid little attention to the possible second-generation effects of coke oven exposure.

So, lead is not unique in the problems it presents, particularly in terms of its consequences to women workers. Nonetheless, the upcoming lead hearings will probably be the first time that the issue of women in the workplace is dealt with in any great depth.

The lead hearings are of real concern to our Union. Many lead smelters and refineries are organized by the Steelworkers. Our members work in battery manufacturing, battery breaking operations, and scrap operations. Steelworkers pour leaded steel alloys. These are a few of the more obvious contracts our members have with lead.

Many companies that handle lead have a more or less formal policy of simply not hiring women into the lead areas of their operations. Sometimes that means the whole plant! This practice has gone on for years and continues today.

At least two of the companies with whom we bargain have allowed women to work in their smelters--that is, up until just a few months ago. Suddenly, these two employers decided they had better "do something," so they issued a policy saying that women of childbearing capacity could no longer work in lead areas.

These companies' actions raise a number of questions--tough questions that we, as the Union, OSHA and the companies must face squarely:

Would the courts judge such a policy as discriminatory against women who are moved off of their jobs? What pay and promotional opportunities should be open to these women if they cannot return to the lead areas for some time? If the highest job categories are in the lead areas and women can no longer work there, shouldn't equal job opportunities be provided for them in the areas where they are allowed to work?

Why couldn't men who are left to work in the high lead areas of the plant allege discrimination, particularly in light of what we know of the effects of lead on spermatogenesis? What if a male lead worker has a child that is born with functional disturbances? Is his wife not also entitled to sue on behalf of that child?

Many women are the breadwinners for their households. Consider, for example, a woman working in a smelter. The smelter is the only employer in town and she is the sole support for her family. She has already had all the children she wishes to have. She has an intra-uterine device (or takes the pill), but she is still barred from her job. Should this woman be driven to the extreme of having a tubal ligation or hysterectomy so that she can present the company with a piece of paper certifying she can no longer have children, just to get her job back?

How can OSHA or any other agency mandate that workers be removed from their jobs for health reasons and yet close its eyes to the personal and dire consequences of such a mandate?

Removal from the job without penalty is a protection for workers our Union is seeking in the coke oven standard. But OSHA seems to feel this is not its responsibility. We do not understand why. OSHA has reserved the right to mandate an employee's removal from the job. If the Secretary of Labor has that authority, then by parity of reasoning, he must have the authority to protect that employee from any adverse employment effects resulting from the removal he mandated. Clearly the power is there.

Is it a fear that we are talking about some kind of welfare?

No. That can't be. We are talking about people who want to work. Otherwise why would they have a tubal ligation, or agree to take chelating drugs as if they were candy, or submit to periodic IV treatments? They want desperately to work.

Does OSHA feel that this would be some kind of windfall for these workers--a stroke of undeserved good fortune?

Certainly not. We are concerned here about sick people or unborn children who could be impaired for life.

We ask only that OSHA recognize the full impact of its regulations from a moral standpoint.

During the recent coke oven hearings, Dr. Eula Bingham of the University of Cincinnati expressed her concern that workers not be penalized financially when they are taken off their jobs for medical reasons:

"My own view is, and it is my own view, but I believe it was shared by many people on the Advisory

Committee, the spirit of the Occupational Safety and Health Act is to truly protect the health of the worker.

"I have been indoctrinated with the point of view that it is impossible to have a comprehensive health program in an industry without providing for physical examinations and surveillance of the workers.

"If a worker is afraid to take a physical examination, if he is afraid to find out that something is wrong with him, then the purpose of the Act is negated.

"I have heard it said that there is a question about the legality of this. If there is a legal question I think we should put something like this on the books and have it tested in court. If we have to go back and make the Act specific, we should do so.

"It is to me an impossible situation for a worker to be afraid to take a physical examination because he is going to lose the job that he uses to feed his family. It is unbelievable."

OSHA argues that it does not require a worker to take a medical exam which might reveal problems and result in a loss of job or wages. Medical examinations are voluntary, according to the Secretary. Unfortunately, workers who most suspect they have something wrong with them are least likely to avail themselves of a medical examination. Thus, under OSHA's scheme, workers who need medical attention the most will obtain it the least often.

In the present case, that of women in the lead industry, the issue becomes more difficult. We are not allowing any woman the right to refuse to take a medical exam, the results of which may affect her earnings. A woman does not have the option of refusing an examination which would reveal she is a woman. Unless she disguises her sex, it is obvious she is a female and she will be transferred off her job.

Yesterday, Dr. Corbett spoke of the effects of anesthetic gases in the operating room on persons working there--the increased incidence of still births and miscarriages among operating room personnel. His study demonstrates that from a reproductive standpoint, women appear to be more susceptible to these gases than are men, even though offspring from both males and females may be affected.

The same statement could be made in reference to lead. Women appear to be more susceptible than men. Whether or not this is in fact true remains to be established. Nonetheless, as Dr. Corbett indicated earlier, it costs relatively little, a hundred dollars or so, to install a scavenging device that will

remedy the problem of stray anesthetic gases in the operating room.

One wonders, or at least I wonder, what would happen if instead of \$100, it cost \$100,000 or even \$1,000,000 to correct the problem? I have a feeling that no one would seriously suggest that we move all women of childbearing age from the operating room. Women as operating room nurses and anesthetists are too well ingrained in our system to give them up so easily. After all, who would hand the doctor his scalpel or wipe his perspiring brow? We could staff the operating room with males, but there would not be enough male nurses, so we would have to pay male doctors to help and you know how much money they want!

If you will, contrast this with the plight of women in the smelter or battery plant where there are jobs that pay well and a plentiful supply of men to fill those jobs. There have been very few women in smelters in the past. Why change now? So society says, "We're going to keep those women out of there for their own good."

How often men mistake their prejudices for the laws of nature!

In the thirties and forties, many states passed women's protective laws which took a variety of forms. Some of these laws prohibited women from working third shift, in order to protect the "weaker" sex from physical harm. It did not matter whether a woman knew karate or carried an ice pick--she was viewed as being more susceptible to attack than a male worker.

Other states passed laws that kept women from lifting objects weighing more than a certain number of pounds. The fact that a woman was stronger than many of her male co-workers did not detract from the requirements.

In 1965, when Title VII became effective, these protective laws were allowed to stand. A series of law suits ensued and the courts ruled that women could not be treated as a class, but must be regarded as individuals. Subsequently, the Equal Employment Opportunity Commission changed its position, saying that employers could not discriminate against women based upon a state's protective laws.

There is a clear parallel between these paternalistic protective laws and the present practice of restricting women from jobs involving exposure to lead.

Individual women are able to defend themselves against physical assault or lift fifty pounds with ease. Just as a woman may take steps to protect herself against attack, she may also take steps to protect against having an impaired child, such as using a contraceptive device.

Thus women are not to be reckoned with as a class, but are to be regarded as individual human beings.

I would like now to spend a few minutes talking about another type of protective device which presents enormous problems for women workers everywhere--respirators. Whenever I visit one of the coke ovens or smelters or battery plants where our members work, I am handed a respirator to wear. First, I take the respirator out of its box and strap it snugly over the top of my ears; then I position the facepiece over my nose and mouth and fasten the bottom strap. Generally, the lower strap will not adjust to fit my neck size, so I struggle to tie a loop in the strap to take up the slack. Now I have the respirator adjusted correctly--except for one thing. If it is a half-face respirator, there is a big gap at the top of my nose, since my face is not long enough for the device. I can shift the respirator to a lower position on my face, but then I find I'm breathing the bottom of the chin cup instead of through the cartridges.

Of course, air follows the path of least resistance (try drawing liquid through a straw with a hole in it sometime, if you want to prove this yourself), and since there is now a large gaping hole between the respirator and my face, the air will elect to follow that path, bypassing the filtering mechanism entirely. There is usually no point in my asking for a different respirator. One model is generally all they have in stock.

When I go into a plant, I am only there for a brief time, perhaps only a few hours or a day. But as I go around and visit work areas, I notice other women and men, whose features are very small or unusual in some way--small chins, concave cheeks, broad noses or jaws--who are trying to wear respirators. These workers are exposed to lead every day, and their ill-fitting respirators are providing them with a false sense of security.*

Those of you who have worn respirators know that respirators do not come in dozens of different sizes like shoes. Respirators are designed for the "standard man," not for individual faces. The American Conference of Governmental Industrial Hygienists'

*"The ability to form a good facepiece-to-face seal depends on respirator design and facial features, and is usually the most important factor in obtaining proper protection with an air-purifying respirator, particularly of the half-mask type." (ANSI "Practices for Respiratory Protection" Z88.2, p. 23.)

Manual on Respiratory Protective Devices* recognized this problem more than a decade ago:

"It is difficult to fit women who must wear personal respiratory protective equipment because no commercially available full face masks and few half masks, are sized to fit them. Although the Bureau of Mines' approval does not specifically exclude women, they are not required to be fitted in the Bureau's evaluation of a respirator."

I bring up the subject of respirators for two reasons:

(1) It is an excellent example of how our society designs for the "average" man. The Germans designed and fitted gas masks for horses and dogs during the War. There is no reason we cannot fit women with respirators. Part of the problem is economics: Respirator manufacturers are not going to design a lot of respirators for women until the market is there to make it a profitable venture. It is a costly undertaking, marketing a new respirator--to design and make a new mold and get NIOSH to certify the final product. It must pay off. Powered respirators might alleviate the fitting problems for women, but few employers are willing to pay a couple of hundred dollars per worker to provide adequate respiratory protection.

(2) The issue of respirators further illustrates the box women are in. Perhaps some women could stay in the smelter, if appropriate protective equipment were provided. But when there is no respirator that fits properly, how can a worker keep from getting an elevated blood lead level? Even a worker who washes thoroughly before eating, who does not smoke, and who wears a respirator faithfully may continue to have an elevated blood lead. A respirator that does not fit does not protect.

Recently, one company with whom we bargain issued a notice to their employees announcing that anyone whose blood lead level exceeded the magic figure of 80 micrograms for more than 90 days would be discharged. The company regarded a high blood lead as incriminating evidence that a worker was not wearing his or her respirator or following proper hygienic practices. Believe me, I could not maintain a blood lead of 80 micrograms no matter how hard I would try, because no commercially available respirator fits me properly.

What is the solution? Accommodating individual differences. OSHA's standards must accommodate individual differences.

**The Respiratory Protective Devices Manual*, American Industrial Hygiene Association and American Conference of Governmental Industrial Hygienists, Braun and Brumfield, Ann Arbor, Michigan (1963).

Let me make one final comment on the subject of economics. Inflationary impact statements have become very popular in the Ford Administration. You can bet that the Council on Wage and Price Stability will be at the upcoming lead hearings to testify that it will cost too much to clean up the lead industry so that it is safe for both men and women.

The following thought has occurred to me later. What if the Office of Management and Budget had been around in President Lincoln's term, as it is now? Would the OMB have required him to prepare an Inflationary Impact Statement before he issued the Emancipation Proclamation? Assessing the costs and benefits of social legislation is a tricky business. Few would argue that freeing slaves was not costly to society. And the benefit? Who benefited? No one, except the slaves!

There is an ancient Greek legend about an innkeeper whose name was Procrustes. When travelers stopped at his inn for the night, he would tie them to an iron bed and then, depending upon the size of the victims, he would either stretch their bodies or cut off their legs so they would fit. Hence the term "Procrustean bed" which is a scheme or pattern into which someone or something is arbitrarily forced.

When companies or OSHA force a woman out of certain jobs and into others because of the possibility she might have a child, they close their eyes to individual needs and differences. The pressures exerted by companies and the government that could cause a woman to have a tubal ligation in order to keep her job are no less immoral than the innkeeper who lopped off his victim's legs.

JOB PLACEMENT OF WOMEN IN THE LEAD TRADES:
THE DEPARTMENT OF LABOR'S POSITION

Dr. Morton Corn
Assistant Secretary for Occupational Safety and Health
U.S. Department of Labor

Members of the Panel, ladies and gentlemen. As the final speaker on this platform, I find that many of the previous speakers have touched upon issues I wish to address. However, I think the remarks I have formulated are a consistent unit and I will, therefore, proceed with them. I have not addressed myself to the content of the proposed OSHA standard for lead. That has been available and I assume that those in the audience are familiar with it.

As the prior speakers have said, the consequences of exposure of women in the workplace to excessive amounts of lead were documented in the second decade of this century in most vivid terms by Alice Hamilton. She also documented the effects of lead intoxication on the mother of the newborn child and on the newborn child. Therefore, we are not approaching, as we have learned, a subject that is new in any sense of the word. It is, therefore, with dismay that one comes to the realization that we still do not have at this point in time reliable dose response data with respect to the effects of lead poisoning on women in the workplace.

I speak to you as the head of a regulatory agency charged with promulgating a standard in this area, as well as enforcing an existing standard. The existing standard is for airborne lead.

The proposal for lead in the workplace was published in the Federal Register in the fall of last year. That is a proposal in the full sense of an OSHA standard, incorporating requirements for other than airborne lead, for medical surveillance, for monitoring, for personal protective equipment, for engineering controls, and so on.

The proposed standard is still pending. Therefore, it is not appropriate at this time to address myself to any conclusions with regard to the posture of the Occupational Safety and Health Administration in this area because the record is still open. We have put forth a proposal reflecting on the ideas we held at the time of issuance.

We will not draft a final standard until that record is closed and all viewpoints can be fully examined. However, it is possible at this time to address certain of the questions which concern us as an agency, which were flagged in the preamble and which have been mentioned here.

At the time of issuance of the proposed standard it did not

appear to us that there were sufficient data to point with any degree of confidence to definitive answers on where to set limits for protecting pregnant women.

We hope that the public record, as well as this conference, will point to some guidelines.

We are also concerned in the lead standard with another susceptible group, those with predisposition to sickle cell anemia. As indicated earlier on this platform, we should be equally concerned with the susceptible male for some of the effects passed on to the next generation.

We are aware that there are complicating aspects associated with any policy for a susceptible group in the work environment. Initially, let me say that the most desirable solution is to deal with all of those exposed at the level which is safe for a susceptible group.

However, we do not know at this time if it is possible to treat both sexes equally in this way by engineering the environment to a sufficiently low level of lead exposure; there is a finite level of exposure which can be engineered. It is not zero.

In the absence of a dose response curve for lead, we do not have a target to aim for as a tolerable daily intake via either the respiratory system or the gastrointestinal tract. Therefore, we cannot be sure that setting the lowest feasible engineering level will still offer sufficient protection to susceptible groups.

If we abandon the optimum approach of equal protection, which is a safe level of protection for both sexes or for other identified susceptible groups, we enter a world of options for regulation, each of which is associated with problems.

Some of the complicating aspects of each of these approaches intrude upon other social mores in our society, and in some cases on other areas of governmental regulation.

For instance, if OSHA takes a position that the employer must reassign a susceptible group to another job, as Claudia Prieve mentioned, will OSHA also take a position with regard to retention of wage rate?

If OSHA takes a position that susceptible groups cannot work in a certain job situation, will OSHA be in conflict with the entire thrust in our society for equal economic opportunity for all?

If OSHA endorses a position of full disclosure of the facts to susceptible groups, leaving the decision-making capacity to the individual, is OSHA ignoring other pressures on the individual?

I spoke to one woman in a lead facility, who very clearly indicated that even if she fully understood the potential effects of continued work with elevated exposure to lead, which she admitted she did not understand, she would have to decide in favor of continuing employment because she was the sole breadwinner and was feeding two children at home. This was a day-to-day concern, whereas the possible effects of lead on another generation were distant, in the future, and difficult to understand. When one appreciates such pressures, it is difficult to take a position of full disclosure without attendant safeguards. Can any governmental agency, in good conscience, offer as a viable solution full disclosure alone to an individual?

The above are only a few of the questions which are demanding our attention in the Occupational Safety and Health Administration. They are not easy questions to answer. Almost all options available seem at this time to have major difficulties associated with them.

We do not claim at this time to have the best answers. We are seeking the best solutions. We are soliciting help. We solicit the help of others who believe they have data and personal insights which will clarify the situation and make the decisions easier.

When I was a graduate student, I often read the words of Pasteur, which were engraved in stone on a college building. These words are particularly appropriate to the topic we focus on today, although Pasteur spoke of scientific research. In a way, our efforts at standard-setting for agents in the workplace is pioneering. So it is not surprising that these words appear appropriate. They were, I believe, as follows:

"Life is short and art is long. The experiment is difficult and decision is perilous."

We are on the forefront of scientific knowledge in the health standard-setting process at the Occupational Safety and Health Administration. Our standards are seminal efforts. There are aspects of standards development which are research in the truest sense of the word.

We are drawing on the data available to present our best judgments in the form of proposals for standards. We are then, through the hearing procedure, probing society's views to establish a course of action that will reflect the best judgment of society because it considers the best available evidence and moves in a direction that society wishes to proceed in the presence of this evidence.

It would be remiss of me at this time not to call your attention to what is firmly impressed upon my mind. We are discussing

what appear to one in my position to be extremely important but relatively esoteric aspects of this subject. However, Congressman Daniels held hearings recently, which indicated that at two lead facilities in this country people are exposed to concentrations of lead 35 times the current standard of two-tenths milligram per cubic meter.

There was gross contamination on work surfaces which could only lead to excessive gastrointestinal tract intake by employees. Subsequently, OSHA reviewed all of its previous inspection contacts with lead facilities, some 800 all told. In response to an offer from the National Institute of Occupational Safety and Health, we have transferred the names of literally hundreds of facilities which had concentrations of lead-in-air in excess of six-tenths of a milligram per cubic meter, or were undergoing abatement for periods of more than one year. We transferred these data so NIOSH can proceed to medically examine persons in these plants to assure the persons and others, including OSHA, that these individuals do not, indeed, have excess blood lead concentrations.

What I am saying is that today, in the United States, we are a very, very long way from achieving the existing standard for occupational exposure to lead. We believe that the two facilities under scrutiny by Congressman Daniels' Committee in the hearings on lead were typical of the situation that exists in primary lead smelters, secondary lead smelters, and battery plants in this country.

Because of these revelations, OSHA is giving primary emphasis to employee exposure to lead in the United States today.

There is little doubt that this conference will serve as a major contribution to the pool of knowledge on this subject. It has been my pleasure to be here with you, and to present to you our concerns.

My role has been, of necessity, and somewhat ungratifying to you, I am sure, to raise questions. I express my appreciation to you and to the members of this society of which I am privileged to be a member, for this most timely conference. Thank you.

DISCUSSION

Sue Nelson, Legislative Associate
House Subcommittee on Manpower, Compensation, and
Health and Safety

Wayne Brooks, Director
Occupational Safety and Health
Organization Resources Counselors, Inc.

MS. NELSON: Let me preface what I have to say by a disclaimer that the views I will express are my own and may or may not reflect the views of the Chairman of the Subcommittee, Mr. Daniels.

For discussion purposes, I would like to consider a question that was raised earlier on whether or not lead is truly unique. It may well be that lead is not unique in its greater toxic effects on women of childbearing age. There may be many other substances with such properties.

However, in view of the fact that much public attention has focused on lead, the lead standard may become a landmark issue for OSHA and for the workers of this nation.

It is in this respect that the public policy formulated on lead becomes especially important. One of the critical issues will be the writing of standards which up until now have been directed primarily at the healthy adult white male worker of 154 pounds of body weight. It is in this connection that I would like to consider from another view.

If it were determined that male workers of childbearing age were the high risk group because lead caused greater damage to the male reproductive capacity than to women in the general working population, would there really be any question in the minds of policy formulators, industry, or even in the courts, which may be called upon to review the lead standard, as to where a permissible exposure limit should be set? I contend if males were the high risk group, there would be no issue.

Now, with Dr. Infante's presentation we may have changed the issue somewhat in the course of this afternoon. Should there, however, be an issue concerning women and/or men of childbearing age or workers with sickle cell trait? I would like for a moment to look at the law in this regard.

Section 2(b) of OSHA says that "The Congress declares it to be its purpose and policy to assure, so far as possible, every working man and woman in the nation, safe and healthful working conditions and to preserve our human resources."

And Section 2(b) says that, "In so doing, OSHA is

instructed to provide medical criteria which will assure, insofar as practicable, that no employee will suffer diminished health, functional capacity, or life expectancy as a result of his work experience.

"The Secretary of Labor is directed in promulgating standards under Section 6(b) (5), in setting standards dealing with toxic materials of harmful physical agents, to set a standard which most adequately assures, to the extent feasible, on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity even if such employee has regular exposure to the hazard dealt with by such standard for the period of his working life."

The guiding language here concerns functional capacity, which clearly would be the human reproductive capacity regardless of sex, and standard-setting on the basis of the best available evidence.

The House Committee Report stated that "The Secretary of Labor must not be paralyzed by debate surrounding diverse medical opinions." In this connection, the question of what we do or do not know cannot be the guiding factor, or the determining factor, in paralyzing OSHA from setting its standard. In short, we do not have to bring the retarded infant into the hearing room to prove the case.

I would also like to point out that in controversial cases, the Supreme Court has declared that the fetus is part of the woman. Furthermore, the functional capacity--in this case the reproductive process--is clearly an appropriate grounds for consideration during the standard-setting procedure.

In the absence of protecting women or men of childbearing age, affected children can bring suit against the employer. This is currently happening in the case of a child born to the wife of a kepone-exposed worker.

Furthermore, if the sons of the kepone workers, who are now approaching their reproductive years, prove to be sterile, as are their fathers, these young men may well at some future date also bring suit.

Would it not, therefore, be the wiser course of action for policy formulators, scientists, industry, and workers to join in a standard for lead exposure which protects all workers?

Dr. Corn has pointed out that we will have some extreme difficulties in so doing, but would this not be the far better course of action rather than tying these cases up in the courts and going all the way to the Supreme Court for years to come?

Clearly, workers of both sexes have a right to demand such protection under the law, and I find no directive in the law that the protections of the Act extend only to healthy adult white males of 154 pounds body weight.

Finally, in the case of lead, the greatest hazards may likely occur in small businesses, lead storage battery plants. This was brought out at the House Committee hearings. These businesses are often nonunionized and have no industrial hygiene departments associated with the operation.

We always hear of the terrible problems facing small business operators. They are overburdened by Federal regulations. These operators claim they need special help. In response, Congress has legislated a number of programs for their assistance. But I believe it is time we protect the workers, the employees in small businesses.

Here in these small lead storage battery shops is where you will find more women workers than in your larger lead smelters.

Yesterday, one of the questions concerned what can we do now to protect our women workers. I believe there is something that can be done. There are 435 House offices and 100 Senate offices that can be visited. Public policy formulators need to know the issues, particularly concerning the exposure of women workers in small businesses. They need to know that the women of this country won't be silent on the issue of standard-setting for men only. They need to know of our concern for preserving our human resources.

MR. BROOKS: You have already had substantial intellectual fare, and there is not much time nor need for me to add some garnish. I should like to extend Dr. Corn's remarks beyond the parochialism of scientific research in the workplace. There is no more important topic in front of us than workers' health and safety. The community's most important economic, social and human asset is the totality of its working jobs. If by medical discoveries, we are making more occasions for persons to be unemployed, we are not satisfying the companion obligation of making the workplace compatible with the physical conditions of such people.

The underlying questions which the conference is confronting are not within the scope of any academic discipline or professional assignment as we know it. Workplace health involves our economic and social structure, our labor agreements, the physical design of plants, equipment, work methods, product quality, hours of work, selection and training of workers--including sex and nationality.

Dr. Prieve said, "I can't get a face mask built for a woman." This raises even further questions about our established

assumptions concerning the workplace and the worker. We probably assume the workers to be white, male, literate. We probably also assume for our purposes here that those of high susceptibility to disease will have been screened out.

The Occupational Safety and Health Act; the requirement that there be no discrimination against race, religion or sex in hiring or promoting; and the new law and regulations on employment of the handicapped require our most refined attention in order that these goals may be achieved.

There are many difficult questions raised. We should not resign ourselves to their having to be answered in the courts. The opportunities which the Society for Occupational and Environmental Health gives to discuss these problems in common concert and good will is the pride and purpose of this organization.

In 1320, Alfonso the Learned, King of Spain said, "Had I been present at the creation, I would have had some helpful suggestions to make to the Creator." We are present at the creation, and we bear the obligation to make some helpful suggestions.

QUESTIONS, ANSWERS, COMMENTS

MS. MOLLIE JOEL COY: I am a medical student from Johns Hopkins, working with the Oil, Chemical and Atomic Workers' Union. I have a few remarks to make about the panel, and I wondered if Dr. Corn might answer some of these comments at the end.

We should be concerned about the proposed standard for occupational exposure to lead, issued by OSHA in October of last year, for two reasons. First, because we are concerned about the exposure of our workers, men and women, to lead. But second, because this is the first OSHA standard to set specific standards for women as a subgroup of workers at risk, and if the standard is adopted, it will become the precedent for many others.

I have three points I would like to make. First of all, the standard, as it is now proposed, is too high for any worker, male or female. Dr. Samuel Epstein, President of the Society for Occupational and Environmental Health, has criticized the standard.

OSHA recommends an exposure level of 100 milligrams per cubic meter. This exposure level is based on two assumptions: that it will give blood levels between 40 and 60 milligram percent; and that such a blood level range represents, and I quote, "an adequate margin of safety." Both these assumptions are invalid.

Without going into the research involved, only an absolute maximum of 50 milligrams per cubic meter could be considered acceptable if we accept the 40 milligram percent margin as a

limit, which we do not. Any standard lower than that seriously endangers all workers, men and women.

Second, the standard sets a dangerous precedent of discrimination against women. The OSHA proposed standard directs that each employer shall provide a medical examination which includes a pregnancy test, where appropriate. A woman found pregnant could be denied a job or transferred out at a cut in wage and seniority. So women who are not pregnant, as well as all men, are not protected, and the pregnant woman is severely economically penalized for her decision to bear a child. All standards should be set to protect the most vulnerable workers in the plant and the work force at large.

Third, this is an excellent example of how the easy phrase "Protect the women and children" actually protects industry and endangers male workers as a result. In fact, blood lead levels greater than 29 milligrams, well below OSHA's supposedly adequate margin of safety of 60 and the U.S. Public Health Service's level of 30, have been shown to result in abnormal spermatogenesis. That means abnormal sperm are produced. This has been associated with chronic impairment of reproductive ability. So the men, as much as or even more than the women, are at a disadvantage in this legislation.

For all of these reasons, the OSHA proposed standards are totally unacceptable.

DR. CORN: I indicated in my remarks that the OSHA issuance last fall was a proposed standard. The agency acted on the basis of information available to it, on a range of proposed air standards between 50 and 150 micrograms per cubic meter suggested by our research wing, NIOSH. And the purpose of the hearing is to bring forth precisely the type of comments you are bringing forth, if they can be documented.

I need not tell you that OSHA is one of the most tumultuous agencies in government. We have consistently been legally challenged regardless of what position we take, and, therefore, we are particularly sensitive to the adequacy of the documentation.

I cannot address myself to the issue of the blood level at 29 milligrams per 100 grams of whole blood. If, indeed, you can document that, I would strongly urge you to put it on the public record.

MR. SID PRASAD: I work for the New York State Health Department. I have two comments to make. Number one, listening to the speakers in the past two days, some of us may have started to believe that because of so many occupational hazards and the proliferation of chemicals into our daily living, the chances of survival of the human male, as well as female, are worse today than 10,

20 or 50 years ago. But the truth is that expectation of life is much higher today than anytime in the history of mankind. At the same time, we find that the expectation of life for females is much higher than the males and so is the infant mortality. The infant mortality for females is much lower than for males.

I am simply trying to say that we should not be so much concerned. This does not mean that I am suggesting that we should stop improving the health condition of the workers. On the contrary, we must try hard within economic constraint until all sources of health hazards to all workers, whether male or female, are eliminated from the workplace as well as from the home environment.

The second point I want to make is that another source of data for epidemiological work can come from state vital records such as the birth certificate, the death certificate, and the fetal death certificate. We collect information on occupation and industry, and we ascertain different kinds of malformations at birth. We ask several other social and economic questions. All this information appears on birth certificates. We have the data to correlate some of the malformations on birth certificates with the occupation and industry of mothers, as well as with the occupation and industry of fathers.

DR. BRIDBORD: I would like to address the first point that the gentleman from New York State raised. If my memory is correct, I don't really believe that the life expectancy in the United States has changed appreciably in the last 20 years in spite of some very great advances in the field of medicine. And I am not sure that this is a very good record to stand on.

DR. INFANTE: I would also like to make a comment about that. Certainly, we have reduced mortality from some causes--from infectious disease because of better therapy and antibiotics--only to die from chronic diseases. I think that we are now in the midst of an epidemic caused by industrialization following World War II. This is what we are trying to work on.

My second point is this: to look only at birth defects is, I think, an insensitive monitor of fetal development and child growth because what concerns us about agents that are mutagenic is that their effects are passed on to future generations. Some fetuses are being aborted through a self-selection process, but what we are also concerned about is the genetic load for future generations.

One of the animal studies I presented showed that when males were exposed to lead, there was excessive mortality not to their offspring, but to the following generation of offspring. This shows genetic damage through the sperm, and these are the things we are concerned about in terms of genetic toxicology.

DR. CORN: I would like to caution the gentleman about the hazards of dealing with the average. What I am most impressed with in my present position is the range of risks that we assume at work -- very high risks to very low risks. And what this Act addresses itself to is that all those at work will be able to take a job with the assurance that they are, indeed, working in a safe and healthful workplace. You can get into the trap of dealing with the statistician, who drowned in a stream with an average depth of two feet. He fell in a hole. So I wouldn't talk in the terms you are using about the subject.

MS. CLARA SCHIFFER: I have a comment for Dr. Corn and a question for Dr. Lerner.

Dr. Corn, I was glad to see you taking notes on the need for fitting respirators to women. Several people have recently spoken to me about the need for safety shoes that will fit women. So would you add that to the list? And maybe that means you had better go back over all these male standards and see how they need to be adapted so that they are applicable to women.

Dr. Lerner, this is a question for information. I have no idea of what it would cost to develop the technology to make the lead workplace clean. But what has the Lead Industries Board and the lead industry been doing to develop this technology?

DR. LERNER: I think Dr. Corn actually addressed himself to that remark. The current situation in the lead industry is far from what the lead industry would like it to be, or what I think anyone in this room would like it to be. I can say that it is better than it used to be. Maybe that is not saying a lot. I don't know how much better because I don't know how bad it was. But I know that today there is lots of room for improvement.

And what we are talking about is the real difficulties of even meeting the current standards of 0.2 milligrams per cubic meter of air. Frankly, I think if we could meet 0.2 today, we would be in very good shape. Maybe not as good as we should be, but far better than where we are now.

I don't know what it would cost to do that. We have two groups of economists working on that very question now; one from the government and one sponsored by the lead industries. I haven't seen either report, so I don't know what the answer will be.

MS. FRANCINE WHATMAN: I am at the Yale Medical School. At this very valuable conference, attention has been focused almost exclusively on occupational hazards resulting from chemical exposure or physical stress culminating in possible trauma. I would like for a moment to redirect our consideration to those females and males occupying sedentary jobs, who are at greater risk for

cardiovascular morbidity and mortality.

Since we are particularly concerned with females, there is a very great proportion of the employed female population working in clerical jobs with a 9:00 to 5:00 routine, who have little opportunity for a concentrated physical activity program.

What I would like to see considered at some future date is the possibility of employers providing physical exercise facilities, if not programs, for their employees. While this may be viewed as a prophylactic measure as opposed to the more dramatic elimination of noxious agents in the workplace, I suggest that its consequences might be just as dramatic.

DR. LERNER: Those are very interesting comments and I am all for exercise. But there is a danger that you run into. One large company had two executives drop dead while exercising. So you have to be careful of who you exercise and how fast.

MS. CATHERINE JOHNSON: I am from the University of California at Berkeley. I am speaking for a group of 23 women who met for the first time during lunch to discuss our reactions to the proceedings so far. I want to share our discussion with you. We met because we were concerned about the tone and the perspective of the conference. We would like to make the following criticisms and recommendations to the group as a whole. Furthermore, we request that these issues be made the basis for an open discussion that is scheduled for tomorrow, and that they be included in the conference proceedings.

We want to state that we do not see women's occupational health as strictly a medical and scientific problem. That is the way it has been discussed today. We see that women's occupational health includes social, psychological, economic and political factors.

We want to see a holistic sense of women's occupational health developed. We are critical of the narrow view of women as reproductive vessels. Both men and women are involved in reproduction. Reproduction is not solely the women's responsibility. It is society's and women should not be penalized by being denied the right to work or by the lack of day care facilities.

MS. NAOMI FADIM: I am from HealthRight in New York City. Another problem that we have with the conference is that it ignores specific problems women face, which directly affect their health, while concentrating on problems that should best be discussed in the wider scope of problems for all occupational workers.

What has been ignored are problems that are produced by sex oppression primarily by male bosses, problems that have to do with the long work week that all women have, usually because they have

two jobs instead of one; problems of no day care; the stress of dead-end jobs; the fact that few women have union protection; the problems of organizing women so that they can have safer job conditions; and, finally, the problems of third world women, both here and abroad, who, generally speaking, have to suffer under worse conditions than other parts of the population.

We also object to the fact that discussion at the conference was primarily in technical terms, and directed either to people who were already familiar with the material and, therefore, did not need to hear it again, or to people who were not familiar with the material, and who would not have been able to understand the language in any case.

It is a perfect example of scientists who are unable or unwilling to speak to the people, to the workers who are primarily concerned with the hazards they are discussing.

In addition, the discussion did not acknowledge personal experience. It was not discussion. It was lecture. There was no way for people to respond in any open way to the things that were being brought up.

One of the suggestions we would like to make is that in future conferences all papers be written up; that abstracts be written up and handed out to everybody, not just the press; and that discussions of the results be undertaken rather than a rehashing by the scientists who did the research.

We also reject the statements made by some of the speakers to the effect that if you educate the workers they will have free choice. We think this is an absurd and condescending statement, given the present unemployment rate in this country.

My final point before Sherry goes on is that we object to the fact that the discussions with labor were held at night, out of the range of the general focus of the conference, where few people could come.

MS. SHERRY LIBERWITZ: I am with the National Women's Health Network and the DES Action Project.

We would like to broaden the definition of workers to include all women workers, including those who work in offices and in unpaid jobs in doing housework and childcare in the home. We would also like to question the underlying assumption that the burden for making changes in hazardous conditions is on the workers. The burden of change should not be their responsibility. It is the management that created these conditions and caused these problems. And management must accept the responsibility for the unsafe conditions they have created. We must recognize that if blame comes to one group of individuals, it is not the workers. They are

simply in the position that was created for them.

Finally, we support the statements of the women workers who spoke last night, and we are not opposed to the emotional feelings that they presented because we don't feel they preclude scientific analysis. While we support scientific research, we believe knowledge must grow out of personal experience. The scientific results presented here are simply one means of describing certain problems. It is equally valid to provide personal descriptions of these hazardous conditions because such accounts can speak to scientists, as well as those who have not had access to scientific background. Finally, we support emotion because it leads to anger, which we hope will produce positive change in these realms, scientific and otherwise.

DR. MUIR: Because the topic for the continuing discussion on lead will carry on to topics not specific to lead, I would like to ask the speakers on the lead panel if they would sit up front. We will try to address questions related to lead and comments related to lead, initially, and then go into a more general discussion.

DR. RIEKE: I am a private practitioner in Portland, Oregon. I want to talk for just a moment about lead and sympathize with my young friends because I really have had a similar experience with them. It is very hard to get attention to lead intoxicification from the people in the lead industries. They have been prone to say that the situation was under control.

Our first speaker this afternoon seemed to me to be speaking of subclinical lead intoxicification as a peripheral concern. I think he is wrong, flat out. The workers with whom I have worked for 30 years are a bunch of marginal workers in shipscrapping--a temporary postwar business that is still booming and that will probably go on for another 20 years.

Most of these workers are relatively unskilled. They are very heavy consumers of alcohol, which has confused our diagnostic attempts. But I did learn that lead is a soporific sort of material. It dulls one's awareness. We found that workers in their histories were really quite inaccurate. They not only didn't want to tell me about their symptoms for fear someone would fire them--a very important problem for alcoholic, marginal workers--but they also didn't seem to have complaints because they were half asleep from carrying a burden of lead.

As a result, many of these workers encountered very grievous accidents and serious burns. They would cut off metal bulkheads that would fall on them. In fact, I found myself taking care of these people as patients with hospitalization and very serious problems.

So that the subclinical aspect of lead intoxicification is a

very real entity even though they did not have complaints. They did not have wrist drop, or cramps, or other things, and they were very difficult from a clinical standpoint to identify.

But as we were able to delead them, we got a much more accurate history. But it is certainly true that from a clinical standpoint, physicians, employers, and the academic community have a much eschewed picture of what lead intoxicification is. In my judgment, it is a much more widespread problem than most people are aware of.

DR. BRIDBORD: The one point I wanted to make to Dr. Rieke is that I did note the subclinical effects. I noted they were a point of contention, but I also expressed my personal opinion which was, in fact, that they were very important. So I agree with many of the comments that he just made.

DR. RIEKE: I think Dr. Corn knows, or if he doesn't I will send him copies of the letters, that I, too, protested at the 80 microgram level. I really have seen very sick workers with 56 or 60 micrograms and this individual variation is really very wide. So that publication of a proposed standard at 80 micrograms, I felt was really far too high. By the same token, I have seen people with well over 100 micrograms who couldn't think of a single symptom, and healed up very well after major burns, or after a fractured leg, and so on. So that the linking of symptomatology with blood levels is not particularly well correlated.

DR. FRANK LUNDIN: I am an epidemiologist with the Bureau of Radiological Health, of the Food and Drug Administration. I am a chronic disease epidemiologist. I would like to comment on one speaker's statement that he was amazed that after so many years there weren't dose-response data for lead. I would also like to make a general comment about my impressions about the meeting. There doesn't seem to be much realization of the difficulty of doing some of the long-term studies that are needed to get the information for future actions.

I would like to say a few more words before we go on to debating what actions could be made based on what are perhaps not sufficient scientific data. Like it or not, what has happened in the occupational environment are what we might call natural experiments, but these really aren't experiments until someone comes along and makes observations. The process of making these observations requires a high degree of technological, administrative, and economic organization. It requires a lot of money. It requires enlightened administrative support for studies. It requires a good, supportive, competent technical staff. It also requires a legal basis for access to necessary employment, medical, and administrative records, and for protection of research data from subpoena.

Some of the recent privacy act legislation is actually threatening the very existence of the kind of studies that are needed for future action.

DR. LERNER: We have heard several comments here today about the effects of lead on the reproductive capability of males. This was probably all directed to the paper by Iona Lancrejan from Bucharest. If anyone will take the time to look at that paper, here are a few comments of mine that you can either confirm or deny for yourself. One is that the author did not actually measure in any way, shape or form actual reproductive capability of males, other than by extrapolation. The measurements were made only on spermatogenesis and the assessment of asthenospermia, teratospermia, and hypospermia.

No definitions were given for these alterations of sperm. I am not sure exactly what all of them mean. They talked about controls--about 50 controls--and I have just told you as much about the controls as they have told me in their paper. In other words, we don't know where the controls came from, or how they were studied. They were not intermixed with the test subjects.

Furthermore, of 50 workers in the lowest exposure group, they excluded 50% of them from their analyses, saying that these workers were excluded because they had other conditions which were known to have adverse effects on spermatogenesis. That seems like an awfully high background incidence of other effects known to cause adverse effects on spermatogenesis.

Then there was another concern that I had. They made a definite statement in their methodology that in order to collect a good sperm count, they obtained the cooperation of the workers in having abstinence from any form of activity which would release sperm. It would take a lot of cooperation for me to get a group of people that would agree to do that for three days.

And then they went on to say something to the effect that it was impossible to get accurate data on the reproductive capabilities because of poor cooperation between the workers and the experimenters. So it is hard for me to understand why it would be so difficult to find out from a worker such things as how many times his wife has been pregnant or how many children he has sired, when it is apparently easy to get this worker to abstain from ejaculating semen for three days, and then to produce some either by coitus interruptus or by masturbation.

So all that I would say is that it is an interesting piece of literature. It is one piece of literature. I think that we need far more data and scrutiny of this and other literature before we jump the gun, so to speak, and extrapolate that we have good data on either alterations in spermatogenesis or alterations in reproductive capability of human males.

DR. HECTOR BLEJER: I think Dr. Lerner is quite right in saying that there is scant evidence and data on many of these aspects. But I would like to ask him and also anyone from the Lead Industries Association what studies the industries are sponsoring to clarify these areas that lack data.

I believe you have been handling lead as an industry in this country for upwards of a century, and I would be happy to know what funding there has been for studies in, say, the last year, concerning reproductive effects of lead in males or females. I would also like to know if Dr. Lerner is involved with any such studies himself.

DR. LERNER: I am not personally at this time involved in any such studies. I happen to know that the Lead Industries Association has looked into what it would take to do an epidemiological study of reproductive capabilities and fertility. We have some feedback and it is still being considered as to what study might be done. There is a real problem in the design of a study in which the frequency of events is low--if, for instance, you might expect an event to occur one out of 1,000 times.

I would expect that if there is a problem with lead at today's levels, we are probably dealing with a low, low frequency event. To detect a doubling of an event that only occurs one in 1,000 times would take 23,000 observations.

I would like to ask NIOSH what they are doing to study this problem.

DR. BRIDBORD: First of all, to backtrack a little bit, NIOSH has been doing studies with respect to effects of lead upon workers for a number of years. I might add that we have just about completed a very extensive field investigation of workers at the primary smelter at Bunker Hill in the State of Idaho.

NIOSH also is desiring to increase its efforts with respect to studies of lead. We have identified three main areas for important emphasis. These include the effects of lead upon the reproductive process, the effects of lead upon the nervous system, and effects of lead upon the kidneys.

Finally, in the general area of effects of lead upon the reproductive process, NIOSH has submitted a proposal for an initiative which would specifically look at this whole situation, including but not limited to lead, and we are very optimistic that we will begin to do a number of long-needed studies in this regard.

I don't profess we will have all the answers tomorrow, but at least we are trying to do our best.

DR. WILLIAM CHEN: I am Chief Occupational Medical Officer for the District of Columbia.

I want to address a more practical problem which Dr. Corn explained is practically impossible, the so-called zero-level standard.

So I would rather like to see us request that OSHA and NIOSH do more research in the area, not only in the females, but also in the males. I would request and suggest that NIOSH should pursue more the study of reproductive hazards of both sexes; then OSHA should try to do the most to reach the theoretical zero level.

MS. ODESSA KOMER: I am Vice President for the UAW, Director of the Women's Department. A curious chain of events brought me to this meeting. For example, some time ago I read an article, a special reprint of the Department of Labor, in which the question was posed: Are women workers special? And there was a paragraph in there by Dr. Jeanne Stellman, which said, "Look for discrimination against women workers in this route. They will try to eliminate women of childbearing age from certain jobs."

Believe me, I felt that she was an alarmist. I felt that with Title VII, and with all these beautiful laws that are opening up employment opportunities for women, how can I believe this woman?

Then, in December of 1975, General Motors x-ed women of childbearing age out of the battery part of the shop. They moved them right out unless they could prove to the satisfaction of the company that they could not have children. And so I found out that Dr. Stellman was not an alarmist. And I feel that the move by General Motors in Canada is just the beginning, the tip of the iceberg. You know, every time I pick up a newspaper, I read about a study of what is injurious to a fetus. Caffeine is injurious to a fetus. Noise is injurious to a fetus. Every woman of childbearing age's job is in jeopardy.

You have made all these gains and they are going to wipe it out. I can see it coming. You sit here and listen to what is happening. You listen to more studies and more data. They tell you we need this, this, and this. And you are going to be out of a job. But, you know, even people that can get pregnant have to eat.

And the same people who rant and rave about women being on welfare or Aid to Dependent Children would x you out of a job that fast. I think we have to say over and over again that it cannot happen. It is up to OSHA and NIOSH--and they have done a credible job in a lot of areas--to insist that industry make it safe enough for a fetus. Then it will be safe enough for men and women.

DR. WAGONER: I would like to make a general comment and also, in a sense, throw down the gauntlet.

The question that has been raised over and over during the past several days concerns itself with the effects of industrial exposures, either on the male or the female, and some of the effects on the next generations.

The question was raised as to what studies the government was doing. I should now like to ask the question: What corporations in the United States are undertaking any study of the reproductive effects either of the female employee or the wife at home of the male employee? I know of no such studies being undertaken.

Second of all, we have decried the absence of data at the lower levels of lead. And this reminds me of a story and an experience I went through with uranium miners, and that was how can we study the effects of low level lead or low concentrations of radon daughters when the plants are operating in excess of standard.

DR. INFANTE: I want to make a comment about Dr. Lerner's criticism of the study that was done on sperm. He comments that, "Well, they looked at the effects on sperm. Why didn't they then look at the effects on reproduction?"

Well, this study was conducted on 150 male workers. So he is saying, "Why didn't they look at the effects on reproduction in 150 male workers?" But when he was asked to respond about the study that industry would do, he said, "It would take 23,000 pregnancies to be able to find an excess."

So, if it would take 23,000 pregnancies in order to be able to determine an excess, then how could you expect to find a significant event even if there were, indeed, a twenty-fold excess from a population of 150.

DR. KERNER: I am from the University of Illinois. I would like, first of all, to commend Dr. Lerner on the proceedings of a conference held by the SOEH in Chicago on occupational diseases from lead and arsenic, which has just been published by NIOSH. I would commend this to all of you.

Secondly, at Cook County Hospital in Chicago, a very large hospital that deals only with working people, we will be happy to show him lots of workers who have lead poisoning. You don't have to pick a special day. They barrel in almost every day.

And, thirdly, one of the problems that we have in doing studies is not methodology. I think that the data on workers is more guarded than the mint. If we had the data on workers, I think we could develop the methodology and do very fine studies. But those data are not available. I don't think they are available to NIOSH and they are certainly not available to anybody else.

DR. CORN: I would like to take the discussion to a second stage of examination. We are facing some very large decisions with respect to implementation of standards.

Let us hypothesize for lead that the costs become enormous and that it is argued that we can achieve protection with a whole new generation of respirators at a very reduced cost, and that we can, indeed, protect everybody involved.

What will be our posture? OSHA has consistently assumed a posture that we wish to change the physical aspects of the work environment, and that personal protective equipment is not a way to do this.

However, this argument has not surfaced to the level that I believe it should. Now, yesterday, to illustrate how enormous this kind of contrast can be, we released our inflationary impact statement for noise. Well, for the two scenarios painted by the contractor, an 85 decibel and a 90 decibel, the capital investment costs were \$18 billion and \$10.5 billion respectively. I understand that on the radio and the television today it was said that \$41 million or so would take care of this, if we just issued ear protectors.

So, the second stage of discussion of these factors in the work environment is how will you do it. If we reach the conclusion that we will, indeed, protect everyone, then you get on to how you want to do it.

And I think we should spend a little time talking about that because certainly in the noise area we see it in its stark reality.

MR. MAZZOCCHI: I am from the Oil Workers Union. I would like to start challenging some assumptions because I think we always get trapped into the assumption that somehow we have to mold the worker to conform to what is, to conform to the industrial workplace as we understand it to be. It is a losing argument as far as workers are concerned. It is a no-win situation.

The question that more information is needed really doesn't mean anything to people who are at the point of production. With increased awareness and increased consciousness, is the position of a worker better today than it was 10 years ago when we ran our first series of conferences on occupational health? No, it is far worse. Ten years ago the workers in our union throughout the U.S. and Canada described their work environment. They said, "This is what takes place in our work environment." They weren't trained epidemiologists, but they knew just by casual observation--not knowing anything about statistics, mind you--that an abnormal number of people died of cancer of a particular site or had various infirmities. And if you look at transcripts of those conferences, you get a pretty accurate description of what work is

truly like in America and what happens to people who enter the workplace.

Now, why is the situation worse? Simply because--and this is what we have not discussed here--occupational health is essentially a political and economic subject, and unless you introduce the politics and economics of occupational health, there is no way in which you are going to address the concerns of all workers.

I have heard two honest statements out of the Federal Government recently. One was by William Simon of the Treasury. He said very clearly, "Listen, we are in a period of capital transference. Those who don't have, have to give to those who do have." That is a very astute observation because that is precisely what we are living through and passing through. It is a massive capital transference program.

How does that take place? Well, you have to increase productivity. How does productivity increase in a workplace? well, first of all, you lay off a large number of workers, first in the maintenance areas, and then in the operating areas.

We had refineries some years ago that would produce 150,000 barrels a day with 3,000 men. That same refinery produces 500,000 barrels a day with 900 men. Granted, there has been some improved technology, but essentially it is because the plant is kept on-stream until it breaks or blows up, which it does with increasing frequency.

You go through a modern oil refinery and it is leaking all over the place, both into the water and into the air. And what happens as a result of that reduced operating and maintenance capability? Somebody is paying. The community is paying. It is no secret that the plants where we represent workers are in cancer belts around the country. Look in New Jersey at the petrochemical industry and the aniline dye industry. Look in Jefferson County in Texas, where we have discovered the cancer of the week, leukemia, the styrene-butadiene industry.

Now, the second document that I would like to address myself to is the inflationary impact statement on coke ovens. If you discuss nothing else at this conference, that document ought to be discussed because I think the whole tale of occupational health and safety is contained in it. The document says that there are 15,000 coke oven workers, and that in order to make the workplace safe you have to add 5,000 more workers. Now, that is not a very profound discovery as far as we are concerned because that is what we have been talking about all the time. You need more workers in order to correct the problem.

There are irreconcilable conflicts in occupational health. One is you can't have increased productivity and have a safe

workplace. That is the type of mythology that has to be destroyed. It is not a question of a little more hardware here and there. Industry would make the capital investment for the additional hardware. It is the productivity factor.

And that is why, yesterday, I posed the fundamental problem: If industry is serious about occupational health, it will provide us with the information that we need.

Well, industry is not willing to do that simply because with that information we are not going to depend so much upon the law. We recognize the limitation of law. We understand the game is stacked against us when we see that OSHA, for all its good intentions, is operating with about \$150 million. Now, you can't address a question of the magnitude of the occupational health dilemma in America with that type of funding. So the deck is stacked against the worker to start with.

However, if we receive information, we will go into a conflict situation, and out of the resolution of conflict there will be some improvement for workers in the workplace.

Now, I think I have some skills in political economy but I would be considered an illiterate when it comes to many of the science questions discussed yesterday and today. I am sure you wouldn't call upon me to perform surgery, but I am always amazed at scientists who get up and make profound statements about collective bargaining and the economics of the workplace. I think it cuts both ways. And yet most of the statements I heard in relation to most of the papers had within them the basic economic assumption that things can't be done unless you accept a certain amount of assault in the workplace.

If I became a plant manager tomorrow morning, I would be talking in a completely different tone because the interests I would have to represent would be those of that corporate entity. If I didn't represent that interest, I wouldn't remain there very long. I have never known a corporate executive to say to anybody in the lower echelon that he could go out and be freewheeling. His job is strictly to maximize profits; otherwise, he is not in that particular industrial setup. So I am saying that in order to really discuss occupational environmental health, we should really be talking about what happens to the community, what happens to the worker, how the community is impacted, and who pays.

Right now industry is a big industrial welfare basket case. They are on a welfare dole. Workers give years of their lives to keep the industry going. So the argument is over essentially should workers withhold this subsidy to industry. This is what inflationary impact statements are all about. The billions of dollars that Dr. Corn talks about is precisely where it is. Someone pays. That cost-benefit ratio--it costs someone and it bene-

fits someone. It costs workers to benefit profits for management and stockholders.

That is an essential ingredient of the occupational health dilemma and I agree with Dr. Corn that we have to talk about it.

We ought to talk about the lead problem. Why is there a problem? I know there is a problem for men and women and the variables don't interest me very much. I don't care whether it hurts someone a little bit more than it hurts someone else.

The mere fact that it is affecting workers and affecting us in many varied ways is what we ought to talk about. What are the economics that dictate that this should take place? Why, after all these years, is the situation worse today than it was yesterday?

I think unless we discuss some of these questions, we are on a treadmill in any attempt to speak to the occupational health dilemma. I think we have to change the ground rules. The ground rules are too constrained for discussing occupational health questions. Unless we tie in political economy to our questions, those of us who are responsible for representing the people we do represent are going to lose this fight. We are not going to represent them as far as improving their welfare. All we are going to do is continue to listen to papers about how bad things are and attempt to deal with this in a very fragmented way.

I happen to believe that industry, as it is now composed, can't make the workplace safe. When I read a Mobil ad in the New York Times about the necessity for capital formation, I accept it as a basic fact. I also know that the capital is going to come from our hides, and that I can attempt to alleviate the situation by moving into conflict with that particular oil company because, as I pointed out, their move to increase productivity has to make that workplace more unsafe.

And I think unless we introduce that dimension of the problem into these discussions, it is a losing proposition whether the subject is lead or any other. The economics have to be discussed; not how much it is going to cost, but who pays. That is what has to be discussed.

For instance, let's talk about why there aren't tumor registries in this country. Why aren't third party payers required to put occupation and job classification on a claim form? That would do more good than 1,000 conferences of this sort. You just put those two items on a third-party-payer form and we will be able to secure information about what happens to workers. I know there are 10 million lives insured in the State of New York alone. If those people went into the doctor's office, and if their illness, job, and job classification were put on the claim form, we would know a

lot more at the end of the year than all the studies you could possibly institute.

But that is by design, not by accident, and the fact there aren't tumor registries is by design and not by accident. The system is designed to deny us the type of information that would agitate us to be propelled into action. And that is what we need. We need to be agitated more than we are now. So I think there ought to be a statement insisting that third party payers code their claim forms and that tumor registries be instituted. The third party payer would change things dramatically and there should be no conflict on that question. I don't see how anyone could legitimately object to providing us with that information.

MS. NELSON: I want to continue for a minute on the line of discussion that Dr. Corn mentioned, and that has to do with standard-setting, whether you control the environment of the workplace or put the burden on the worker to wear a respirator.

The Act clearly places, under Section 5(a), the duty on the employer to furnish a safe and healthful workplace. It is an expense that must be borne by the employer. In the case of lead, lead is something we are exposed to every day of our lives. We cannot have workers working in establishments with unacceptably high levels of airborne lead, given all their other lead exposure in the environment, and then put it on their backs to wear that respirator constantly, hoping that it is a properly fitted respirator.

So, as we get into the lead standard, these are the kinds of things that are going to come out, and these are the kinds of testimony or economic blackmail that the Labor Department is hearing right now.

Secondary lead production will be replaced by imports. This is if we go in and make them clean up. Secondary production would be replaced by new secondary production. In other words, what they are saying to the workers is, "We will close down this smelter because we can't clean it up and you lose your job while we try and build another one."

Lost secondary production would be replaced by primary production and all sorts of implications that the industries will go overseas.

So I think that this is one of the issues we are going to have to face. We cannot just let the standard rely on the biological monitoring of the individual worker. We have to clean up the shops.

They have known what this standard was for five years. Yet we are still talking about excessive lead exposure far and above

the 200 microgram level.

MR. DON LYNAM: I am Manager for Environmental Health for International Lead, Zinc Research Organization, and I wanted to respond to Dr. Blejer's question, which was perhaps directed at me.

As the research arm of the lead, zinc industry, we are sponsored by the primary lead and zinc mining and smelting industry throughout the world, and that industry has been sponsoring much research in the environmental health area. At any one time, we have approximately 20 to 30 projects ongoing in the environmental health research area.

We had a meeting in the spring with Dr. Finklea, Dr. Bridbord, Mr. Becker of the Steelworkers, and Mr. Samuels of AFL-CIO, to discuss areas of research in the occupational health area. Government is doing studies, industry is sponsoring studies, and many times the studies are of the same population groups. Sometimes there are arguments about the protocols of the studies, depending on who they are sponsored by. At a meeting on lead and arsenic in Chicago, which was sponsored by SOEH, a committee was formed with Dr. Muir as Chairman, to evaluate a study that we had sponsored, and a study that CDC had done on the population living around the smelter in El Paso. Perhaps Dr. Muir could let us know what the status of that report is.

At the Spring meeting, we gave NIOSH a list of all the environmental health projects we are sponsoring. Earlier, we gave NIOSH a report done by the contractor on the design of an epidemiological study of the effects of lead on the reproductive processes in women.

We have been wrestling with this question for some time. We have been trying to evaluate what size population is needed in order to be able to draw valid conclusions. You cannot go into a plant that has 10 or 15 women, perform an epidemiological survey, and feel that the results are valid because they certainly do not constitute a sufficient population. We need to know what size population is needed, and whether such a population of women, who have been exposed for a sufficiently long time, would be a sufficient population to evaluate.

Here are some of the figures on follow-up required to detect various reproductive manifestations with 5,000 women in a study group and 5,000 women in a control group. If there is a decrease of one-half in the birth rate, you will need one year to follow up a study population of this size. For a double prematurity rate, you would need two years, two years with 5,000 in each study group.

As a result, we have a proposal to carry out an epidemiological study of this type, and this was given to NIOSH when we had a

meeting in April with Dr. Finklea on Bunker Hill. So I think the industry has been sponsoring quite substantial research, for at least the last 10 to 15 years, since the existence of the International Lead, Zinc Research Organization.

Research in the environmental health area is expensive and takes a significantly long time, and I think the industry has approached this on a cooperative basis. In fact, this type of research is something that lends itself to a cooperative basis, not only within the industry, but also between industry and government.

I think everyone should be able to agree on the best protocol to try and answer a specific question regardless of what your bias is or who you represent.

DR. MUJR: Let me comment on the El Paso study. There was an ad hoc committee formed as a result of the meeting in Chicago, which did look into the three El Paso reports. That ad hoc committee, which I had the honor of chairing, has submitted its report to the Society. The report has been accepted, and the Society is seeking avenues for publication of it. I would anticipate publication shortly. If somebody has a crucial need for a copy of the report prior to publication, I understand that perhaps a Xerox can be made available, but at a rather large cost.

At this point, let me close the session with respect to specific comments on lead. Dr. Bingham will take over chairing the meeting now for purposes of more general discussion.

DR. BINGHAM: I would not want to close the meeting to any comment on lead, but I would invite other comments and discussions, things that you have on your mind. If you have suggestions for meetings or approaches to occupational health problems in the future, please feel free now to begin with those.

MS. WHATMAN: My name is Francine Whatman. I am with Yale Medical School and the work I do involves the use of the Connecticut Tumor Registry data base.

I would like to tell those people who are here that there are tumor registries in the United States. I cannot tell you all the states in which they exist, but I know offhand that California has one. It may be regional within California, not the entire state.

Connecticut's data base covers the entire population of Connecticut. Upstate New York has one. And as Director of Field Research there, I can tell you that we would be most eager for the cooperation of the unions because we are very interested in occupational studies.

One of our prime difficulties will be getting occupational history data on workers, whether we are looking at people who have been diagnosed with cancer and have subsequently died, or looking

at current workers. It is very difficult to get accurate occupational histories. If the unions have this data available on their membership, we would be most appreciative of any cooperation. I am sure other tumor registries would feel likewise.

DR. WAGONER: I should like to follow up on this question of the tumor registries. Last year I was fortunate enough to attend a meeting that took place in Lyon, France, under the auspices of the World Health Organization, where the subject of tumor registries was discussed. There is no tumor registry in the world that takes an occupational history of the individual at the diagnosis of cancer. Yet we talk about getting more specific histology of the tumor.

MS. MAUREEN O'BERG: I am with the duPont Company. And I would like to tell you that duPont Company does have a tumor registry, which was established in 1956, and is maintained up to the present date. With this tumor registry, we record the age at diagnosis and also occupational information is available. This is for duPont employees only.

I would also like to answer Dr. Wagoner in terms of studies of birth defects in employees. DuPont has not made any such studies. I feel that it probably will not make any such studies because we have enough trouble getting information on smoking histories, alcohol habits, and personal habits of the employees themselves. There is a feeling that the company invades the privacy of employees when we ask such questions. I feel there would be real reluctance on the part of employees to provide information about whether or not their wives have had children, whether they have been unable to have children, and the results of children who have been born.

I think that this may be one area where industry cannot contribute. I think that perhaps universities will have to make this contribution.

I would also like to indicate that we are making many epidemiologic studies on materials which our workers handle in the workplace. This involves both men and women.

DR. BINGHAM: Is this tumor registry generally available to the government or academic communities so they could use it in their studies?

MS. O'BERG: It is not generally available although mortality and morbidity statistics, which would include cancer incidence rates and mortality rates, were offered to APHA. I presume the information would be available although it hasn't been published per se.

DR. JEANNE STELLMAN: I think it is a mistake to argue about whether one tumor registry is better than another, or how extensive

they are. The fact is that for all intents and purposes preventive medicine and public health are virtually nonexistent in our society or in the world. And occupational health is part of that situation.

The fact is that if one looks at the National Center for Health Statistics and their publications, if you look at the basic minimal data set required for hospital admissions and hospital records, you will find that under "occupation" it very clearly states that it is too much trouble for the admitting office to collect occupation data. At the present time our department is negotiating with the National Center for Health Statistics because they are interested in changing this policy.

But under the current system of private medical care, as diverse and distributed as it is, and with not a single medical school in the country really teaching anybody about occupational medicine, one can't hope to have any real changes.

But that is not what I really wanted to say. What I find really amazing is to hear someone like Tony Mazzocchi get up and talk about political economy, or hear women get up and talk about a basic problem with this conference, such as the lack of communication, and then just go back to what we had been talking about before.

The cost benefit analysis of industry is what we should discuss. Who is paying the costs, and who is getting the benefits?

It is during the last 125 years that the workplace has been removed from the home, and that women have been faced with the conflict of home versus child versus husband versus housecare. Women have always had the double duty of providing an economic contribution to society, and of providing children and domestic care. But it has only been in the past 150 to 175 years that double duty has had to take place in two different places. And with the creation of the nuclear family, that double duty not only is in two places, but falls solely and completely on the mother of the family.

I think the basic question is that someone has to bear the children. This is not going to be the last generation on earth. There will continue to be families. And there is a very real question in my mind as to whether we can continue to have men and women working 40, 50, 60 hours a week on the job, and maintaining separate nuclear families at home. We have to start reevaluating things like part-time work, shared work, flexible shift, job retraining, and economic support for the social function of bearing children. We need a total recasting of what our priorities and aims are.

Certainly it is going to cost 18 billion or 20 billion, or

whatever it is, for noise on the job and noise retooling. We are talking about 20 billion as a figure of capital investment because there is no price on losing your 6,000 frequency range, or your 4,000 frequency range, or not being able to hear the television unless it is really turned way up, or on not being able to communicate unless you look at someone's lips.

There is no price on women's stress, the cost of women's social well-being, mental and emotional well-being. You can only add up the capital on one side. I think we are really going to have to start talking about whether we can equate capital costs and capital benefits against human costs and human benefits. We have to keep in mind the perspective that we are only talking about a very recent phenomenon in human history. And we also have to keep in mind that the well-being of women in every society in the history of the world has been the mark of the well-being of society as a whole. In short, unless we solve women's problems I make the point that we are not going to solve anybody's problems.

MR. HARRY SLATON: One thing we have been talking about is how to get some data. Part of the OSHA law was to review the Workmen's Compensation laws in the states. The Workmen's Compensation laws were set up essentially for limited liability. One of the offshoots of the Workmen's Compensation law is the state death certificate. Now the state death certificate usually states the last employment of the deceased, even though an individual could have worked for 30 or 40 years in a hazardous area, and had emphysema, and had to have left that job and taken a job as a church janitor. So he dies with emphysema as a church janitor. But because of limited liability, these death certificates are not changed. If you change them to show the true facts--that this individual acquired his emphysema or byssinosis or pneumoconiosis through some work experience he had--then this employer would become liable upon his passing away. And this, I feel, should be remedied so that we can get accurate data on what causes people to die.

MS. CLARA SCHIFFER: I would like to speak as Co-Chairperson of this conference and reply to some of the issues that were raised by the women who presented their statements about the conference.

I think that some of the criticism was warranted. I think that the Chairpersons failed before each session to pull it together, to tell why, for instance, we were talking about DES. Several people came over to me and said, "Why are we talking about DES? DES isn't an occupational hazard."

We were talking about processes that we have to understand if we are going to understand occupational hazards. But this was not explained. And I think the criticism that there was not sufficient explanation was absolutely warranted.

I think the criticism that there was too much technical language without an explanation was also warranted. I think that we cannot have science watered down to a third grade level. On the other hand, if a scientist can't explain his work in language that all of us can understand, then we are not in a position to either understand it or apply it. So I think that that criticism was warranted.

I think you have also put your finger on one of the difficulties with this particular Society and with organizations in general. How do we combine scientific conferences and scientific explanations with what to do about the problems and with general explanations.

That is a very difficult thing to resolve, but I think we came a long way in this conference. Believe me, if you had been at some of the previous SOEH conferences, you would appreciate how far we went in this one toward trying to solve the problem of combining scientific knowledge with experience, of working out what to do about it, and of understanding the implications.

Yesterday, the Council voted to set up a committee to go over the results of the conference and to think about where the Society should go next, and also to advise the Council on what it should do in future conferences. I think this was a very sound decision that the Council made. And I would like to ask that you write your recommendations down and present them either to me, to Eula Bingham, or to Joe Wagoner so that when the committee meets we can seriously consider them.

I would also like to say that we have welcomed all of you. This is the broadest representation we have ever had in the Society and I think it has just been wonderful. I would now like to ask all of you, who have a new interest in this subject, to join the Society. It is \$25 a year. You will get a great deal out of it, and we will welcome you young people coming in.

MS. MAGGIE WOLNER: I am from Medicine for the People in Montreal, Quebec. I think that one way to improve communication would be to have a conference where the workers and the people talk to the scientists. I am sure that the scientists would understand us, but I am not sure that they will listen because I think that one of the difficulties is that scientists always want to talk to someone, but they never want to hear what the workers have to say.

At the meeting last night with the CLUW women, there weren't a lot of scientists there to hear what working women had to say about the conditions in which they were working. And the women really didn't have much of a chance even to talk about what their real conditions were. They were sort of overridden by men who had other things to say.

So I think it would be very important to include in the day-time scheduling opportunities for working women, to talk about what problems they have in the workplace, and to have scientists listen to them, so that the scientists can then direct their research more immediately to the problems.

DR. BINGHAM: I think you are absolutely right about workers addressing scientists and other individuals in this whole area. As Chairman of a Coke Oven Committee to recommend a proposed standard, I can tell you that we have always had workers in the audience. They were not necessarily union leaders; they were workers. At every session, I took the prerogative of being Chairman, and asked the workers to speak and to contribute. And they certainly did contribute to our overall knowledge of what the workplace was like.

Frequently, there were people on the committee who were either representing unions or representing industry, who did not know what the job situation actually was. But we had workers who enlightened us and who straightened us out many times, and if you go back and look at the record, you will find that it is there. So I agree that it is very important for us to listen to workers.

MR. FRANK WALLICK: I am with the United Auto Workers. The UAW members pay me to communicate, so I am very sensitive about this whole question of communication. I think it is a chronic problem of scientific meetings. In fact, one of the things that pleased me was to find out that the scientists don't understand each other. I have been muttering in the corridors about the fact that I can't understand all these slides. It is an affectation of scientific meetings to think that you are not being scientifically correct unless you have slides. Still, there are occasional scientists, who do manage to communicate, who can be scientifically accurate and understandable at the same time. And I think that this is a problem that this Society should take very seriously. I happen to think that you can be accurate and at the same time be understandable. So I applaud those people who raised the question and I think that the Society should continue to work in this area.

However, I think that those who are critics of the Society should understand that the Society, because of its interest in the problems of women at the workplace, has galvanized the Coalition of Labor Union Women to set up a task force in occupational health. Some of us who have been urging CLUW to do such a thing are delighted that there is this much interest. I think that the sensitivity of the women's movement may ignite the occupational health movement as it has never been ignited before.

This particular movement has been laboring long and hard to try to get public recognition of the problem, and I think that CLUW, because of its special interest and its sensitivity to human problems, may be able to get the occupational health movement a much wider audience and concern in the country than it has

had previously.

MS. LOIS KOOGLERMASS: I am an organizer for the United Electrical Workers. Our people have been concerned about the technical language and about the lack in many cases of worker and union participation. I also want to say that when you are talking about occupational health, you are talking about politics and economics.

Now, there is another matter that should be brought up because it is important to consider the implications of all of the kinds of studies that are done. I call attention to the study presented this morning on physical stress. Since the protective laws were taken off the books, the pressure is being brought in many workplaces to get women to do as much work and as much heavy lifting as possible. In terms of upgrading into jobs, the company will sometimes say, "We will tack on heavy physical labors," just to try to eliminate a woman from moving up in a work grade. In other words, they will say, "You have to lift 50 pounds twice a day before you can have this higher paying job."

Now the point about the study this morning that struck me is this: Having been a worker in such a plant, I know that the company's interest is to get me to lift as much as I possibly can no matter how exhausting that is.

So I want to ask: What will the use be for these kind of studies? If I say I do not want to lift a 40-pound box, seven times a day or 20 times a day, but these stress studies show that I am physically capable of doing so, what kind of protection is there for the worker on the job?

MS. WOLNER: I want to affirm what you just said because these issues are always raised. Why don't workers want to give duPont Company information on smoking and alcohol and what late hours they keep during the weekends? Why don't workers want to have tests of how much they can lift? Why don't workers want to have their hearing tested? Why don't asbestos workers want to have tests of lung function since supposedly it is for their own protection? And, of course, it is always the question of where the information goes and to what uses it is put. This is an issue that we haven't addressed in this conference at all. Scientists are always calling for more data. But to what purpose? Who is going to use it?

DR. RIEKE: I don't share all of Tony Mazzocchi's feelings that everything has gotten worse. I have been waiting for this meeting for 35 years and I am just delighted. I am very distressed that we are not moving ahead faster, but I must say that we have never seen so much attention to cleaning up, to trying to find out what the hazards are, and to doing something about them. I don't know what is going on east of the Mississippi, but in Oregon things are moving, and I must say they are better. Now they are not good.

As a matter of fact, they have got a hell of a ways to go. But at least we have got wonderful people like yourselves in the act, and I hope you will stay there.

I am concerned. The woman in the workplace has been a concern to us. In the shipyards during the war, we had 35% of our women doing work that the men were doing. Actually, they didn't do quite the same thing as all the men. But, on the other hand, the men were either 4-F or too old, or too young, and the healthy guys were all off in the service. So we managed to get on with the war somehow under those circumstances.

Now I saw a lot of regrettable deaths and a good deal of injury. This convinced me that we really did need to have physicians try to learn and talk with union people. But one of the things that union people haven't been totally honest about, I think, is that you want to know what the hell is the matter with medical people, but you don't trust us, and you don't want to pay us. Yet we are trying--at least I am--to work with working people. That is my career. So I must say that if we can get talking here, we are making progress.

As for the OSHA problem, we have got laws that are five years old. As you well know, industry is still trying to get them declared unconstitutional. I hope that all of you will stay in there punching. I hope the Congress won't buckle to the pressures of people who are trying to say that these laws, which are designed to improve situations for working people, should be thrown out.

Now I am well aware of another statistic which I think is criminal. When we looked at the situation two or three years ago, women were paid on the average 60% of what men were paid for the same job. This is the economics that Tony Mazzocchi was talking about. The situation is still way out of focus. As for Dr. Corn's concern about a few billion dollars, when you are talking about billions, you are talking about that difference between 60% and 100%. We are talking large sums of money, but we are in a trillion-dollar economy, and I have the strong feeling and conviction, after many years of working in the field, that we are not going to go broke fixing the situation up and making it healthier.

We know that cleaner, healthier working circumstances are much more efficient. The cost benefit is all on the side of cleaning up. And even though I consider Tony Mazzocchi a real demagogue, which I am not, the fact of the matter is that we are fighting the same war. We are just fighting it in different ways.

The situation is not good, but it has gotten better. There is a hell of a lot of room for improvement, so stay in there and punch.

SESSION VI

A SAFE WORKPLACE: CURRENT PERSPECTIVES AND FUTURE NEEDS

MODERATOR: Jane A. Lee, R.N.
National Institute for Occupational Safety and Health

Good morning to you and thank you for staying with us through this Saturday morning presentation. My name is Jane Lee. I am the Occupational Health Nurse Consultant with NIOSH in Cincinnati. When I began to study industrial nursing, I learned that Jane Addams and Dr. Alice Hamilton were very close friends, and I found it quite inspiring last evening at the banquet for Dr. Harriet Hardy to realize that Jane Addams, the social worker, had worked with Alice Hamilton, the female physician, who was a preceptor to Harriet Hardy. Let us hope that with so many women in our audience their very fine inspiration and challenge will continue.

On Thursday we learned a lot about the scientific data collected through research mechanisms, and on Friday we were questioned about the results and the implementation of some of them. Today we hope that our speakers will assess the present trends and issues, and attempt to predict some of the future needs for a safe and healthful workplace. I should like to introduce our first speaker who is Julia Makarushka of the University of Syracuse in New York.

WORKERS' COMPENSATION: THE LONG-TERM CONSEQUENCES OF WORK-RELATED INJURY FOR WOMEN

Dr. Julia Loughlin Makarushka
Sociology Department, Health Studies Program
Maxwell School, Syracuse University

Although the identification of sources of industrial accidents and illness is the focus of this meeting, it is clear that all work-related injury cannot be prevented, even with the most stringent safety standards and constant monitoring. Nor can we forget the needs of those who were injured in the past, and who continue to be impaired and even disabled as a result. The Workers' Compensation system was established to meet the needs of these workers. Although there are somewhat different workers' compensation programs in each state, and two federal programs, each was developed to meet three goals: compensation to the worker for wages lost as a result of the injury; reduction of the personal and social costs of industrial injury by encouraging the rapid return of the injured worker to the labor force; and the promotion of safe practices by internalizing the costs of compensation insurance to industry.

Several studies have been conducted in the past to explore the extent to which the various programs meet the first two of these goals: adequate and equitable replacement of lost wages, and the efficient return of the worker to the labor force (Adams, Berkowitz 1960, 1973, Cheit, Jaffe, Morgan). None of these studies, however, has included a comprehensive analysis of the long-term effects of industrial accident and illness for women. Are they injured in the same way and to the same extent? Is disability more likely to result for women than men? Are women as likely (or unlikely) to be provided with rehabilitation services as are men?

Data from other studies suggest that the long-term consequences of such injury may be more severe for women. Jaffe, for example, found that the more marginal, less highly paid workers were more likely to suffer an uncompensated wage loss and

This research has been supported by a grant (No. 004-P-20-2-74-02) from the Department of Health, Education and Welfare and the Interdepartmental Task Force on Workers' Compensation. Additional faculty support was provided by the Herman Goldman Foundation. The research was conducted by the staff of the Health Studies Program.

**Following Saad Z. Nagi's definition (1969), impairment is the physical limitation resulting from injury. The term disability refers to limitation in labor force participation as a result of impairment.*

were less likely to be provided with rehabilitation. Women tend to be more marginal workers, with less consistent work histories, lower pay and lower ranking positions (Epstein, Kreps). Furthermore, women are, in general, less likely to receive rehabilitation services than are men, and with comparable levels of physical impairment, are less likely to be employed (Safilios-Rothschild).

The characteristics of the Workers' Compensation programs themselves suggest that the long-term consequences of industrial injury for women may be more severe. Compensation benefits are usually set at a per cent of the weekly wage at the time of injury. Although there is a minimum compensation level, and a maximum, compensation benefits are generally set at a specified level less than full replacement in order to encourage the worker's return to employment. This partial replacement, which varies among jurisdictions, is not based on any data about the relative disincentive effects of higher benefits, nor is it related to knowledge about the levels below which limited household resources hinder the full and speedy recovery of the injured worker (Berkowitz, 1973). In most discussions of injured workers it is assumed that the worker is either single or the head of a household in which his wife can substitute for him in the labor market if he is disabled, thus replacing his lost wages. While the adequacy of this assumption has been called into question for men (Johnson), only a very few households in which a woman is injured would include an adult able to replace the lost wages. An unknown but substantial number of working women who are injured already have disabled spouses, are widows, or heads of households with dependents. In some states, e.g. California, compensation benefits are based, in part, on an estimate of future earning power. Thus, the inequality of the labor market for women may be permanently built into any continuing compensation they may receive for their expected wage loss.

As women continue to increase their participation in the labor market, and enter a wider range of occupations, more of them will be affected by the operation of the Workers' Compensation programs. Two years ago we began a study of seriously injured workers to determine what the long-term consequences of their injuries had been in terms of income replacement, the adequacy of benefit levels, the distribution of rehabilitation resources, and their employment histories. A survey of workers in New York, Florida, Wisconsin and Washington was begun a year ago and has just been completed. This survey was conducted by the Health Studies Program of Syracuse University for the National Task Force on Workers' Compensation. The sample included workers injured in 1970, whose injuries had been serious enough to result in a permanent impairment of total body function (TBI) of at least ten percent. In this paper we will report some of the preliminary findings for the 231 women, 16% of the total sample. The fact that seriously injured women represent a smaller share of injured workers than their proportion in the labor force may result from

three factors, which we will explore in a later paper: women may be less likely to suffer certain types of injury because of their distribution in the labor force; they may be more likely to be employed in occupations (such as domestic service) or in smaller establishments which are not covered by compensation; many of the injuries which women suffer are not recognized as such by Workers' Compensation jurisdictions, since most industrial illnesses remain uncovered in most jurisdictions.*

Since so little was known about injured women workers, a preliminary analysis was done of the population of New York State workers injured seriously in 1970 to determine whether special samples of women needed to be selected for this survey. This comparison of 3058 workers showed that 18% were women,** and that the women were somewhat older and less well paid than men. Injured women were, as expected, in different industries and occupations than men; they were more heavily concentrated in service and clerical areas. In terms of the extent of total bodily impairment, however, the distribution of the severity of their injuries was the same, as was the distribution of the numbers of weeks of work lost because of the injury.

A review of the survey results for the four states reveals a similar picture. The 231 women are 15% of the total sample. They are older than the men; although they have similar educational achievement, they were working for a lower weekly salary before their injury and this year they were as seriously injured and lost as much time from work when injured. After the injury, 17% of the women and 11% of the men never returned to work.

When we look at present labor force participation, however, as Table 1 indicates, there are differences by sex. Sixty-seven percent of the injured men are now employed, but only forty-seven percent of the injured women are in the labor force.

Since women are somewhat more likely to say that their present health is fair or poor, rather than excellent or good, we looked at the relationship between sex and employment holding health status constant. As Table 2 shows, however, for every health status except "excellent", women are less likely to be

*This point was emphasized in a recent interview by Dr. John F. Finklea, director of NIOSH, when he noted that Workers' Compensation "does not cover birth defects or mutagenic defects." David Burnham, "Rise in Birth Defects Laid to Job Hazards," New York Times, March 14, 1976.

**This is the same proportion found in Vroman's study of a sample of even the most severely impaired New York City workers, injured in 1968-1970.

presently employed than are men.

The women are somewhat older than the men in the sample; 35% of the women and 23% of the men are now age sixty or over. When we control for age, it appears that the differences in labor force participation by sex are enhanced for the younger respondents and eliminated between the oldest groups of workers.

The interpretation of these differences in labor force participation between men and women is not clear. When we find men dropping out of the labor force after an injury we assume disability, but when women drop out, they have additional alternative and socially acceptable roles. Younger women may assume the responsibilities of non-employed wives or mothers. Older women may not have these options as readily available, may have invested more in their jobs, or may be more generally attached to the labor force. Married women are also more likely to have another adult wage earner in the household; even if their lost wages are not replaced, household subsistence may be guaranteed. The older married women, of course, are less likely to have employed husbands than are the younger women.

Table 4 summarizes some of the important relationships between household size and employment by marital status for women. Several categories have been collapsed because of the sample size. These data support the suggestion that the existence of other options and household needs are affecting the labor force behavior of the women in the sample. Married women seem to be "pushed" to work only when household size reaches four. The presence of any others in the household of the divorced, separated or never married women, however, seems to represent a demand for their wages.

When we look at the relationship between health and employment for women in different marital statuses (Table 5), marriage again seems to permit women more choices. While health affects the employment of all three groups of women, it is the not-married women who are more likely to work when their health is only fair or poor. It is these women who present a serious problem in the calculation of adequate compensation benefits. If the payments are too low to support their physical, psychological and economic recovery from work-related injury, they have neither the wages of husbands nor the pension benefits which widows may qualify for as a source of support.

Another question which should be raised but cannot be answered with the data available is the extent to which the permanent impairment resulting from these work-related injuries was a "push" out of the labor market for those married women with the option of not working. In a later paper we will explore the household income losses for the families of married and unmarried women and the labor force behavior of this sample of injured women with the labor force behavior over time of a national sample of working

women.

One mechanism with the explicit purpose of minimizing the effects of industrial injury is the rehabilitation system. In the survey states of New York, Florida, Wisconsin and Washington, the Workers' Compensation agencies include special rehabilitation units with the responsibility for identifying injured workers in need of medical or vocational rehabilitation and either providing services directly or referring workers for service, usually to the State Office of Vocational Rehabilitation. The survey instrument included a series of questions about rehabilitation services which was designed to produce an inventory of service types and sources. Table 6 summarizes the rehabilitation experience of the survey sample by sex.

The types of rehabilitation services which are most likely to be reported are those which promote immediate healing, such as casts, crutches, or prostheses; standard rather than extraordinary aids, such as eyeglasses, hearing aids, or basic surgical repairs. Very few received the vocational retraining or the elaborate support mechanisms such as specially equipped automobiles which seem to capture the public imagination when rehabilitation is mentioned. For each type of service, women were as likely as men to receive it, and those women who received rehabilitation were as likely as the men (about seventy-three percent) to report themselves generally satisfied with the services they had received.

Summary and Conclusion

We have reported data from two studies: a comparison of men and women workers seriously injured in New York State in 1970, and a sample of seriously injured workers in New York, Florida, Wisconsin and Washington, who were injured in 1970 and interviewed in 1975-76 about their subsequent recovery, labor force behavior and household characteristics.

These data can be summarized in terms of three central points. First, women do not appear in the Workers' Compensation population in proportion to their representation in the labor force. Second, those seriously injured women who do qualify for Workers' Compensation benefits are as seriously injured as men, both in terms of total permanent impairment and in terms of weeks of work lost. Third, five years after the injury women are less likely to be working than are the men in the sample. Their absence from the labor force is related to marital status, household size and age.

More questions have been raised than answered. For severely impaired women, as for women in the labor force in general, continued participation cannot be predicted in the same way that it can be for men. The exclusion of women from previous studies seems to have been in one sense supported. Women are different

from men and their behavior might inflate estimates of wage loss and disability. On the other hand, this neglect has also resulted in ignoring that group of not-married women for whom the consequences of impairment may be the most severe, since they have neither the additional household wage earner of the married women nor the potential earning power of their male counterparts.

TABLE 1

Sex of Injured Workers by Present Employment Status

	<u>Women</u>	<u>Men</u>
Employed	47%	67%
Unemployed	<u>53%</u>	<u>33%</u>
	100% (231)	100% (1264)

TABLE 2

Percent of Men and Women Workers Presently
Employed by Health Status Now

<u>Health Status</u>	<u>Women</u>	<u>Men</u>
Excellent	79% (33)	80% (237)
Good	61% (87)	77% (517)
Fair	35% (65)	59% (333)
Poor	13% (45)	32% (176)

TABLE 3

Percent of Men and Women Workers Presently
Employed by Age

<u>Age</u>	<u>Women</u>	<u>Men</u>
20 - 29	57.8% (11)	88.3% (154)
30 - 39	53.8% (26)	80.0% (235)
40 - 49	57.8% (45)	79.1% (268)
50 - 59	52.4% (61)	66.9% (311)
60+	31.2% (80)	32.4% (296)

TABLE 4

Percent of Women Presently Employed by Household
Size and Marital Status

<u>Household Size</u>	<u>Married</u>	<u>Widowed</u>	<u>Divorced, Separated, Single</u>
Live Alone		Live Alone 42.3 (26)	57.3 (21)
Two-person household	36.1 (61)	Live with Any Others 26.6 (15)	61.3 (31)
Three-person household	35.7 (28)		
Four or more persons	53.1 (49)		
Total	42.0 (138)	48.8 (41)	57.7 (52)

TABLE 5

Percent of Women Presently Employed by Marital
Status and Health Now

<u>Health</u>	<u>Marital Status</u>		
	<u>Married</u>	<u>Widowed</u>	<u>Divorced & Single</u>
Excellent or Good	60.3 (73)	63.6 (22)	84.4 (25)
Fair or Poor	21.5 (65)	33.3 (18)	33.3 (27)

TABLE 6

Percent of Injured Workers Receiving
Rehabilitation Services by Sex

<u>Service</u>	<u>Women</u>	<u>Men</u>
Special device, e.g. prosthesis, glasses, braces	18.8%	15.5%
Special equipment, e.g., wheelchair, automobile	7.0	5.3
Vocational training	6.1	4.0
Total receiving any rehabilitation service, including counseling, visiting nurse	27.0%	27.0%

REFERENCES

- Adams, Walter and A. J. Jaffe. "Too Little and Too Late...The Economic Plight of Very Seriously and Permanently Disabled Men and Their Households in the New York City Metropolitan Area," Supplemental Studies for the National Task Force on Workmen's Compensation, II, Washington, D.C., 1973.
- Berkowitz, Monroe. Workmen's Compensation, the New Jersey Experience. New Brunswick: Rutgers University Press, 1960.
- Berkowitz, Monroe. "Workmen's Compensation Benefits: Their Adequacy & Equity," Supplemental Studies for the National Task Force on Workmen's Compensation, III, Washington, D.C. 1973.
- Cheit, Earl F. Injury and Recovery in the Course of Employment. New York: John Wiley & Sons, 1961.
- Epstein, Cynthia Fuchs. Woman's Place. Berkeley: University of California Press, 1970.
- Jaffe, A.J.; Lincoln H. Day and Walter Adams. Disabled Workers in the Labor Market. Totowa, New Jersey: The Bedminster Press, 1964.
- Johnson, William G. and Edward H. Murphy. "The Response of Low-Income Households to Income Losses from Disability," Industrial and Labor Relations Review, 29, 1, October, 1975.
- Kreps, Juanita. Sex in the Marketplace: American Women at Work. Baltimore: The Johns Hopkins Press, 1971.
- Morgan, James N., Marvin Snider and Marion G. Sobol. Lump Sum Redemption Settlements and Rehabilitation, A Study of Workmen's Compensation in Michigan. Ann Arbor: Survey Research Center, 1959.
- Nagi, Saad Z. Disability and Rehabilitation. Ohio University Press, 1969.
- Safilios-Rothschild, Constantina. The Sociology and Social Psychology of Disability and Rehabilitation. New York: Random House, 1970.
- Vroman, Wayne. "Serious Injuries, Workmen's Compensation and Public Assistance in New York City," Supplemental Studies for the National Task Force on Workmen's Compensation, II, Washington, D.C., 1973.

GUIDELINES FOR EVALUATING
THE "DISABILITY" OF PREGNANCY

Dr. Leon J. Warshaw
Vice President and Corporate Medical Director
Equitable Life Assurance Society of the United States

I must acknowledge my thanks to the Society for Occupational and Environmental Health and to the Program Committee for the privilege of sharing in the so well-deserved tribute to Harriet Hardy last night. I have known her, learned from her, and loved her for many years, and it was a real treat to participate in so touching an occasion.

My presentation today is intended to acquaint you with a project that is just starting, to let you know how it came into being and where it is going, to direct your attention to it, and to invite your participation, because without your participation it will be meaningless.

The goal of this project is to protect the immediate and long-term health of the pregnant woman and her child. Admittedly, this is only one facet of the total problem of women and the workplace and women's rights. We heard yesterday that there is concern that we might focus down too sharply and perhaps neglect or pay too little attention to such important issues as economics, social welfare, legal rights, moral rights, and so on. However, I protest that we are a long way from one solution that will satisfy everyone or that will instantly be imposed and deal effectively with all phases of the problem.

Reality dictates that we approach it in a bit-by-bit fashion, seizing those facets of it that we can approach and resolving them as well as we can, constantly being aware that they are part of a larger framework and continuing to work toward the total goal.

Recent and pending laws, judicial decisions and much publicity governing equal employment opportunity for women have put the spotlight on the issue of pregnancy as a disability.

Most of the attention has been paid to such problems as discrimination in hiring or job placement on the basis of pregnancy; eligibility for various benefits; and the question that we have discussed so much at this meeting, the safety of both the woman and her child and of future generations. There has been a not-unexpected tendency toward polarization of the views of many of those concerned with particular aspects of the problem. As a result, positions have sometimes been taken on an arbitrary and all-inclusive basis that conflict with sound medical recommendations with respect to the health and safety of mother and child.

Medical judgment in this area is frequently clouded, first,

most important of all, as we have learned here, because we don't have the information. And, secondly, because obstetricians and those who care for the pregnant women are ignorant of the demands and the potential hazards implicit in many types of work, while industrial physicians have lost or, indeed, never acquired the expertise to guide women safely through pregnancy and delivery.

Several years ago the Council on Occupational Health of the American Medical Association moved toward the creation of a Subcommittee on Women and Work that was to be headed by Dr. Forrest Rieke of Portland, Oregon, who has spoken to this meeting a number of times. The Subcommittee also included Dr. Marc Bond, who was one of the discussers earlier in the program. Unfortunately, the Council and the Subcommittee were swept into discard when the need for economics forced the AMA to reorganize.

In any case, I am inclined to doubt that the subcommittee, whose broad charge extended to all aspects of women at work, would really have been able to come to grips with this particular facet of the total problem.

In the intervening years, Dr. Rieke, Dr. Bond, and several others who have been concerned with this problem have talked about it in the corridors of various medical meetings, and so on, and have waited patiently for action in some quarter to address it. Nothing has happened. And so several months ago, in view of the urgency of the problem as we saw it, we decided to see if we couldn't mount an independent effort to address it.

Because all of us are or have been officers or on committees of various professional and other organizations, we recognized that most organizations move slowly, particularly when they are professional organizations, and even more important, when the issue is one that demands collaboration among different organizations comprising different kinds of expertise.

So instead of going the other way around, we took it upon ourselves as self-appointed missionaries to address this problem, find a handle to it, and then present it to the appropriate professional and lay organizations for consideration, endorsement and implementation.

This has moved forward. The project is now formulated and it is about to get started. As I said, the goal is to protect the immediate and long-term health of the pregnant woman and child, and to do so on an individual basis.

The product of this project will be a set of guidelines to assist the practicing physician in developing appropriate objective clinical recommendations for the woman he is treating at the time he is seeing her. These guidelines will involve two fundamental considerations: the presence and the extent of any physical

and emotional impairment of the woman and/or the fetus; and her ability to meet with safety the requirements of her particular job. They will not deal with the question of whether or not she is or is not to be hired; whether or not she is to continue in employment; whether, if declared disabled, she is to be paid or not to be paid and at what rate; or whether or not the issue is one that is to be the basis of a grievance or labor-management discussion.

We recognize that all of these are important issues, and that they call for decisions. But those decisions rightfully involve the participation of many individuals other than the physicians. While they can be properly understood and made if based on appropriately sound medical recommendations developed through application of objective clinical judgment and upon the best available scientific information, they are neither the responsibility nor the prerogative of the physician.

Let me explain where the project is and how it will be organized, and then discuss the concepts which will guide its formation.

After considerable thought and much discussion, we decided to place the project, most appropriately, we believe, with the American College of Obstetrics and Gynecology, which has agreed to accept it, and to accept responsibility for staffing and directing it. We feel that this is fortuitous. ACOG, as is well known, is an organization comprising over 18,000 practicing obstetricians. It sets standards for clinical practice and its involvement will, we feel, facilitate the acceptance and use of the guidelines.

It will involve a staff of trained individuals who, in fact, are already at work, gathering basic information, accumulating research reports and collecting all of the data relating to pregnancy and work activity that are available.

The project will be organized along the following lines: There will be a "core panel" made up of occupational physicians and obstetricians who will serve as primary representatives of the key disciplines involved, and who will monitor the project, guide the staff, and be a focal point for identifying and reaching out for additional resources.

There will be an "expert panel" made up of representatives from the various scientific fields and disciplines that are involved. These will include experts in toxicology, teratogenics, genetics, oncology (the science of cancer and neoplasm), safety engineering, ergonomics, sports medicine, psychiatry, public health and others.

This group will involve a number of key individuals who will meet with some frequency and be available to staff. It will be supplemented by other representatives of the various disciplines

who will be involved through personal contact by staff or by mail and phone in review of particular points at issue. The purpose of this panel will be to expand and validate the scientific information developed by the research for this project from which the guidelines will be drawn.

Then, to make sure that we don't inadvertently do injustice to any of the other important considerations in our focus on scientific fact and medical opinion, a third panel will be established to provide a social dimension to the project and offer insights into the nonclinical effects of the clinical decisions made by the physicians who will use the guidelines.

This panel will consist of lay persons representing such fields as labor advocacy, personnel administration, women's rights, law, regulatory agencies, minority affairs, social affairs, health and disability insurance, and others.

These panels are in the process of formation. Some individuals in this room have already been asked to participate. There is still room for others and one of my missions here is to invite you to make nominations for people to participate in this way.

The guidelines will be intended to be just that, not hard and fast rules because such rules are not always applicable to an individual case before you. They will fundamentally be guidelines to the collection and organization of the data base upon which the physician will make his recommendations with respect to a particular woman at a particular time in her pregnancy.

They will call for inquiry into and examination of her state of health, the progress of her pregnancy and the condition of the fetus. They will call for consideration of the activities in which she is engaged and the hazards to which they expose her. When I say "activities," I mean not only work and the physical, emotional and other stresses involved in performing her duties, but also the special hazards such as radiation, noise, heat, cold, vibration, emotional stress and the dusts and chemicals to which she may be exposed.

I also include what I believe to be the most significant hazard to which most women and men are exposed in the course of their employment: the difficulty of commuting from the home to the job and back again.

Also included are the hazards to which women are exposed in the home: the physical work they must do; the chemicals to which they are exposed in the course of housework and hobbies; and the sport and recreational activities in which the family engages.

These guidelines will force the thorough collection of pertinent data and its arrangement in the framework of a systematized

decision logic so that at given points we can make sure that the proper questions have been asked, that the proper information is there (to the extent that it is available), and that an appropriate objective clinical decision is made with respect to proceeding further.

Because pregnancy is a dynamic activity that changes from month to month, indeed, from day to day, these guidelines will be subdivided on the basis of the various stages of pregnancy.

Thus, the conceptual framework says that we want to consider three variables: the worker, the pregnancy, and the job. To do this, we have constructed a matrix, if you will, that will consider the following prototype relationships at the sequential stages of pregnancy:

1. The normal woman with a normal pregnancy performing average work, work that is neither strenuous nor potentially hazardous.

2. The normal woman with a normal pregnancy performing work that does present certain potential hazards (e.g. radiation, chemical substances, biologic agents, climatic stress, strenuous physical work) or work that demands a high degree of physical skill, coordination and alertness.

3. The same conditions in terms of a normal woman with an abnormal pregnancy, either an individual with a very high-risk pregnancy or one with indications of some difficulty that calls for special attention in order to preserve the fetus through to the time of delivery.

4. The woman with a medical problem such as heart disease or diabetes, the so-called "abnormal woman," if you will, who has a normal pregnancy, and requires special consideration from the perspective of her own health.

5. And finally, the abnormal woman, if you will, the woman with a basic medical problem with a pregnancy that may be at risk because of an indication of difficulty of the fetus.

As I indicated, these will vary, indeed, women may move from one category to another, in different stages of the pregnancy.

Another question to be covered in these guidelines is the extent and duration of postpartum disability: when is the woman who has delivered able to resume her activities and does she need any special protection at that point?

By the way, the milestone chart for this project calls for the production of the first draft of the guidelines in a period of nine months.

We see these guidelines as a singularly useful and potentially important contribution. Their primary purpose is to permit the physician--indeed, to force the physician--to make appropriate recommendations based not upon the mythology of pregnancy, as Dr. Rieke so aptly calls it, but upon the best available scientific information, and examination of the condition of the particular woman in a particular job at a particular time. He is to make those medical recommendations objectively, using his best clinical judgment, letting the chips fall where they may, and allowing the woman herself and all others concerned with her "disability" to make decisions with respect to all of the other questions that her pregnancy may have evoked.

The primary objective of this project, then, is to protect the immediate and long-term health of each working woman and her unborn child by providing a process through which the practicing physician can exercise objective clinical judgment and develop sound, generally understood recommendations on her behalf.

We hope that these guidelines will be accepted and understood by those who will receive those recommendations: the industrial physician, the insurance carrier, the personnel director, the union representative, and all the others who will be involved in the other social, economic and other decisions with respect to the woman and her job. If all start with a common understanding that holds foremost at all times, the welfare of that particular woman and that particular child, conflict and disappointment are less likely.

We foresee a number of by-products of this project. First, that it will help to identify gaps in existing knowledge and set priorities for research that needs to be done.

Once accepted, these guidelines will set a standard for practice. In other words, it will force the practicing physician to take an interest in and to make inquiry into what it is that his patient does on the job, something that Ramazzini propounded centuries ago and for which we in occupational medicine have been striving for generations to accomplish.

It will also create a constituency comprised of practicing physicians and the one and a half million working women who have children each year to force the availability of information about the hazards to which workers are exposed on the job.

It will force a more equal partnership between the patient and the doctor. The patient must provide the information or see that it is obtained, the doctor must see that he gets and uses it, and they both must understand the reason for acquiring it.

This means that the physician must educate his patient about the significance of that information and explain how he uses it as a basis for his recommendations.

This will provide a proper basis for the most important decision of all: the decision that the woman, herself, must make with respect to her willingness to accept the risks of rejecting the recommendations that the physician has made.

Parenthetically, I recall vividly my experience some years ago when I was practicing as a cardiologist and was in charge of two cardiac clinics in New York City, each of which looked after a sizable population of young women with congenital rheumatic heart disease. One was an unaffiliated general community hospital; the other was a Catholic institution. I noted gravitation based upon decision making on the part of these cardiac women that governed which hospital they decided to attend when they became pregnant. Those who were concerned about their own well-being, who were concerned about being around to look after their older children, or wanted to be sure, if their condition developed to a point where a critical judgment had to be made about the risk of continuing the pregnancy, that the decision would be made in terms of the health of the mother, went to the clinic at the community hospital where therapeutic abortions were available and where, if a crisis occurred in the course of delivery and a choice had to be made instantly, the choice would be made on behalf of the mother.

On the other hand, those women to whom having a baby was more important than anything else, including their own safety, those who wanted to have that child at any cost and were willing to pay the price, turned up in the clinic at the Catholic hospital where therapeutic abortions were not practiced and where, at a critical juncture in the delivery room, the decision would be made in favor of the child.

This impressed me and has stayed with me through all as a demonstration of patients' rights to decide how they are going to live, what state of health they will have, and indeed, how they will die.

This may be an aside, but I strongly feel that we need to see that to the greatest possible extent patients, both men and women, have the responsibility to be partners in their own health care. They also have a right to understand the basis on which medical recommendations are made, and their implications, and to participate in deciding if, when, and how they will be implemented.

Now, they do it, unconsciously perhaps, by either changing doctors until they find one whose prescription they are willing to accept, or not filling the prescription and not following the doctor's recommendations. Very often that creates guilt in the patient and antagonism and rejection on the part of the health

provider. These deprive the patients of the benefits of professional support in following the decision that they, themselves, have made.

Another critically important by-product of this project will be the application of the guidelines in the development of educational materials for the pregnant woman. She needs to know them so that she can understand and participate in the decisions. She needs to know them so that she can hold the physician to whom she goes accountable for their use and their application.

We recognize that these guidelines will be incomplete, that we do not have now the knowledge that we need to govern every case. We know that they will require continuing revision as new knowledge is acquired and new types of practice are developed. But we feel very strongly that they will be useful and will provide, we trust, a starting point from which to address the major problem, the overall problem of women at work.

Again, I invite your interest and I invite your participation. This project is neither proprietary nor secret. It is not an arcane research project. The core of it will depend upon scientific information and expert opinion. Its application will call for mature and objective clinical judgment. But whether or not it works successfully will depend upon the way it is perceived, understood, and used by all those who are concerned with the worker who becomes pregnant.

And so, as I said at the outset, the purpose of this presentation is to acquaint you with the project and to invite you to communicate to me directly or through the American College of Obstetrics and Gynecology, or the Society for Occupational and Environmental Health, which will forward your communications to me, any comments that you may have with respect to this project, any suggestions or concerns that you may wish to express in terms of its design and execution, and the names of any persons, including your own, whom you would like to nominate for membership on one or more of the panels that I described.

Thank you very much.

PANEL DISCUSSION OF
SOCIETAL RESPONSIBILITIES AS SEEN BY WOMEN

PANELISTS: Andrea Hricko
Health Coordinator
Labor Occupational Health Program
University of California at Berkeley

Jeanne Culler
Staff Assistant for Occupational Safety and Health
Amalgamated Clothing Workers

Carolyn Bell
Industrial Hygienist
United Rubber Workers

MS. HRICKO: This Conference on Women and the Workplace has highlighted some of the health hazards women face on their jobs, but working women have other problems that intimately affect their health, both mental and physical. These cannot be ignored. Women are very real victims of discrimination and this discrimination takes many forms. Some facts are in order.

Although more and more women are working, they still earn much less than men. In 1974, the median earnings of women were under \$7,000 compared to earnings over \$12,000 for men.

Women work in less interesting, more dead-end jobs than men. Seventy-seven percent of all clerical workers are women compared to only five percent of all craft workers.

Women have been channeled into low-paying jobs like waitressing or into the lower paying jobs within certain professions and are denied equal access to higher paying jobs. For example, ninety-seven percent of all nurses are women; yet only ten percent of doctors are.

Many employers don't hire women at all, or hire women only if they can no longer bear children. Many refuse to accept women in apprentice programs, thus cutting off women's access to nontraditional jobs that are exclusively male. Some employers fire women when they become pregnant. Others take away a woman's seniority when she returns to work after having a child.

Nearly one-third of working women also have families. Many of their children are very young. Six million preschool children have mothers who work outside the home; yet there are only 900,000 licensed day-care slots in this country.

As a result, many women must cope with their jobs and home responsibilities at the same time and by themselves. Of all women workers, one out of ten is a family head, but one out of

five minority women workers support their families. Many of these women are too poor to afford decent child-care. Many of them are too fatigued at the end of a workday to carry the burden of household work that most often falls on their shoulders, even if a man is present in the household.

Inevitably, forcing women to work for low wages increases this country's social problems in terms of the worker's health and peace of mind, and also of her child's health, well-being and education.

Why are women still so far behind men economically and professionally? There are a wide variety of reasons, but one of the main ones is that there is a profit to be made by keeping women in low-paying jobs and it is certainly not a profit that benefits society as a whole.

Society has a responsibility to provide education, employment and economic equality for women and it has a responsibility to protect the welfare of children. Many countries around the world have responded to these societal responsibilities in positive, nondiscriminatory ways. They fully endorse women's employment. They provide free or inexpensive day-care centers and they have liberal maternity leave policies, as well as protection for pregnant women in hazardous jobs.

But job discrimination can affect women in other ways. The California Workers' Compensation Board recently awarded two women settlements for job-related depression. In one of the cases a 57-year-old woman worker had been employed by her company for over 30 years only to find that younger men, whom she had trained, were being promoted while her own role was diminished. She suffered severe depression as a result.

We also need to examine the consequences of channeling women into certain lines of work. Many of the hazards of jobs that employ predominantly women workers have never been adequately studied. I would like to briefly look at three of these job categories.

First, clerical workers. Their hazards range from noise in typing pool rooms to fumes from copy and duplicating machines to a proliferation of office machine chemicals and correcting fluids, poorly designed chairs, boredom, repetitive tasks, and finally to demands for more productivity and efficiency made by their predominantly male bosses. There are over ten million women clerical workers in this country and most of them are currently unorganized.

Over 100,000 women work in drycleaning establishments and in laundries. In 1863, women laundry workers in Troy, New York, went on strike over hot and oppressive working conditions and

low wages. Over 100 years later these same complaints are still being voiced. Perhaps there is no better example of workers not being informed about their job hazards than with workers who unknowingly launder industrial clothing contaminated with asbestos and other cancer-causing substances. There have already been cases of cancer and other diseases caused by workers who have brought clothing into their homes and contaminated either their wives or other members of their family.

For example, there are children in Tennessee who have high lead levels as a result of their parents bringing home contaminated work clothing and having the children touch them. Certainly, people who work in laundries and dry cleaning establishments face very serious hazards when they launder and dry-clean these clothes with no idea of the kind of chemicals that are on them.

Dry cleaners work with a variety of solvents. The widespread use of perchlorethylene raises the greatest cause for concern. A survey by the Industry of Dry Cleaning Establishments in Washington, D.C. revealed that peak levels over the allowable limit were common and that one plant had levels of perchlorethylene that was six times the standard. The National Cancer Institute is currently completing studies to see if perchlorethylene is a cancer-causing substance.

Finally, the electronics industry. Most of the workers are women. Many of them are nonwhite. A high percentage do not speak English. And few of them are organized. They work with a wide variety of organic solvents, including chloroform and trichlorethylene, which have already been demonstrated to cause cancer in animals.

The electronics workers have little information about the health hazards on their jobs. California workers with whom I have spoken say that dark-skinned women, including Filipinos, chicanos and blacks, are often assigned to certain departments in electronics plants where chemicals are used that cause skin rashes. This is done because the skin rashes are not as obvious on the dark skin and, therefore, the workers don't complain as much and the company doesn't have to deal with the complaints.

These women electronics workers report that some plants actually do not allow any language other than English to be spoken on the job, even in departments where the majority of women are non-English speaking. We must ask how these women are warned about job hazards by their supervisors or how they are able to read warning signs about job hazards.

A group of concerned workers in an electronics plant in Santa Clara Valley in California prepared a leaflet called "The Printed Circuit," which had information about the hazards of trichlorethylene in it. In order to circulate that pamphlet to the

workers, they had to leave copies of it in the women's rest room so that the male management people would not snatch the copies away before workers were able to obtain them.

Over seventy-five percent of the country's 271,000 electronics workers are estimated to be women. To my knowledge, there has been no systematic studies of any of the health experience of workers in the electronics industry, the dry cleaning and laundry industry or any systematic study of hazards for clerical workers.

Society has an obligation to provide jobs to all workers who need and want them and to make those jobs safe and healthful. This obligation affects the well-being of women, men, children and society as a whole. Until the obligation is met, women workers must keep struggling to change an inequitable system that provides them with less than equal protection under the law.

Only seventeen percent of women are organized into unions or associations in this country. Many women's organizations and unions have begun active campaigns to organize the unorganized, to promote concerns of special interest to women such as equal pay, entrance into apprentice programs, day-care, maternity benefits, voluntary overtime and occupational health. Other efforts have been made to increase the number of women among elected union officials and women elected to government offices.

As women gain an equal voice in this country's power structure, perhaps our priorities and values will once again reflect that people are a vital resource in this country, and that this country should do its best to preserve that resource.

MS. CULLER: Over the past couple of days we have heard a lot of impressive and alarming statistics and facts about women and the hazards they face. Thursday evening at the Coalition of Labor Union Women's open forum, we heard from the women themselves, the women who work in these hazardous environments. We heard of their fears, their anger, and their frustration in dealing with bureaucratic tangles. I would like to relate some of the comments that were made at that meeting because I think they will serve to remind us that behind the facts, figures and statistics we have heard over the past few days, there are people, human beings who need protection and help.

Here are a few of those comments.

"Someone has to listen. What happens when we forget about people? We need your help."

Now, I get kind of tired of hearing the phrase, "You have come a long way, baby," because I think it is really deceptive. I think it is really misleading. Certainly, the women's movement

has made great strides forward. We have all made great strides forward in changing this country and turning it around and in trying to advance the goal of equality of the sexes, but we still have a long way to go.

The duplicity of our society's attitude towards pregnant women really demonstrates how far we do have to go. The fact is that we romanticize motherhood. We treat women, particularly when they are pregnant, in a discriminatory way. "We," meaning our society, and we all have to work to change it.

In this context I would like to talk about health and economics. The health care system inadequately provides for the costs of pregnancy and birth. For example, one study revealed that under a Pennsylvania Blue Shield Plan, thirty-seven percent of the average doctor's charge, including hospital costs, for a normal delivery was covered. However, the comparable coverage for an appendectomy under the same plan was seventy percent of the costs. I think that is a pretty wide gap.

Our country ranks 17th -- which isn't too much to be proud of -- 17th in the countries of the world in terms of deaths of infants during the first year. A study done by the School of Public Health of Columbia University estimated that more than fifty-one percent of infant deaths in some New York hospitals could have been prevented with proper prenatal care.

Also, the lack of emphasis on preventive health care is very important, particularly in the case of occupational diseases which, like all other diseases, if caught early, can be dealt with more successfully. So I think that the fact that our current health care system lacks that emphasis is really critical in terms of our occupational exposures.

All of this adds to the picture of an inadequate health care system that is costly to the consumer, and that discriminates against women, particularly pregnant women.

The expenses relating to pregnancy and the insufficiency of services available present problems that are compounded by the threat of job discrimination. I think we have heard enough in the past couple days to know that a woman may lose her job because she becomes pregnant, or lose her seniority, or may not even be able to get a job in the first place just because she is fertile.

Now, all workers are subject to the threat of environmental or occupational blackmail. Implicitly or otherwise, industry presents the worker with this choice. If you really want to press on this health issue, then you may lose your job. We may close down. We may go someplace where they don't have OSHA standards.

And the case of a pregnant woman is really no different. It

is the same kind of threat. It is the same type of intimidation. The main difference is simply that it has a new dimension because the fetus is involved. I believe that the main reason employers are keeping women out of certain types of employment which are hazardous to the fetus is to protect themselves from financial liability, and not out of an overwhelming concern for the mother or the fetus.

So the pregnant woman is treated as a second-class citizen in terms of her health coverage, in terms of the health services available to her, and in terms of the costs that she pays for them. And all these problems are compounded by the fact that at the time she most needs financial security, her job is threatened.

Like their male counterparts, women are inadequately protected from on-the-job hazards. Part of the problem is the failure of OSHA to adopt standards for which criteria documents have already been developed. Of some twenty criteria documents covering health hazards, such as benzene, toluene, noise, heat stress, and the handling and labeling of hazardous materials, only a few have actually been turned into actual standards that are enforceable under the law. Enforcement is another question and another problem. There aren't enough inspectors. Serious hazards are often treated as non-serious hazards. And, because abatement periods for health violations are often unreasonably long, people go unprotected for long periods of time.

Added to the onslaught of unchecked workplace hazards and dangerous chemicals are uncontrolled dangers in the water we drink and the air we breathe and in the products we buy and use in our homes.

I have one issue in that area I would like to bring up just to show that OSHA isn't the only bureaucracy that is not responding to its citizens' needs, and also because this particular item has to do with pregnant women. The Center for Science and the Public Interest, which is a Washington-based, public interest firm, requested that Dr. Theodore Cooper, U.S. Assistant Secretary of Health, have HEW warn women of childbearing age and physicians that caffeine may cause birth defects and other reproductive problems. The CSPI documented this for Dr. Cooper. I think they even referred to an FDA study that had been made. The letter they sent on February 3rd to HEW ended up at FDA. Who knows when they will get around to doing something about it?

But women have to be aware of the hazards they are exposed to so they can make judgments; so they can decide whether they want to cut down on their caffeine consumption; so they can assess what kinds of hazards they are exposed to at work. We are not going to have protection other than that which we feel we need for ourselves, and which we are willing to go after. We have to have information.

Before I close I would like to make one suggestion. I think that we can approach it through legislation. I think that occupational health hazards and the hazards in the home are not really going to be taken care of for a long time, and that a long time will pass before we get around to covering synergistic effects, the combination of all these hazards we are exposed to. And if we don't have a good health care system that really acts in terms of preventive medicine, then we are going to go unprotected. So I would urge you to support two kinds of legislation: one for national health security and the other for toxic substances control.

Finally, I would like to say that I think one of this country's priorities ought to be to provide health care and health protection for all of its citizens regardless of their race, regardless of their sex and age, and regardless of whether they are fertile, gravid or lactating.

MS. BELL: When I was initially contacted regarding participation in this Conference on Women in the Workplace, I had mixed emotions towards the idea of addressing such an issue, because I sincerely believe that a worker is a worker and that all of us should be guaranteed the right to a clean and healthful working environment regardless of sex.

I, like many women present at this conference, am somewhat fearful of what the repercussions will be towards our economic and social stability if we, as women, allow ourselves to be treated separately.

It has taken us 188 years, 1776 to 1964, to get an act passed which makes it illegal for American citizens who are women to be discriminated against because of sex. I, for one, would not like to jeopardize these gains by getting caught up in an age-old tactic of divide and conquer just when we, as women, are beginning to show some visible signs of progress.

There are those among us who would say, "Oh, that is hogwash. We wouldn't dare discriminate against women. We are solely interested in protecting you and your unborn child."

But I say to you -- and I speak from experience as a black American and as a female American -- don't you believe it. We must fight to protect our current status and we must continue to go forward.

As an example of sexist discrimination, I would like to relate to you briefly some problems that I encountered when I tried to visit a URW vinyl chloride facility. On March 30, 1976 I visited one of the URW vinyl chloride plants on the eve of the mandatory usage of respirators in the event of exposure to vinyl chloride above one part per million over an eight-hour period.

The purpose of the visit was to observe workers who presumably would have to wear their respirators for a full eight-hour period, in order to comply with the OSHA standard, because no clean-air areas had been established to give workers relief periods during which they would not have to wear the respirators.

When I arrived at the plant at about 11:30 p.m., I was informed that I would not be allowed to enter because the plant had a policy which did not allow women of childbearing age to come into the facility. The situation was finally resolved and I was told that I could go in if I wore a respirator. Can you imagine what the problem would be for a woman who would want to be hired by that plant as a vinyl chloride polymerization worker? Exactly. Her chances would be nil.

Fortunately or unfortunately, there were no women workers at that plant except for the secretarial staff. I was informed later that the secretaries do visit the floor of the plant periodically to hang messages on the bulletin boards and that they do not wear respirators. When I found this out, not only did I feel discriminated against, but I felt that I had received a snow job in the process.

I was especially impressed with the comments made by previous speakers regarding the fact that many of the chemicals we are concerned about may affect the male's ability to produce a normal offspring, as well as that of the female, who may be working alongside him.

As a representative of the United Rubber Workers Union, and in view of a large number of URW workers, who are exposed to vinyl chloride, vinylidene chloride, trichloroethylene, chloroprene and other halogenated hydrocarbons, I wish to reiterate and support the question raised by Dr. Joseph Wagoner in his presentation. Does society plan to consider the problem of mutagenicity and carcinogenicity as a female issue only, or will it also address itself to the offspring that will be produced by the male worker who obviously is at risk?

The question of a female exposure to teratogens and trans-placental carcinogens obviously forces one to address one of the major physiological differences between the male and the female. Women are equipped to bear children and men are not. The answer to this question is an easy one. You simply prevent exposure. But the method by which you prevent exposure could be very complex and could contain many willful or unintentional ways of discrimination that could economically and socially devastate the life of a female head of a household, as well as her dependent family.

Can you rightfully say to a woman, "You can't work because you are pregnant?" The right to work is her privilege. Can you envision the mess that would occur if you tried to separate women

on the basis of light, medium and heavy exposure to transplacental carcinogens?

"Are you pregnant? Well, if you are, you should receive light exposure."

"Do you think that you may be pregnant within the next two months? Well, if you are, you should receive medium exposure."

"Do you take the pill? Well, since you won't be getting pregnant soon, you can receive heavy exposure."

These questions, obviously, are ridiculous and can serve as no approach for handling the problem.

My opinion is that women of childbearing age should only be exposed to safe levels of hazardous chemicals that have been determined by Federal agencies designated to establish standards in order to protect the health and safety of all workers, male or female. If such levels cannot be achieved and there is absolutely no other way to protect the woman of childbearing age except to remove them, then let them be moved without economic penalty. This transfer or removal without penalty should be guaranteed by the Federal government.

This is the first societal responsibility that I bestow upon you. Secondly, the woman worker is a part of society and must assume a political role to influence the elements of society which are responsible for developing acceptable levels of exposure. Women must become more visible with national standards advisory committees such as NACOSH, the National Advisory Committee on Occupational Safety and Health; and the advisory committees to the OSHA standards, which are now in the process of being promulgated, and NIOSH criteria documents. I don't know what the statistics are in terms of women participation on these committees, but it is at these levels that we must begin to sound off our specific complaints regarding the unfairness of Federal standards, which are being designed to protect or wreck us.

We need more women participation in local union activities such as the joint labor-management safety committees or local union officeships.

At this point I would like to quote some statistics from Andrea Hricko's recently published book, "Working for Your Life, a Woman's Guide to Job Health Hazards."

The 4,524,000 women workers, who belonged to labor unions in 1972, constituted 21.7% of total union membership; thus, two out of every five workers in the U.S. are women, but only one out of five union members is a woman.

In union leadership, men generally still hold the most posts on national governing bodies, even in unions where a majority of members are women. In the 24 unions with more than 50,000 women workers, only six of 187 national offices and officials were women, and only 18 of the 556 executive board members were women according to a 1972 survey.

Women workers, as well as male workers, have a responsibility to develop effective contract language that would address itself to safety and health hazards experienced in the workplace. Currently, the Rubber Workers are negotiating contract language that would provide medical benefits for the female worker who becomes pregnant. The recommendation is as follows: make maternity benefits the same as benefits for any other temporary disability, including but not limited to leaves of absence, hospitalization coverage, sick and accident benefits and seniority rights.

In addition to this language, we have recommended a monetary increase in our negotiated joint occupational health program with Harvard and the University of North Carolina Schools of Public Health. Additional monies will allow these schools to step up their research efforts and to initiate the Ames mutagenic test to evaluate the cancer potential of currently used and new chemicals in the workplace.

Workers must negotiate contract language that will require the company to supply the worker upon request with medical surveillance reports, environmental sampling data, and a list of all chemicals that workers are being exposed to in the workplace, as well as the toxic properties of these chemicals. It is not enough just to know the names of the chemicals. We must know how they will affect the male worker, the female workers, as well as the unborn child. If the chemicals are used at levels which may be harmful to the worker, then he or she should be informed of these hazards without request.

While organized labor represents some 22 million workers in the work force, who is available to inform the other 56 million workers of the occupational safety and health hazards which may confront them? Society has the responsibility to assure that these workers are protected and are not overlooked by the representative governmental occupational safety and health agencies. All workers must be educated and made aware of their rights under the Occupational Safety and Health Act. It is up to the society to afford the worker this important knowledge to which he or she is entitled.

Finally, I would like to address the issue of control measures being economically and technically infeasible. I have heard now on numerous occasions that it is impossible to use engineering techniques to control a hazard in some instances because the economic impact would be overwhelming or because we have not yet

developed a control design that would be effective.

President Ford, speaking before the Chamber of Commerce last year, commented that the estimated figure of over \$30 million would be too much to spend on noise control. I say that this is not the right approach. I say to you this morning that society must reassess its priorities. If this country can put a man on the moon some 238,000 miles away from this planet Earth, then I refuse to believe that we cannot plug up a few leaks and holes and install some control techniques that would protect the worker right here on earth. We want standards to protect us. We want them enforced. And we want them now.

If we can afford to provide services to the elderly, to the handicapped, to the economically deprived, and if we can send people to the moon, and if we can pay people salaries to do nothing but sit in Congressional offices and be convenient playthings, then certainly we can provide a safe and healthful work environment for people who go to work every day and pay taxes which support these other services.

Remember, that without the American worker, America would cease to be.

In testimony before the House Subcommittee on workers' compensation, speaking on behalf of URW President Peter Bomarito, I made the following comments:

"In this bicentennial year of our great country, it becomes appropriate to say at this point that the lack of a uniform workers' compensation program to ensure workers adequate, prompt and equitable compensation for occupational illnesses and injuries suffered is akin to taxation without representation.

"We are taxed with the obligation to work, to make a living, yet we are not adequately represented at the compensation tables."

As I sat through this conference during the last couple of days, the more I was convinced that the woman worker and her unborn child are in the same predicament as the worker who does not receive adequate workers' compensation benefits. We are good citizens, and we do go to work every day and we pay our taxes. Yet where is our representation? Standards must be developed and control measures must be implemented that will protect all workers, and these levels must be low enough to include the pregnant mother and her unborn child.

In conclusion, I would like to read some comments which were given to me by a female URW member, Ms. Shelby McLaughlin.

"In the factory where I work, we have had ten people hospitalized within the past few months with blood clots; one male and nine females, all from the same department. The male died. He was 42 years of age with a wife and two children. I know what some people think, that women who take birth control pills are prone to have blood clots. But I assure you that Mr. James Coffey did not take birth control pills."

We are investigating these cases, but we don't know what caused the blood clots. I certainly hope someone finds out before more children are left fatherless and motherless. In closing, women in the workplace is not a happening which just recently started to occur. I have five brothers and sisters, and my mother worked for 33 years. So did most of the other mothers in my community. In black America this was and still is a necessity for survival, and in many instances this situation is identical among women in households of other communities. Today women are in the work force in numbers greater than ever and I can assure you that we are here to stay.

QUESTIONS, ANSWERS, COMMENTS

MR. ERIC FRUMIN: I am with the Department of Occupational Safety and Health, the Amalgamated Clothing and Textile Workers Union. I have a response rather than a question for Dr. Warshaw. Doctor, you said that the purpose of your panel is to make decisions based upon appropriate, sound medical judgment based upon available information.

I think if most workers made such decisions, they would get out of the workplace as soon as possible. The available information to them is that a workplace is a dangerous place, it is a lousy place to be, although they want to work and they would like to work.

On the other hand, they are not allowed the freedom, the luxury of making such decisions in the absence of the economic considerations which you and your panel apparently are choosing to ignore in wrestling with the problem. Their next meal is certainly as important to them as the woman who would prefer to kill herself rather than let her child die in a therapeutic abortion, which means that for workers, if they have to choose between starving the next morning and coming down with cancer in 20 years, the choice is rather obvious.

As for the American College of Obstetrics and Gynecology, I am a little enraged by the tone of what you had to say. I feel that involving them in this activity represents a severe conflict of interest given their record of tendency to abuse women in this country with unnecessary hysterectomies. I can't imagine a more

fertile ground for unnecessary hysterectomies than fertile, lactating or gravid women, who are exposed to toxic substances in the workplace. So I think the ACOG should be gotten out of there entirely.

The fact that you should quote Ramazzini in some reference to cleaning up the workplace is very interesting. Dr. Kilburn yesterday quoted Ramazzini as identifying cotton dust and flax dust as a hazard back in 1709. The corporate medical profession in this country attempted to keep the cover on that one all the way through 1965, and textile workers are suffering by the thousands because of it.

Now, the point that I want to make is this: We are facing a conspiracy of silence in this country on the problems of health as they affect workers in the workplace. Women, in particular, have suffered because of it. And I would like to say that it is up to those of us in this room, who are serious about doing something with the problem, to seek to get that information. Further, I maintain that the information is not solely contained on the North American Continent. I am shocked and dismayed by the fact that there are absolutely no representatives from other countries here. We have no one to tell us about the experience in Western Europe, in Eastern Europe, or in the Soviet Union.

Now, anyone who has ever looked at those countries and what their experience and their efforts have been knows that in many ways their experience is far superior to ours. They have national health services, which we do not have. Women do not face the type of discrimination in employment, and their occupational safety and health epidemiology is, in many ways, far superior to ours. Unfortunately, to try to get something translated in this country is impossible. So to those of you who are serious about doing something to protect women in the workplace, get that foreign medical literature, and force NIOSH and OSHA to start translating it so that we don't keep sitting in a closet in ignorance.

MS. LEE: Perhaps Dr. Warshaw would like some equal time.

DR. WARSHAW: Your comments are indeed well taken, but for one point. I made a special point of saying and emphasizing that the economic, work rights issues and so on are important, but I decry keeping women and others in the dark. I decry the method of volunteering for hazardous duty that was so often employed in military service when they lined up a company and said, "The first line will take two steps forward. You have just volunteered for a particular assignment," and so on.

Yes, I think that the question of women's rights and the question of whether to accept risks or not in terms of work, I think all of these are important. But I protest that we must start with an understanding of what those risks are. I don't

think the union has any more right than management or anybody else to make that decision on behalf of an individual woman. I think we have to start from a basis of sound scientific knowledge, good clinical judgment, and then take it from there and let the chips fall where they may.

DR. LEBRUN: I am here talking on my behalf and not on behalf of the company of which I am the Medical Director, namely, FMC.

I would like to limit my remarks strictly to the medical aspect of the question. That does not mean that all the other aspects, political, social, economical, financial, moral, et cetera, are not important. They are, but it is impossible in a few minutes to review those things. We have been here three days. We have not solved much, I feel.

I believe I can say that in this meeting there is the following consensus:

(a) Women react, generally speaking, the same way as men to most of the industrial stresses.

(b) There are probably a few agents to which the susceptibility of either sex may be different from that of the other sex, and I submit that in such a case the physician has to take this difference into consideration when dealing with a worker.

(c) There are probably some agents for which, because of the presence or absence of a specific target organ, either sex may be at a particularly high risk. I wonder whether we would have heard of Percival Potts if chimney sweeps had been women instead of men.

(d) We are left -- at least as far as I am concerned -- with the particular function of women, childbearing. I insist childbearing, not conception.

We are dealing here with a different person altogether, namely, the fetus, who has its own individuality legally and physiologically, and who technically should not be present in the workplace. We do not allow the presence of children under a certain age in the workplace, let alone of the youngest, namely, the fetus.

From what I have heard here it seems that many people are equating protection of the pregnant woman with protection of the fetus. These are two completely different subjects. The question still would be the same if it were the man who were bearing the child.

The pregnant worker -- I use a sexless word purposely -- the

pregnant worker needs protection of a quality compatible to the one given to somebody whose physiology is temporarily impaired such as after surgery, for instance.

The fetus, because of its much greater susceptibility to many agents and also because of an ignorance of the possible effect of new agents which are brought into the workplace daily, needs a much higher quality type of protection.

I have heard a suggestion that levels should be set to protect the fetus, also. Although I wholeheartedly support a general concept of zero exposure, I would like to see whether it is technologically, economically, or socially feasible. And I apologize for the pun, but we should not throw the baby out with the bath water.

Be that as it may, we know that the various solutions that have been considered or suggested are long-term solutions, but women are getting pregnant every day, and what is the physician going to do Monday morning with a woman of childbearing capability or a pregnant woman exposed to a potentially harmful substance. I think this problem needs immediate solution and I do not know what exactly it is from the social standpoint. I know what it is from the medical standpoint.

It is, therefore, clear to me that the solutions of those various problems that we have considered are going to be based much more on a societal response than on a scientific response.

There was a suggestion made this morning that management is much more interested in the liability aspect of harm to the fetus than in the medical aspect of harm done to the fetus. I challenge that because I think that there are many more people really interested in doing good around them. That management is also interested in the liability aspect is quite normal. It is part of their responsibility toward their shareholders. Let's not forget that many people in this room may be shareholders, too.

We have always to balance cost and benefit, not only in the matter of dollars, but also in society's cost and benefit. To take an example, about 40,000 to 50,000 people are killed every year on the road. Yet we do not ban the cars.

MS. HRICKO: Doctor, I believe that you were expressing your concern about the problems of pregnant women workers and if I understood you correctly, you were representing yourself, but you are with the FMC Corporation?

DR. LEBRUN: Right.

MS. HRICKO: I would like to ask you what you would tell the male workers at the FMC Corporation, Nitro, West Virginia, who are

exposed to carbon disulfide. I would like to explain that there is a Romanian study published in an Italian journal which says that male workers can suffer from sexual problems after exposure to carbon disulfide; and that after exposure to carbon disulfide for three years, male workers were found to have decreased libido, which is decreased sex drive. Males were also found to have erection troubles during intercourse after exposure to carbon disulfide. In addition, males were found to have a five times higher sperm abnormality rate than did a control group who was not exposed to carbon disulfide.

What do you tell these workers in Nitro, West Virginia?

DR. LEBRUN: I think that you have missed my point. My point was directed toward the fetus. I insisted very much at the beginning that we should not confuse the problem of the pregnancy per se with the problem of the fetus after conception. Now you are shifting the discussion.

If we are talking of the risk to an adult individual, I have to tell the employee, male or female, what are the risks involved in working in a particular situation. Then he or she can make a choice. The fetus has no choice.

MS. HRICKO: Are you suggesting that sperm abnormalities have nothing to do with conception?

DR. LEBRUN: I am not talking about conception. I said very clearly "childbearing" not "conception." These are two different problems.

The problem that we have with the fetus after conception is different from the problem that we have with the reproduction capability, or the mutagenic or teratogenic effect of a compound. An adult can make a decision about that, whether he or she wants or doesn't want to take the risk of conceiving children who may have some deformity. But that decision is taken before conception.

MS. HRICKO: So you do recognize that before conception something can happen to the male that can result in a birth defect in the offspring?

DR. LEBRUN: I said from the very beginning that we agree on that.

MS. HRICKO: I am glad you recognize it. Thank you.

MS. GLORIANA ARCENEUX: I am with the American Nurses Association and I have two questions directed to Dr. Warshaw. I was wondering how are occupational health nurses to be involved, if at all, in the formulation of the study plan? And secondly, are there any occupational health nurses appointed to any of the three panels?

DR. WARSHAW: Occupational health nurses are involved in two ways: One, as members of an appropriate professional group. They are not on the basic core panel; they are on the specialty panel that involves various professional groups which have different insights professionally and technically into the problems of pregnancy. We are also including the nurse midwife, the obstetrical nurse, and others.

The nurse is also involved and undoubtedly will be represented on our advisory panel. There she will be a woman representing a group of professional women and bringing her expertise and knowledge to the conference table and to the consideration of issues. The advisory panel is intended to deal with things from the standpoint of laypersons and not in terms of technical and professional expertise.

MS. ARCENEUX: They had no input in the formulation of the study?

DR. WARSHAW: Oh, indeed they do. May I make a point that all three groups are involved in the formulation of the standards. May I also remind you that the protocol I gave is a tentative one. The protocol will have to be approved when recommendations are formulated by all three panels, not just the core panel of physicians, not just by staff, but also by the advisory panel of lay individuals. Through their understanding and their interpretation of the project and the guidelines, these lay individuals will make sure that we don't cloak answers and decisions to social and societal problems under the guise of medical judgment and recommendation. The whole purpose of this is to separate the two, to make the physician function as a professional person, as a scientist, and as a counselor, who makes recommendations purely on clinical grounds, and to prevent him from being in a position where he might be accused of distorting his recommendations for purposes other than clinical judgment. The recommendations that he makes are just that, recommendations. I want to make that very clear. Decisions are made by others. Once he has made his recommendations the physician becomes a citizen and his voice carries no greater volume and no greater strength than that of the pregnant woman or those who are interested in the problem on her behalf.

PANEL DISCUSSION OF
LEGAL REQUIREMENTS AND IMPLICATIONS

PANELISTS: Peter Robertson
Director, Office of Federal Liaison
Equal Employment Opportunity Commission

Portia Y. T. Hamlar
Attorney, Legal Department
Chrysler Corporation

Anne Trebilcock
Assistant General Counsel
United Auto Workers International Union

MR. ROBERTSON: As most of you probably know, the Federal law making it illegal to discriminate in employment was actually passed originally in 1964 as part of the Civil Rights Act of 1964.

The Equal Employment Opportunity Commission was given the power to investigate complaints and to attempt to eliminate discrimination by conciliatory means, but not power to go into court and enforce the law.

In 1972, Congress found that that mechanism had failed and they gave EEOC the power to go into court and sue. When Congress gave EEOC enforcement power, they advanced the theory that the main reason the voluntary mechanism had failed was that the average employer lacked the technical perception to recognize that his system was discriminatory. And then they went on to sketch out what they meant by this technical perception phraseology. And, basically, what they suggested was that our perception of discrimination, the way in which we think about discrimination, has gone through three stages.

In the first stage, we looked at intent or motivation, and used such words as "bias" and "bigotry" and "prejudice." An investigation was an attempt to find the bigot.

In the second stage, we began to focus on the behavior of employers and we looked at a limited set of their actions, those that were unequal. Blacks and whites similarly situated were treated differently. Males and females similarly situated were treated differently. Anglos and chicanos similarly situated were treated differently. That was the second stage, an examination of unequal treatment.

In the third and much more important stage -- the one in which Congress found that the average employer lacked the technical perception to recognize that his system was unlawful -- is the stage at which one begins to look at the functioning of an

employment system and at its impact.

Basically, that is what EEOC had been doing as it made findings of discrimination. And what employers were saying when they refused to conciliate voluntarily is, "We don't think you have any business looking at our system. Tell us about bias, bigotry, prejudice. If you can find a foreman who said he didn't want to promote a black, we'll deal with that. Tell us about unequal treatment. If you can find that we are paying men and women different amounts of money for the same job, we'll deal with that."

But EEOC was handing them pieces of paper that said, "You have got a seniority system that excludes blacks from certain jobs and you have got to change it." EEOC was handing them pieces of paper that said, "You have got a recruitment system that uses a word of mouth method and blacks never find out about your jobs. You have got a promotion system in which male foremen are evaluating women on a subjective basis and women end up not getting promoted and you have got to change that system."

And the employers were saying, "Yes, we agree with you on the facts. That is an accurate description of our system. But that is not illegal because it is equal treatment. Blacks and whites go through the same seniority system. Men and women go through the same system. We take a black who walks in the door for hire as well as a white or a male as well as a female." Employers claimed that they were applying an equal treatment standard.

So the law has gone through these three stages: intent, equal treatment, and a stage at which we look at the impact.

Now, let me tell you one of the ways in which this third stage definition has been defined by the courts. When you look at the impact of an employment system, you don't just say that because it has a negative impact on blacks or women or chicanos it is illegal. If it has such an impact, you shift the burden to the employer to show that there is a business necessity for the practice.

And the courts have developed a very interesting definition of business necessity. It is a two-stage definition. First is an old common sense business definition. Is the practice in question necessary for the safe and efficient operation of the business? Secondly, and this gets to the heart of Title VII, is the employer able to prove that there is no alternative available that could have achieved his business purpose without excluding blacks, women, chicanos or whatever the group?

For example, if you have got a departmental seniority system in which women are frozen into certain departments and they want to transfer to the previously all-male department, but they have to go to the bottom of the seniority ladder, the courts have held

maybe there is a business purpose to a seniority system. You get more experienced people. But can the employer prove that the particular seniority system he had was the only way to do it?

In fact, after looking at two types of seniority systems, a company system that had an exclusionary impact on blacks, and an alternative system that did not, one court said, "If there are two ways of doing it, don't tell me the one that operates to exclude blacks is necessary." Another court said the key to business necessity is whether the employer has a choice, and that is really the key word.

There are some cases with good old garden variety intent where an employer is using a health hazard as an excuse because he doesn't want to get women in a job. You can deal with that fairly easily. There are some examples of unequal treatment where health hazards that probably impact pretty much the same on men and women are used as an excuse to exclude women only. But the bulk of these situations look to me like we are dealing with a situation in which an employer, who has some kind of hazard, chooses to deal with that hazard in a way that impacts on women. The law says he has got to prove there was no other way to deal with it. The law says he has got to prove he had no choice. If an employer has two ways of dealing with a health hazard, and if he picks the way of dealing with the hazard that excludes women, he sure better be able to prove that there really was no other alternative, because if there is an alternative that wouldn't have the exclusionary impact on women, the law says he has got to take the alternative without the impact. And it is not enough to prove cost. Now, I suppose if he can prove bankruptcy-type costs he might win, but the courts have pretty consistently said that marginal costs don't meet the business necessity defense.

A quick example: Blacks tend to have garnishments more than whites. A company excluded employees who had their third garnishment and the employer attempted to prove business necessity by talking about the costs of processing the garnishments. And the courts said that that was not sufficient proof, that excluding people who had garnishments basically operated to exclude many more blacks than whites and the slight cost attributable to it was not a business necessity defense.

I think the courts will apply the same kind of principles to employment discrimination resulting from health hazards. If the employer has options and he selects the option that excludes women, I think he is going to have a very strong burden to prove why he couldn't use that other option.

Now, an old colleague of mine, Harriet Hendler, who is with the Pennsylvania State Commission that enforces their antidiscrimination laws, has dealt with some of these, which I have never done. I asked her for some thoughts and she threw out a whole new

way of thinking about the problem. It is her opinion that if these jobs had been traditionally held by women, the health standards would have been framed from the start in terms of women, and that an unequal treatment case may exist simply because the health standards have been framed in terms of the men in those jobs. And so, as a matter of law, the answer may be simply to frame the health standards in terms of the women who occupy the jobs.

So here are the trends of legal thinking that I would like to leave with you this morning as you think about employment discrimination. If you have a case in which a health hazard is being used as an excuse because somebody just wants to get rid of a woman, that is fairly easy to deal with.

The second situation is to try and apply an unequal treatment standard. Has the employer chosen to set his health standards for men rather than for women? Is that unequal treatment?

And third, if the employer has a choice of methods and if he has picked the method that has an exclusionary impact on women, what are his choices? Could he transfer the women to other departments instead of laying them off? Can he give them medical leave instead of firing them? There are probably more choices than occur to me at this time, but the essential point is that the burden is on him to show that he couldn't have used one of those choices that did not have an exclusionary impact on women.

MS. HAMLAR: I speak to you today about the legal dilemma of women's occupational health problems not only as a representative of industry, but more importantly as a lawyer with considerable experience in OSHA litigation, and with present involvement in EEOC proceedings.

During the past two days, attention has been focused upon certain health hazards to which women may be exposed by virtue of their employment, and upon the problems presented to industry in its attempts to reduce the effects of those hazards. In this connection, American industry is subject to regulation by the Occupational Safety and Health Administration, more commonly known as OSHA, and by the Equal Employment Opportunity Commission, or the EEOC. Regulation by these two government agencies presents a dilemma frequently experienced by industry -- that of conflicting government objectives. However, the dilemma presented by the revelation of women's occupational health problems is new, basically unexplored, devoid of extensive and conclusive scientific knowledge to aid its direction, and generally uninterpreted by the various administrative agencies and the courts.

What is the core of this dilemma? The 1970 Occupational Safety and Health Act mandates that employers provide safe and healthful workplaces for employees, while the EEOC implements

Title VII of the 1964 Civil Rights Act, the Equal Employment Opportunity Act, and Executive Order 11246, which prohibit discrimination against women in the workplace. In short, these anti-sex discrimination laws require that women are to be treated the same as men, in the absence of a bona fide occupational qualification which would provide an acceptable basis for differentiation between men and women in employment opportunities. These conflicting requirements exist notwithstanding the primary biological difference between men and women--that difference known as the female's reproductive capacity. But also not to be ignored are the possible hazards presented by industry to the genetic systems of both men and women.

How can industry effectively and fairly deal with this dilemma, while simultaneously complying with the mandates of both OSHA and the EEOC?

Law, the peacemaker between conflicting forces in our society, has traditionally applied balancing and policymaking tests to arrive at equitable decisions. In court determinations and reviews of administrative actions, the balancing test is better known as the "substantial evidence test," while the policymaking test is reflected in decisions which are determined by the goals, principles and equities inherent in our democratic way of life.

Speaking first to the policy test, there can be no basic argument between government agencies and industry as to the overall policies that industry should seek to provide safe and healthful work places for employees; that employees should not be subjected to scientifically proven hazards which could be injurious to the employee or to an unborn child; and that no employee should be subjected to illegal discrimination in employment.

On the other hand, the balancing or substantial evidence test will provide the practical guidelines and solutions to this complex governmental-industrial conflict.

What are some of the factors which must be considered in applying this balancing test?

First, as to OSHA, there is the requirement in Sec. 6(b) (5) of the OSHA statute that safety and health standards must be based upon the latest available scientific data. However, many courts have approved the position that some OSHA standards may be based primarily upon policy goals due to the mere threshold level of much scientific knowledge concerning certain hazards. The implication for OSHA is that massive personnel and funds must be allocated for research concerning hazards to women in the workplace to develop the valid and conclusive scientific data mandated by the Occupational Safety and Health Act.

Most OSHA exposure limits for toxic substances were not

established with consideration of protection for women and fetuses. Consequently, modification of some standards may be required during the interim period of research toward conclusive scientific data. However, the legal test to be applied to such modifications and/or new standards will continue to be the substantial evidence test. Therefore, OSHA will bear a heavy burden of proof to sustain new or modified standards for the protection of women and unborn children.

Moreover, the substantial evidence test should be applied so as to provide a reasonable approach to the dilemma of the employer and the employee during the interim research period. What is a reasonable approach?

While it is not reasonable or legal to prohibit all women, or non-pregnant women, regardless of age, from working in areas considered safe for male employees, it is reasonable to establish special standards and exposure levels which will protect women of childbearing capacity, pregnant women and unborn children from injury. Again, such standards must be based upon substantial scientific evidence that hazards to this special group do exist.

It is reasonable to require warning signs, documents and training concerning areas in the workplace which are known or suspected to be hazardous to pregnant women or fetuses, and to require labelling of scientifically proven teratogenic and abortifacient substances in the workplace.

It is reasonable to require employees assigned to, or already working in, areas which could be hazardous to pregnant women to carry the burden of informing the employer of the existence of childbearing capacity or a pregnancy. Only the female employee and her physician know whether she is pregnant. In all fairness to non-pregnant female employees, the pregnant female employee must carry this burden as the employer would invade the female employee's right of privacy by requiring periodic examinations to determine the existence of pregnancy among employees.

However, another problem exists as to this burden upon the female employee. The average period elapsing prior to conception and the confirmation that a pregnancy exists is generally four to five weeks. One writer has stated that the most dangerous period as concerns toxic substance injury to the fetus is during the first three months of pregnancy.* Thus, industry's motivation for prohibiting female employees of childbearing capacity from working in certain hazardous areas is based upon the premise that during the

*Phyllis Lehmann, "Women Workers: Are They Special?" Vol. 3 No. 4 Job Safety & Health Magazine, April, 1975. (U.S. Dept. of Labor, OSHA).

first four to five weeks of pregnancy, a female employee may not know that she is pregnant.

What is the reasonable solution in this instance?

Industry and the courts are likely to choose the more protective approach for certain toxic substances, and condone the prohibition against women of childbearing capacity working in certain areas where scientifically validated hazards for pregnant women have been proven to exist. But this solution should be limited to the hazard which has been proven by conclusive scientific evidence, and the female employee must carry the burden of proving that she lacks childbearing capacity.

Please take note that I use the term "childbearing capacity" as opposed to the term "women of childbearing age." The 1967 Age Discrimination Act prohibits employee discrimination based upon age and the age at which women cease to have childbearing capacity varies extensively. Therefore, there is no rational basis for a legal classification known as "women of childbearing age," while there may be a valid basis for the classification known as "women of childbearing capacity."

Now, turning to the EEOC, industry's feeling in this conflict was well stated by one industry official when he said "...we'd rather face the EEOC than a deformed baby."** Industry probably voices the same opinion concerning the matter of an aborted child.

At this juncture, it is important to recognize the difference in structure and operation of the EEOC and the OSHA. OSHA enforces specific standards which may deal with toxic substances, employee exposure limits, labelling, warning signs, employee education, physical examination, etc. The EEOC and other federal agencies enforce Title VII of the 1964 Civil Rights Act and Executive Order 11246 which prohibit, in very general terms, sex discrimination in employment. Rather than enforcing specific standards, the EEOC establishes sex discrimination by reliance upon allegations and proof of factual circumstances which form the legal basis for a finding of sex discrimination. Thus, the EEOC is dealing with composites of miscellaneous facts which may or may not establish sex discrimination, while OSHA is dealing with very specific standards.

In reconciling these opposites, it is encouraging to note that while EEOC litigation commences in Federal District Courts, it is subject to review in the same federal appellate courts which review decisions of the Occupational Safety and Health

**Statement by Dr. Norbert Roberts, Medical Director, Exxon Corporation, quoted by Phyllis Lehmann in "Women Workers: Are They Special?" (See Footnote *).

Review Commission. This future prospect of reconciliation should provide a motivation for reasonableness and lack of conflict in decisions by both the EEOC and OSHA.

How can the EEOC proceed reasonably in this dilemma?

It seems logical to conclude that in cases of illegal sex discrimination based upon health hazards to pregnant women, evidence concerning OSHA standards and regulations should be entered as substantive evidence in an EEOC proceeding. Of course, the substantial evidence test would again be employed to determine whether the alleged discrimination was defensible. In other words, if there is valid and conclusive scientific evidence that a hazard to pregnant women existed and that the employee barred from a job in the hazardous area was, in fact, pregnant or had child-bearing capacity, the alleged discrimination would appear to be defensible. If such was established, two ancillary issues would surface.

First, was the employee temporarily transferred to a non-hazardous job location rather than temporarily suspended? And secondly, if a transfer was effected, did it involve loss of pay or seniority?

As for the propriety of transfer as opposed to suspension, many court decisions have established the proposition that a pregnant employee is entitled to work as long as her physician deems it safe for her to do so. Therefore, a transfer from a non-hazardous job seems to be the more proper alternative. Of course, whether such a transfer can be effected depends upon whether at the time of the request for transfer, the employer has vacancies in any jobs in non-hazardous areas within the particular workplace. It is conceivable that in certain types of businesses, jobs non-hazardous to pregnant women or women of childbearing capacity simply do not exist.

As to retention of seniority, such agreements are generally governed by employer-union contracts. However, while retention of seniority is subject to union bargaining, equal pay pursuant to a temporary transfer presents a totally different problem. Such an adjustment would raise the issue of wage discrimination against permanent employees in the location to which the temporary transfer was made as well as nullify the employer's pre-established job-wage classifications. Such a result does not appear to be reasonable or legal.

An analogy to the employment of the handicapped is appropriate. Although the employer is expected to make job modifications in order to employ the handicapped, those modifications do not include pay for a job the employee is unable to perform due to a physical limitation. For the same reason, a pregnant employee should not receive pay which is higher than that allocated to the

assigned job.

In conclusion, it would appear appropriate for the EEOC and OSHA to come together and develop mutual guidelines for protection of pregnant women against occupational health hazards, and to attempt to define what circumstances in this respect do and do not constitute sex discrimination. It would be more sensible, safer and reasonable to have such joint guidelines developed, rather than to leave the matter totally to case-by-case decisions by the appellate courts.

Certainly, it cannot be argued that industry, OSHA, or the EEOC condone the risk of deformed or aborted children.

Let us come together.

MS. TREBILCOCK: I am an attorney for the United Automobile Workers in Detroit. I also deal with OSHA and equal opportunity matters among other legal problems connected with labor relations.

I would like to do two things this morning. First, I would like to discuss the problem that the UAW is facing now with General Motors in addressing a policy that the corporation has announced. It sounds, too, as if we are going to have some problems with Chrysler. Secondly, I would like to discuss some legal aspects that haven't been touched on entirely by the other two speakers.

Many of you have heard of a woman named Norma James, who is a worker in a battery plant in Oshawa, Ontario, Canada, where hourly employees are represented by the UAW. General Motors in Canada followed a corporate policy, which they have announced for this country, but which they have not yet put into effect here. This policy is that there should be no woman of childbearing capacity on a lead-exposure job. Norma James was told that she could no longer keep the job that she had in the battery plant, and, rather than become unemployed, she had herself sterilized.

We have come to a sad state in this world when a woman must choose between her childbearing capacity and her job.

In the United States, General Motors, to our knowledge, has not yet forced the transfer of women from lead-exposure jobs. They have threatened to do this. They have also stated in legal documents in a lawsuit that they do, indeed, have a policy of not hiring women of childbearing capacity in lead-exposure jobs at their battery plant in Muncie, Indiana.

Well, the UAW obviously had to respond to this policy. What was that response? The Director of the General Motors Department, Irving Bluestone, who is also a Vice President of our union, has

instructed the local unions to grieve this violation of rights, and to demand restoration of a woman's job if the corporation attempts to transfer her in violation of the collective bargaining agreement. He has further instructed that if that transfer is for some reason held not to violate her rights under the grievance and arbitration procedure, she should still get full seniority protections under the contract.

Another Vice President of the UAW, Odessa Komer, also faces the lead problem. She has under her jurisdiction four battery plants, as well as some other manufacturing plants, which involve lead exposure to women employees. Vice President Komer sees the lead issue as the tip of the iceberg. The door for women was finally being opened. Women were finally getting access thanks to the Civil Rights laws, thanks to the pressure from the unions, and thanks to the pressure from women themselves. They were finally getting access to higher paid, better jobs. Now they are threatened with the loss of those job rights and it is a serious threat indeed. Obviously, as you have heard earlier this week, lead is not the only problem.

Vice President Komer has been instrumental in alerting our union to the problem and its implications for women's job rights. Her position on the issue is this: She feels the lead question is being used to discriminate against women in their job rights. As she says, the mother of the fetus needs to eat.

She also feels that the answer is fairly obvious. The plants should be cleaned up in a way to protect everyone's health. That health includes the reproductive health of both male and female workers. A worker should not have to choose between having a job and having a child.

We all know, however, that that goal cannot be achieved immediately. In the meantime then, Vice President Komer urges that each employee be informed of the dangers to his or her reproductive capacity. This burden of information falls primarily on the employer, but whenever we in the union can assist in it, we should.

Once the employee is informed, he or she should be free to make a voluntary temporary transfer to a job without the lead exposure problem. That transfer must not penalize the workers in any way. It must be at the same rate of pay. It must be with the same fringe benefits and other contractual protections that the worker had at the former permanent job. And, finally, the worker must have complete rights to return to the former job once his or her child is born.

This solution comes the closest to any that I have seen of meeting all the legal and other concerns posed by this problem. And I would like to say that I think that the UAW owes a great debt to Odessa Komer. She has been very helpful in helping the union

clarify its thinking on this question.

At this point, we need to step back and ask why we face this problem at all? I believe there are two primary reasons. The first is that the employers are not meeting their legal and moral obligations to provide a safe workplace for all employees, because they think it is going to cost them too much money.

We have heard talk of economic feasibility. However, in the original OSHA legislation in the Federal code, there was no mention of the word "economic feasibility." Employers have pushed this in the courts and unfortunately the courts have adopted it.

The companies would rather try to find a superworker who can withstand high exposure levels to various chemicals. Obviously, pregnant women are not going to be included in this group. Employers are seeking legal standards which will fit their aim of reducing costs for another reason. The employers fear potential lawsuits by women whose reproductive capacity is harmed on their jobs, and to the extent that those suits can get around workers' compensation statutory bars, the employers may have something to worry about.

They are also worried about suits from a child who has an independent right to sue, which cannot be waived by the mother. This could occur in the case of a child who is damaged as a fetus because of the employer's unhealthful working conditions. So the employers are worried about money.

There is a second reason they are trying to pursue this and it also relates to money. Sex discrimination is a handy tool for saving money. Over the years sex discrimination has saved companies millions of dollars, and a company's policy toward pregnancy is a fundamental mechanism in the operation of sex discrimination.

The EEOC guidelines clearly understand and reflect this in the way they are written: a policy which discriminates on the basis of pregnancy is illegal sex discrimination. It is interesting to note, too, that women's role as a potential childbearer was also an important underpinning behind the state protective legislation. For example, there was a Michigan law that prohibited a woman from working in any job that endangered her capacity for motherhood. The idea behind it was, of course, basically good. But protective legislation should have been extended to men. Instead, it was used to severely limit women's job opportunities and earning capacity.

The lead issue is raising the same specter. We believe that it will be used in the same way protective laws were used in the past. For that reason, we are highly suspicious of the whole framing of this issue.

Now that the government may be considering taking some sort of action on the lead standard, we would like to remind the government that it should go back to basics. The government has a legislative and a constitutional responsibility to prevent a discriminatory policy from being adopted and tolerated.

Let's take a hypothetical situation. Suppose the Occupational Safety and Health Administration issues a lead standard that includes a phrase that women of childbearing age shall not be allowed in lead exposure areas. Would they also issue such a standard for black people due to the sickle cell anemia problem? I do not believe that they would.

If OSHA issued such a regulation barring all fertile women from lead exposure jobs, I believe that they would walk smack into two important decisions of the United States Supreme Court, which are commonly known as Cohen and LaFleur.

These decisions are important because they say that the state cannot make presumptions about women as a group and base action against an individual on those presumptions. So if OSHA were to enunciate a standard of that kind, they would be presuming that all women who are fertile would in fact become pregnant and carry a child to term. I believe that this would be an unconstitutional presumption.

OSHA is also required to follow its own mandate under the law which created it, and that mandate includes several things. OSHA is supposed to set standards which most adequately assure, to the extent feasible on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity.

The purpose of the statute, indeed, is to assure as far as possible every working man and woman in the nation safe and healthful working conditions. Another purpose is to preserve our human resources.

Finally, there is an obligation under OSHA to use medical criteria which will assure, insofar as practicable, that no employee will suffer diminished health, function capacity, or life expectancy as a result of his work experience.

In addition, of course, there is the general duty clause which Ms. Hamlar already discussed with you.

The ability of a woman to bear a healthy child is a function, in part, of her health as well as the question involving the health of the infant. The reproductive capacity of a woman and the physical fitness necessary to carry a baby full term and bear a normal child is not something the employer should be able to take from her at the cost of her earning a living.

A workplace which causes such extreme reactions as genetic mutation, or which creates toxicity, or which affects the fetus by exposure through the mother's circulatory system or skin, is not a safe workplace for that woman.

Therefore, I think under the Occupational Safety and Health Act itself, there is a mandate for the Federal Government to set standards that will protect all workers and to enforce the law against employers in a way that they will have to provide a safe workplace for all workers.

This is also consistent, I believe, with employers' obligations under Title VII of the Civil Rights Act.

Mr. Robertson has already given you a very thorough and clear explanation of the way in which this problem would be analyzed under that law. I would add only one thought and that is that I do not think it is outrageous to say that at the moment employers are violating Title VII by maintaining workplaces which have a disparate impact on the health of women and on their reproductive capacity. So right now we even may have a violation of that statute through the employer's refusal to set a standard and to maintain its facilities at a level that protects women as well as men.

As a union attorney, of course I am concerned with union liability, and it is clear that unions also face serious liability under Title VII if they either agree to an employer's policy that discriminates against women illegally, or if they even acquiesce in an employer's discriminatory policy against women. So the union has an obligation, which I think most of the unions are meeting, to ensure that the employer is not discriminating on the basis of sex in its health and safety policy.

Since unions do not control the workplace, I don't believe that the unions share liability in the personal injury area. Where you have an employer who transfers a woman involuntarily, that employer is probably doing so in violation of the collective bargaining agreement in several ways.

One, there is usually a transfer policy in the agreement. The employer may not have followed that. Two, there are seniority protections in the agreement and if the transfer was not done in accordance with those seniority protections, the employer is violating it.

We also have antidiscrimination clauses in our agreement. So if the employer is transferring in a way that is sex discriminatory, that avenue can be used as well to pursue rights of employees.

Section 301 of the Labor Management Relations Act gives unions the right to enforce these contract provisions in Federal

court. We also, of course, have the grievance and arbitration procedure.

Finally, we have a very important right under UAW contracts and that is the right to strike in the middle of the contract's term over health and safety conditions.

One last point is that the union owes a duty of fair representation to its employees. Part of that duty is to not discriminate against employees on the basis of sex. Indeed, unions do not wish to do this. On this basis, also, a union should avoid being hoodwinked by an employer into agreeing to an unsound position concerning the possibility of an individual suit against the employer for failing to maintain healthy and safe working conditions. The reason for this is that under OSHA the labor union does not control the workplace. Management functions include the decisions that will determine whether or not the workplace is safe.

There is another aspect to this problem, and that is its relationship to the labor relations acts and practices which are in effect in this country. An employer who unilaterally transfers a fertile woman against her wishes, and against the past practices in the factory, may be taking illegal action. Indeed, I think he is taking illegal action.

Unilateral action taken by an employer as to the conditions of employment is a violation of the National Labor Relations Act. The section of that Act is 8(a)(5), which indicates it is a kind of refusal to bargain with the union over essential job conditions. The employer must first bargain to impasse with the union, and must thoroughly discuss the problem and bargain in good faith with the union concerning mandatory subjects.

There is something beyond this as well, and that is the power of unions to negotiate collective bargaining agreements. That is the guts of their existence. Collective bargaining agreements contain rights which protect employees.

And finally, in this issue, as in all issues, the union must step back and ask itself why the employer is taking this action? Why is the employer propounding this position? Is the employer really concerned about fetuses? Is the employer really concerned about children? Is the employer really concerned about the health of women workers, and the health of male workers?

If the employer is truly concerned with these more than he is concerned with profits, he will clean up his plant and resolve the problem. He will do this instead of using the lead issue as an excuse to perpetuate illegal discrimination against women. Instead, he will clean up his premises. Only in this way can all the legal mandates of the Labor Relations Acts of this country, including Title VII and OSHA, be met.

We must join together to fight those who seek to attack women's job rights, and who seek to weaken our contract rights and our rights under both the Occupational Safety and Health Act and Title VII.

QUESTIONS, ANSWERS, COMMENTS

MR. JEFFREY BERGER: I work for OSHA in the Solicitor's office. I would first like to point out to you that there are a lot of people from OSHA here and we all came because we are interested in safety and health. We try to create safe and healthy working conditions, but you have to realize that we are exceedingly understaffed and underfunded.

We deal with an enormous bureaucracy. We are now dealing with a Federal Government program to cut back our standards. I heard a number of people say employers are only interested in costs. Well, that is true. Employers are very much interested in costs, but I would say that unions, too, are very much interested in costs. And I would suggest that perhaps union negotiations could begin to take the costs of employee safety and health into account just as they take wages into account.

What I am trying to suggest is that this is a massive problem which is going to have to be dealt with in three different ways by employers, unions and the government, and the thing I keep coming back to is the feasibility question.

Now, I disagree with the statement that OSHA does not have a mandate to take economic and technological feasibility into account in these standards, because there is legislative history which supports such a mandate. There are several decisions of the Court of Appeals which give us this mandate. The situation comes down to something like this: Suppose you have a substance to which a man could be exposed safely at 100 ppm over an eight-hour time-weighted average period, and you have a woman who can only be exposed to it safely for five ppm over an eight-hour period. Now, an employer is going to say, "Well, I could have men work here, but if I have women work here it is going to be economically infeasible." We would deal with that. I deal with these cases on a trial level where we have to prove that an employer is not giving us an accurate picture of his workplace.

Now, if he says to you, "I am going to have to shut down if I have to put women on this job," how do you deal with that? Would you rather see a job shut down and no one working rather than just have men working? I think the suggestions that transfer be made to keep seniority and wage rates at parity is a good one.

All I am trying to suggest is that economic and technologic feasibility are important considerations which have to be taken

into account; that OSHA is so exceedingly underfunded and understaffed that we should not and cannot handle all the responsibility that has been placed on us; and that union responsibility has to be taken into account here, as well as the responsibility of the employer.

MR. ROBERTSON: The question is what do you do when the government doesn't have enough money to do the job. In outlining Title VII, I failed to mention that even though EEOC now has enforcement power, the original power under Title VII included the power of the individual charging party to go into court and sue. That power is still there. So that if the OSHA and EEOC enforcement program seems to operate to discriminate against women, the victim of that discrimination has the right to file her administrative complaint and then go into court, if it is not dealt with in 180 days.

MS. TREBILCOCK: Just for clarification, the phrase "economic feasibility" is not in the statute. I did say that it is incorporated in court decisions.

As to collective bargaining, obviously it is not in the unions' interests for the employer to go out of business. That is a basic fundamental thought that puts a constraint on bargaining. However, we have companies pleading poverty all the time and we have a right under the National Labor Relations Act to get economic data which verifies those claims. We are just a little suspicious because many companies that have pleaded poverty suddenly aren't pleading poverty any more when you say you are going to look at their books, or they turn their books over and you can show that it is, in fact, a false claim. So we are extremely suspicious in that regard.

MR. JAMES ROBINSON: I am from OSHA, but I don't claim to represent OSHA; I am speaking as a citizen. My question is addressed to Portia Hamlar and it is basically a request to respond to any number of issues, which have been raised, but which I thought your speech did not adequately regard. For example, the LaFleur and Cohen decisions, and the Grigg's business necessity definition. But even beyond that, look at the Rowe and Doe Supreme Court decisions on abortion. If the Supreme Court states that there is not a sufficient interest on the part of the state to get involved during most of the first and second trimester, how can the corporation possibly have a more compelling interest than the state when the fetus doesn't even exist?

In other words, you have a Supreme Court case which speaks to the existence of a fetus, and the court comes down and says that the woman has a certain basic decision. By extension, you are saying that Chrysler Corporation, in the absence of a fetus not existing, has an even more compelling interest and will make a decision that may affect the woman's health and economic security.

Beyond that, if you look at the whole fight for equal credit, you will find that the bankers came in and said, "Well, how can we possibly extend credit to a single woman or a divorced woman or, in many cases, how can we count the income of a woman because, obviously, the first thing she is going to do is go out and get pregnant and then she will default." That was a pretty good argument except Congress, in effect, reversed that when it enacted amendments to the Fair Credit Reporting Standards Acts of 1974, which went into effect last October. These amendments say that banks can no longer take reproductive possibilities into account, that they have to give people some credit for being rational to the point of understanding that they can get pregnant, but that they have to pay it back.

And my final point is, while I welcome Chrysler's concern that working may be unhealthy for the mother, how about the fact that not working may be equally unhealthy? I mean we know about incidences of alcoholism and women on tranquilizers. NIMH has just finished a massive study which says when unemployment goes up, people commit suicide more often, are more depressed, and unhappy. I think we have to focus on this side of the equation. If people can't eat, if people aren't going to be happy, they are going to have other problems. Under a temporary disability collective bargaining agreement, it seems that it is Chrysler's obligation to find a job, and with X-tens of thousands of employees, it just baffles my mind why you can't find something useful for somebody to do for a few months. If you can't, I would be happy to come on and be a consultant to you because I am convinced I could find something.

MS. HAMLAR: Your comments reemphasize the conflict which I noticed when I began my comments. I would like to reemphasize a couple of points that I made. I stressed the right of privacy, which the female has. That, of course, comes into play when she decides whether or not to disclose that she is pregnant, and whether or not to disclose that she has childbearing capacity. So she does have the right to make those decisions.

But getting to the other side of the coin, transferring out of certain job locations, I said at the beginning that industry is faced with governmental regulations that conflict. OSHA is saying on one side, "You provide a safe and healthful workplace." EEOC is saying, "You don't transfer in a discriminate manner."

Now, those two objectives are nowhere near reconciliation, but industry is trying to approach them in a sensible manner. We happen to feel that the health of the employee and the unborn child should carry a higher priority than someone retaining a job.

I am well aware of all the problems that unemployment brings about, and I stress to you that that is a consideration which we face when we make these decisions, but it is a matter of where we

place our priority. I doubt that many people in this room would disagree with that priority, but I close my remarks by saying that the courts will end up balancing the possible unemployment against the health risk.

MR. PAUL STERN: I am a practicing industrial hygienist at the National Institutes of Health, and I am also a mechanical engineer.

I heard some talk when I was down in Atlanta in the middle of May at the AIHA conference about third party lawsuits. In other words, if I don't do my job, I can be sued as an individual, or the medical director where I work can be sued, or any other individual involved, as well as an agency or a company. This is what they call, I believe, a third party lawsuit. Can you tell me anything about it?

MR. ROBERTSON: I can say that there have been a number of such lawsuits brought against government officials with varying degrees of success, as is often true with lawsuits. In the civil rights field, there have been two or three that have been successful. There was a suit against the Secretary of HEW on the grounds that he was not vigorously enough enforcing certain civil rights responsibilities.

There was a suit brought by the Legal Aid Society of Alameda County against one of the contract compliance programs, and court orders have been issued in these suits directing government officials to do certain acts that they were not otherwise doing.

MS. TREBILCOCK: Besides the government as a defendant, there are some other potential defendants. Normally, the workers' compensation law in a state prevents a worker from suing the employer for injuries received in the workplace because the workers' compensation award is supposed to be the only remedy.

Therefore, some people have tried to think of creative alternatives. Some asbestos workers who suffered very serious health effects, including death, in Texas, sued the supplier of the asbestos. In other words, they sued the company that provided the asbestos material to the employer on the theory that it was an inherently dangerous substance and that there was a duty to warn the employees about that hazard.

There have also been suits filed against some doctors for medical malpractice on the similar theory that the doctor knew ten years ago that the worker was being exposed to a substance which was damaging his or her health, and that the doctor did not act as a normal physician would in exercising his duty to care and to warn the worker.

There also have been a couple of suits against unions for failing to inspect a mine in which there was a disaster. Those

have not been successful because the only duty that the union has to its members is the duty of fair representation. The legal standard is very different under that duty than it is under regular personal injury actions.

DR. TERSHEN: I am with the Institute of Policy Studies. Many of you received a statement distributed during the coffee break by the Women's Political Action Caucus, Occupational Health. Our statement is addressed to the conference organizers and participants. We recognize and wish to thank the organizers for the efforts they made to take account of some of our concerns, but we would like to reiterate those concerns once more.

We pointed out the failure of the conference scientists to address the full range of women's occupational health problems by focusing too narrowly on women's reproductive function; by speaking technically to other technicians rather than explaining hazards to women in the audience; by absenting themselves from the women workers' evening discussion; by ignoring women workers' lack of options in their oppressed situation; and by placing blame and responsibility on these women. Ann Zablonksi, who is standing next to me, has some concrete comments and suggestions to make.

MS. ZABLONSKI: The Women's Political Action Occupational Health Caucus would like to formally thank Vicky Bor and Sandy Zimmerman for their help in getting our statement typed and Xeroxed. At this time we are asking the conference organizers for a formal commitment to publish our statement in the conference proceedings.

DR. BINGHAM: It was recorded. It will be part of the proceedings.

MR. ERIC FRUMIN: I am from the Amalgamated Clothing and Textile Workers Union. Anne, could you give a few examples of the powers a union has to fight illegal transfers? You mentioned your right to strike under your contracts over issues of health and safety. There was another that you mentioned which I didn't catch.

MS. TREBILCOCK: What I was referring to were provisions in the contract that govern transfer procedures. There is also Section 8(a) (5) of the National Labor Relations Act, which makes it unlawful for an employer to unilaterally change a condition of employment that is the subject of mandatory collective bargaining without first bargaining to impasse with the union.

MR. MATT AMBERG: I am with the International Union of Electrical, Radio and Machine Workers, which is just about entirely manufacturing in electrical and electronics. We are one-third women.

I must confess that I am in a perverse sort of way grateful to the gentleman from the Solicitor's Office of the Labor Department for the remarks he made, because I think nowhere else have we had so bold and brazen a statement to the effect that we have to

trade with management the pay increases that our people need for their occupational health and safety.

He told us that those are cost items that we should be negotiating and I can just see us asking in our next go-around, "Well, now what is it going to cost to make this plant safe, and how much of this has got to come out of our pay envelopes?"

You know, management has always insisted on its prerogatives, and those have included how the plant is set up and how the processes work. Management has set the standards. They have set the production rates. They have set everything except what we have been able to win a hand in regulating.

And now all of a sudden we are being asked to share the economic burden of correcting hazardous conditions by trading off necessary wage gains. I have been nurturing the suspicion for a long time that the Office of the Solicitor has probably been the greatest stumbling block of all in the implementation of the 1970 Occupational Safety and Health Act. I tried to tell the CLUW women's caucus the other night how long it takes between the time that NIOSH sends over a criteria package to the Occupational Safety and Health Administration and the time OSHA formulates a proposed standard. And then, at least in some cases, those standards are kicked to pieces in the Solicitor's Office. For example, take benzene, which is a killer substance. There was a standard that was formulated by OSHA and sent to the Solicitor's Office in 1974, and when I called the other day to find out just what is the present status of that standard, I was told, "Well, we are drafting it."

And I know what the Solicitor's Office will say. They will say that "Unless we have an absolutely foolproof standard, it won't fly in court." Well, nobody is asking for slovenly standards, but nobody either ought to be asking for 100% absolute perfection while people are dying.

Let me go further. I am not a lawyer. But I get the impression that there have been court decisions indicating that where there are urgent questions of safety and health, you don't have to have absolute 1,000% proof.

I wish OSHA would get about the business of issuing standards and enforcing them. Now, we have been saying there are not enough bodies to do the job, and I agree with Mr. Berger in that regard, but for Pete's sake don't use those few bodies to blockade us.

They have taken forty bodies from the OSHA standard-setting people, and sent them out in the country to find out from industry how to weaken, water down and eliminate standards. Of course, when they finally find out how to cut down on standards, then they are going to spend all their time in the Solicitor's Office cutting

down standards instead of tightening up and implementing more standards.

So the emphasis they have is all wrong. And, finally, I do want to agree with the UAW lawyer. I, too, have been able to find the word "economic" in the law in connection with feasibility. "It isn't feasible to have a safe place. It costs money. Why spend money? We can get more people in and hurt them and send them out or kill them and send them out. Why spend money?"

Incidentally, on third party suits, let me suggest that the machine builders are scared of being sued when hands get chopped off to the extent that we now have them as allies in the question of the no hands and dyes provision which we are fighting to get back into the standards after the OSHA cut it out.

MS. TREBILCOCK: I would like to thank the brother from IUE for saying things I wish I had said. I think he made a real important point that we shouldn't have to bargain for rights we are supposed to have under the law, and that employers have a legal obligation to make the workplace safe.

That also raises the interesting question of the relationship between workers who are not unionized and workers who are unionized. The position that the gentleman from the Solicitor's Office was urging would suggest that workers who have unions have less rights under the Federal law than workers who don't have unions, because workers who don't have unions can't bargain. It is a pretty ridiculous position.

Incidentally, I thought of an example for Eric Frumin. The Steelworkers had a situation in Idaho in which the employer acted arbitrarily and took women off of lead-exposure jobs in a lead smelter. The Steelworkers have pursued the grievance procedure and have fought the company tooth and nail. There have also been some Title VII charges filed against the company because of that incident.

MS. JEANNE REILLY: I am from the Professional and Technical Engineers Union. I would like to ask the industry lawyer what is the basis for the corporate policy decision that says the American worker is an expendable asset in this country, and that I, as a woman of childbearing age, must bear the burden of unemployment? When I go to my friendly obstetrician, his first question is what is my medical coverage. When I go to my friendly industrial relations agent and ask him if I may have a disability leave, he says, "Good God, woman, you are not disabled, you are pregnant."

So what is the basis of the corporate policy decision that says the American worker must work in an uncontrolled environment, while the computer used to store the data about what the American worker is producing is given a quality controlled environment?

What is the corporate policy that says hardware is an asset, but that the worker is expendable?

MS. HAMLAR: In responding to that last comment, I would just like to say that I don't believe industry holds the position that the worker is expendable. You may look at it that way because of the problems which we are focusing upon, but, in fact, industry could not operate without workers. We all know that.

I think that we are going to have to be a little more realistic in assessing this problem. I feel that the day is coming when the pregnant employee will be considered a temporarily disabled employee and possibly will be able to recoup the insurance benefits that go along with that classification. We are not at that stage yet, but I think it is coming.

Industry essentially looks at a pregnant employee as being temporarily disabled. There are certain jobs that employees cannot safely do. But that is not a special situation. There are other employees with physical problems due to accidents or temporary illnesses, and they are also temporarily disabled. They are also transferred out of certain jobs temporarily. So the pregnant woman is not unique in that.

I think you have to look at the problem realistically and say, "Well, we are not going to place a woman in an unsafe position." Industry is forbidden to do that by the Rehabilitation Act. When we hire people who are permanently disabled, we still have to put them in a safe job. This is all we are trying to do for the pregnant woman. As for the degree of expertise that goes into quality control of the hardware in a plant, that is not a particularly good analogy because you are trying to compare machinery with people, which, I think, is very difficult.

MS. KATHY HUNNINEN: Just for the record I want to state that I am speaking for myself and not for Tennessee OSHA. What should be mentioned is there are Small Business Administration loans that are available and it is my impression that small businesses are not making use of these low interest loans as much as they probably could.

As an industrial hygienist, I know that controls are available and I think that economic feasibility should not be an issue in setting standards.

I want to thank Dr. Bingham and Clara Schiffer for producing this meeting, and I want especially to tell Dr. Bingham, since I studied under her, that I appreciate the efforts she has made, because I feel very strongly that women need to be in leadership positions and need to be active in the field of occupational health where these standards and all these policies are being made.

CLOSING REMARKS

Dr. Eula Bingham

One of the said purposes of the Society for Occupational and Environmental Health is to provide a public forum in which we can have industry, labor, government and academia discuss occupational health and environmental health problems.

I think we have seen many sides of the question here. We have not settled anything. We have probably raised many more questions than we will have answers to for many years to come.

I think we have only begun to discuss the issues. As you can see, there are people who want to continue on. There was some thought about starting this meeting on a Monday and going through until Friday. Yet we had people who thought we didn't have enough to talk about. Well, it is obvious we would have had enough to talk about. The only reason we didn't start on Monday is that we couldn't find hotel space, and we thought that the first conference of this type should be held in the Washington area.

I would like to say that I think we cannot deny from what we have seen and heard here that many of the occupational health problems we have discussed are entangled with discrimination. That message has come across loud and clear to me. I hope the appropriate people in government have heard that message. I think they have not heard the last of it.

While it is very useful to come to a conference such as this and discuss our problems and see where we fit together with common goals, what are we really going to accomplish with this discussion? Well, we are going to meet people who can work with us on problems, and who can aid us in achieving our goals.

We have laid the issues out. Perhaps this is a preview of what the hearings on the lead standard are going to be. I would like to make one more comment to the women in the audience. When I look at how we are going to influence things in occupational health, particularly issues that relate to women, I become rather discouraged because I think that if we look around us, we find very few women in places where policy decisions are being made. We have seen one or two from industry. We have seen several more from the unions. But I see very few women, in fact, I don't see any women, in OSHA or in the Department of Labor, who are in what I would call policy-making positions. I see no women in the high echelons of NIOSH, who make policy and who guide what will be done.

So how can women help influence policy? I believe that the best way is perhaps through the legal process and through the press. The press is very powerful. I would also like to point out what I think is the influential position of individuals such

as Peggy Taylor and Sue Nelson, who work with Congressmen, and who really have an opportunity to influence the legislature in coming up with solutions to problems.

I would like to thank all of you for coming here, and for your comments and your criticisms. We could not have had a conference without audience participation. I am sorry we did not have time for more, but it would probably have taken another two or three days, maybe even another couple of weeks. Perhaps we can have another conference on this topic in the future.

PRESENTATION OF THE SECOND AWARD OF
THE SOCIETY FOR OCCUPATIONAL AND
ENVIRONMENTAL HEALTH TO
DR. HARRIET L. HARDY

by

Dr. Joseph K. Wagoner
President-elect of the Society

Dr. Hardy, it is my pleasure and privilege to bring to you the congratulations of this Conference on Women and the Workplace, which has been organized by the Society for Occupational and Environmental Health. This Conference which is sponsored by the National Institute for Occupational Safety and Health and the National Foundation March of Dimes, and which is co-sponsored by the Industrial Union Department of the AFL-CIO, the Coalition of Labor Union Women, the D.C. Lung Association and the United Church Board for Homeland Ministries, has attracted approximately 500 participants with broad representation from academia, government, labor, industry and the general public.

The Society for Occupational and Environmental Health considered that on this occasion it is highly suitable for us to express to you our profound respects in recognition for your pioneering and sustained efforts toward the identification and eradication of occupational diseases. As President-elect of the Society, I am indeed privileged to convey these sentiments and to present to you our Second Award. Before doing so, however, it is only appropriate to attempt the challenging task of enumerating for our audience some of your innovative contributions to humanity and to science.

A native of Massachusetts, Harriet L. Hardy was graduated from Cornell Medical College with an M.D. degree in 1932. As a Graduate Assistant in Medicine at Massachusetts General Hospital, Dr. Hardy launched her distinguished career in clinical preventive medicine. For more than a decade, Dr. Hardy directed the Occupational Disease Clinic at Massachusetts General Hospital, an innovative program in American medicine. After World War II, Dr. Hardy spent a year as Health Division Group Leader at the Los Alamos Scientific Laboratory of the Atomic Energy Commission, studying the hazards of radiation and the necessary protective methods. From 1947 to 1952, she was an instructor of industrial hygiene at the Harvard School of Public Health. From 1949 to her retirement in 1971, she was a consultant and assistant medical director in charge of occupational medical service at the Massachusetts Institute of Technology, and from 1955 to her retirement, she was on the staff of the Harvard Medical School, where she was one of the first women in that school's history to hold a full professorship. Following her retirement from the Harvard Medical School and MIT in 1971, she accepted the posts of Visiting Professor of Medicine at Dartmouth Medical School and Adjunct Professor of Environmental Studies at Dartmouth College.

Dr. Hardy's early research work dealt with anemia in college women. This work led to a broadening of medical consultation services for students at Radcliffe College. In 1946 Dr. Hardy's investigations identified a chronic respiratory disease caused by the beryllium used in the manufacture of fluorescent lamps. Through her efforts, the Beryllium Case Registry was established, thus creating a basis for the evaluation of the scope and clinical course of beryllium-induced respiratory disease. As co-founder of the Isotope Committee at Massachusetts General Hospital, Dr. Hardy was responsible for instituting a program for those exposed to nuclear radiation.

Dr. Hardy has numerous scientific publications and she has made major contributions to books including *Dangerous Occupations* in *Encyclopedia Britannica*. In 1949, she with the late Dr. Alice Hamilton co-authored the classic textbook "*Industrial Toxicology*" now in its third edition.

Dr. Hardy's awards include the Medical Woman of the Year Award in 1955, and the Business and Professional Woman of the Year Award in 1963. In 1971, Dr. Hardy received the Alice Hamilton Award of the New York Academy of Sciences. In that same year she also received the Award of Merit of the American Academy of Occupational Medicine. In the following year, Dr. Hardy received the Cornell Medical College Distinguished Alumni Award. In 1974, Dr. Hardy received the Browning Award of the American Public Health Association for her outstanding contributions in the prevention of disease.

The Society for Occupational and Environmental Health has selected Dr. Hardy as its recipient for its Second Award. The award is a scroll inscribed as follows:

For Keeping the light of Alice Hamilton,
for her contributions to American Medicine
as a scientist and teacher, for her
integrity and devotion to easing the
plight of diseased workers, the Society
for Occupational and Environmental
Health makes its Second Award to
Harriet L. Hardy.

On behalf of the Society I would like to present Dr. Hardy an honorary membership in the Society for Occupational and Environmental Health.

ACCEPTANCE BY DR. HARRIET L. HARDY

Dr. Wagoner, Members of the Society for Occupational and Environmental Health, and Guests: Being pretty much on the sidelines of my profession, these days, I am touched to receive this award of the Society for Occupational and Environmental Health.

A few days ago my suspicion of my antiquity was confirmed by a medical student listening to me tell of my training in the 1930's. She exclaimed, "You must have been one of the first women to study medicine." She had always lived in Boston, her men folk went to Harvard, she had gone to Harvard Medical School, where women were first admitted in 1946 to fill empty places left by men who had left to fight in World War II.

In search for the history of women in medicine, stirred by the student's remark, and recalling from personal experience that medicine is an exhausting and hazardous occupation, I went to my bookshelves. By means of review of two comprehensive histories of medicine, I discovered that womens' work in the healing field is recorded ever so lightly. In one of the tales from the Arabian Nights, an accomplished slave girl recites the foundation of Mohammedan medicine. This is thought to have originated in the Tenth Century from Indian and Persian sources. During all those centuries, no mention is made of women in medicine again until the Crimean War in 1854, when Florence Nightingale, by making a war-time hospital clean and aseptic, lowered the death rate from 40% to 2% in six months. In the late 19th century, Elizabeth Blackwell, English born, was trained in medicine at Geneva, New York College. She graduated in 1849. She founded the New York Infirmary and College for Women in New York City in 1857, where I studied, in 1929, while at Cornell Medical College. In 1895, Dr. Blackwell's book appeared with the descriptive title, "Pioneer Work in Opening the Medical Profession to Women." In 1850, the Womens' Medical College of Philadelphia had opened for the education of women as nurses and physicians largely for the missionary field. Both schools continue to admit students of both sexes. Throughout this period the record tells of great effort to improve nursing care and hospital standards which interestingly correlate with pressures to gain the vote for all women and men in the United States. New to me is the fact that freed black men, not women, white or black, were given the vote in 1870, and both sexes in 1918, 1928, 1972, these various dates marking change in the age level at which a citizen was allowed to vote. Through the period since Dr. Blackwell's book appeared, medical schools, especially Land Grant universities, have admitted a small number of women. In fairness to our less chauvinistic male colleagues it is true that a relatively small number applied, a comment on the social position of women before World War I.

This capsulated history and the current feverish progress toward equality of the sexes in our economic, professional and

social hierarchies strike me as similar in character-- the time a century later. Much of the content of the present stresses are, as before, correlated with wars and threats of wars. As an optimist, living in a world oversupplied with prophets of doom, my solution is more and more education for many more people. Did you know that there is evidence that United States labor unions of the mid-19th century were responsible for demanding and getting public schools open to everyone?

Let me take advantage of my great age and, until recently, inferior sex, to report briefly some of my adventures. Be assured I know that long talks, after a day of hearing good papers, enjoying a banquet with a liquid introduction, are "de trop."

Twice, in medical school, I very nearly betrayed the fact that I belonged to the weaker sex. One evening I was alone in a New York City tenement with a woman in labor and a husband of Latin origin. Though I did not understand his language, he made me understand his, by showing me a business-like knife to make clear that the baby, rapidly arriving, was to be a son! As I stand before you, you will know the bambino was a boy! The second time that I was pale and with a racing pulse was while attending my first autopsy with a male colleague, who had asked me to take lunch with him. I doubt he ever entertained for less money. I drank half a glass of water!

To make the point that training for medicine has been a dangerous trade, the fact is that in the 1930's, of my class of 35 residents in a big city hospital, five never used their training fully, four developed pulmonary tuberculosis, and one developed chronic bleeding ulcers --a significant loss in view of the length of training and the relative shortage of doctors. My own near fatal risk, my fault, was being jumped from behind by a maniacal epileptic -- my fault because I had gone to the locked ward without taking an orderly to guard against such assaults.

Describing briefly the steps that brought me, a clinically oriented physician, to occupational medicine, I began practice during the Great Depression in a farming town of 1500. In this town was a large private school where I earned my bread and butter caring for normal adolescents. My village work, 50¢ for an office call, \$1 for a house call, \$2 on Sunday, brought priceless medical experience plus rich training in the humane side of medicine. As the first woman doctor in this New England County from a then-called Grade A medical school, I had humorous and near tragic experiences. Of the former I laugh when I recall the woman with pneumonia I was trying to cheer by telling her of a party I was going to attend. Between painful breaths she said, "Dr. Hardy, don't be worldly." Of the tragic, I remember the call to a farmhouse in the early hours on a cold winter day when I had to get the ice out of the brake lining before the car would start. The patient's family was foreign born, obviously thought I was too

young, probably driving the Doctor's car. At last I saw the four-month old patient, desperately ill with pneumonia in the days without antibiotics. The family reported that the onion poultice was ready and showed me a smelly pan of fried onions. I quickly wrapped the little patient in blankets and drove the 15 miles to the nearest hospital, where she died the next day. The family assured me the onion plaster would have saved her.

With this background plus the knowledge of the variations of the so-called normal age of 13 to 18, I moved to five years at Radcliffe College, now joined with Harvard, studying and caring for healthy young adults, age 18 to 30. I was forced to leave my beloved country practice because of the great occupational hazard of nurses and doctors--excess fatigue. One was only off duty when out of town. Because I alone in the county was trained to fit contraceptives, write pediatric formulae, willing to go to court for the Society for Prevention of Cruelty to Children in incest cases, I was never idle.

During my years at Radcliffe, I came to know Boston medicine and started work in the Massachusetts General Hospital Medical Clinic. Now I had time to think a bit, develop projects at Radcliffe to study questions such as why iron deficiency occurs only spring and fall, and why there were so many diagnoses of low blood pressure. My studies during this period brought me to the conclusion that medicine knows too little of the onset of disease. We might, with more knowledge, intercept and control the development of disease. An easy step was to declare that I would now specialize in Clinical Preventive Medicine. When I sought the help of Dr. Joe Aub, my mentor, famed for his studies of lead and radium poisoning, he grunted, "What's that you want?" But he understood, and a few weeks later in the fall of 1945, I found myself working under Mr. Manfred Bowditch at the Massachusetts Division of Occupational Hygiene, in the Department of Labor, just at the time of the epidemic of disease in the Massachusetts fluorescent lamp manufacturing. I have never had a dull day since! I beg pardon for so much autobiography. It is intended to show how risky medicine is for women (and men) and further what a variety of roads may lead to your specialty and mine, the study and control of man-made disease.

Now for a few serious observations. I want to take advantage of this platform to speak of what this young Society might consider as it develops. By admitting to its membership those who view the world and its problems through the fundamentals of a wide variety of disciplines, the Society of Occupational and Environmental Health has assets and liabilities in taking this basic position. For assets, differently trained professionals must learn to communicate what they learn in order to pool knowledge, and to use it. A mixture of variously educated individuals are more likely to ask the right questions for the benefit of society as a whole than is the potential Nobel Prize winner in his laboratory. Somewhat

against the complete mix this Society has decided on is what is found in the old phrase "a little knowledge is a dangerous thing." And perhaps the openness of your Society harms the very people you plan to help by misinterpretation and exaggeration, not only by the much maligned media but also by publication in scientific journals whose hearts are not pure and whose loyalties are compromised. My experience confirms what I learned from my distinguished senior, Dr. Alice Hamilton, that government agencies, by industry's insistence on their special brand of "right to privacy," fail to serve worker and exposed neighbor. When I complained to an official of the National Academy of Science of this all too common practice, his reply was, "Harriet, we must have industrial input." Perhaps you in the Society for Occupational and Environmental Health can find a way out of this dilemma.

My mail and talks with residents and my colleagues convince me that Earth Day in 1970, a wide variety of consumers' groups, and now your Society for Occupational and Environmental Health have developed impressive steam. Government agencies and industry cannot ignore the powerful counteraction to the evil consequences of the Industrial Revolution, the endless warring. I applaud these important evolutionary changes in our society.

My observation and reading lead me to urge you to consider the concept of risk in perspective. Listening last evening to women workers describe a variety of job hazards made a basic point that this Society should study in depth, as the Cambridge students say. I refer to rating work-related risks. The itch of fiberglass is horrid, need not be endured because it can be controlled by engineering methods or protective clothing, but even uncontrolled does not carry the threat to health that a small amount of carbon tetrachloride does. Do not waste the power of this Society without careful thought as to relative risks. Your leaders must give you hazard lists with priority ratings. Further, there is little written on the fantastic power of organs such as the liver to detoxify; damaged cells are discarded, new ones take their place. It is true that the body can recover from serious insult, a point rarely considered in current observation and techniques for setting standards. Reports of delay in onset of disease from the date of harmful exposure are frightening. But forgotten is the history of different insults during the long latency period -- viral infection, smoking habits, high alcohol intake, surgery requiring toxic materials as anesthetics, other jobs with insults requiring detoxification, storage and excretion, until, finally, the defenses of the body are overcome.

In my studies of the latent period in chronic beryllium disease, I learned that intensity and duration of exposure (I like the expression "dose-rate") were the most important factors in determining the date of onset of illness. It is rarely mentioned that 50% of the Salem Fluorescent Lamp workers became ill with chronic beryllium disease while on the job; precipitating factors

in the latent period need consideration. Well documented in our series of beryllium disease were pregnancy, surgery, and other industrial exposures. Such results from modest clinical observations need emphasis, not supplanting but guiding the present enthusiasm for biostatistical study of large or small groups--statistical maneuvers taking the place of the unknowns.

Finally, I make a plea for human and humane epidemiology--ideally prospective, in searching for correct correlation between exposure and disease. I use the word plea, because you and I will live to see and must insist that cell cultures rather than small animals or human volunteers will become the ideal method for studying the danger of this or that chemical. Hopefully, advancing techniques will make it possible to assess potential hazards before any human exposure. Critical points in my plea are questions. Do those qualified to do so look hard enough for a harmless material to use in place of a harmful one? Is the concept of time-weighted average based on biological or engineering principles?

You have been very patient in listening to me. The process of aging may prevent me from many more such golden opportunities to talk of myself and some of my favorite ideas. Thank you, and best wishes for the success of your Society.

A STATEMENT
BY THE COALITION OF LABOR UNION WOMENS' TASK FORCE
ON OCCUPATIONAL HEALTH AND SAFETY

"As a co-sponsor of the 'Women and the Workplace' Conference, CLUW was well-satisfied that issues relating to the health and safety of all workers were raised; that consideration was given to the woman worker and the question of whether she created a unique problem in the workplace; and that concern about the unborn child was discussed.

"We realize that a key problem is how to balance equal employment opportunities and health. Women workers have the right to work and they have the right to a job that is safe. As the conference made clear, the solution to the problem of protecting different groups in the workplace and, at the same time, of insuring their social and economic equality, has not yet been reached.

"CLUW will continue to work toward protecting the health and well-being of women workers. With the formation of the CLUW Task Force on Occupational Health and Safety, we are now on our way to doing this. Only through the efforts of women workers and their labor organizations can the scientific observations discussed at this meeting be able to best benefit all workers."

A STATEMENT
BY THE WOMEN'S POLITICAL ACTION - OCCUPATIONAL HEALTH CAUCUS

(As delivered to the conference on June 18, 1976)

We are a group of 23 women who met for the first time during the noon break to discuss our reactions to the proceedings so far. We met because we were concerned about the tone and perspective of the conference. While we commend the conference planners for this first important step, as women active in the women's health movement we wish to make the following criticisms and recommendations to the group as a whole. Further, we request that others take up these issues at Saturday's open discussion and that our remarks be incorporated into the conference proceedings.

1. We do not see the issue of women's occupational health strictly in scientific medical terms. Women's occupational health includes social, psychological, economic, familial and political factors. We urge the development of a holistic view on women's occupational health.

2. In this regard, we are critical about the narrow view this conference has had toward women. Women have been seen as reproductive vessels. Both men and women are involved in reproduction. Reproduction is not solely the woman's responsibility. Society is ultimately responsible for the preservation of future generations. We do not want women to be penalized through denial of the right to work or by the lack of adequate childcare facilities.

3. We are concerned by the emphasis on technical terms and presentations at the conference. If the interest was communication between workers and scientists, that purpose was not achieved. Rather papers were presented in a lecture format to people who were already familiar with the material. And, if they were not familiar with the literature, they would be unable to understand the presentations as they were delivered. We recommend that future conferences make scientific abstracts available to all participants in advance and that workshops and open discussions of the results of technical findings and what should be done about them be encouraged.

4. We are critical of the scheduling of the discussion with labor union women which was relegated to an evening session instead of being an integral part of the program. We protest the trivialization of their personal experiences. Scientific understanding should illuminate personal experience; personal experience should inform and enrich scientific generalizations.

5. While we agree that women have special occupational health problems as well as sharing many occupational health problems with men, we are critical of the conference's ignoring crucial problems. These problems include the following: sexual exploitation (usually

by male bosses), the long work week of most women who must hold two jobs, one in the home, the other outside the home, the absence of day care and adequate maternity leave, the stress of dead-end jobs, the lack of union protection for most women, the problems of third world women both in America and abroad.

6. We reject the paternalistic statements which assume that if "you" women workers are educated about job hazards, then "you" women can exercise free choice about "your" employment. Given the present unemployment rate and discrimination against women workers, this is an absurd and condescending attitude toward both women and men workers.

7. We believe that the definition of women workers should be broadened to include all those who perform work. We must investigate occupational situations faced by office workers and house workers, women paid or unpaid.

8. We question the assumption expressed by many at this conference that the burden for improving occupational safety lies with the workers. While we support the efforts of unions and women's organizations to improve conditions within the workplace, it is important to remember that the ultimate responsibility for change must be placed upon management of business and industry. They caused this problem; they decide where the money will go; and they must be held responsible for unsafe conditions in the workplace.

9. We support the statements of the women who described their work situations during Thursday night's program. While we value scientific knowledge and research, we believe such inquiry must appropriately develop in response to personal experience of the lay public. It is certainly valid to provide personal descriptions of hazardous occupational conditions, because such accounts speak both to scientists and those without scientific background.

We support emotion; it does not preclude or negate a scientific approach. We support emotional expression because it may lead to a justified anger--which must be the basis for positive change.

A STATEMENT BY THE LABOR SAFETY AND HEALTH INSTITUTE

Women in general and black and other minority women in particular are systematically excluded from professional and skilled trades. They make up, for example, less than 3% of professional engineers and dentists and 9% of all medical doctors. Women are not hired into higher paying skilled construction trades jobs. However at the same time women are entering the workforce in ever-increasing numbers. Where are women working?

Employers tend to segregate women from mixed workplaces and into industries such as the garment and textile plants, human service work, certain electrical industries and within some plants into what has been incorrectly termed "light work" areas.

A few examples of this "light work" are the severely stress-producing work of telephone operators; retail meat wrapping which has been found to produce severe respiratory ailments; high pressure, speed-up employment in the needle trades which are now introducing known and unknown hazardous chemicals and solvents.

The historic use of the term "light work" is an indication of the lack of seriousness with which management and government officials have often seen this kind of so-called "women's work." In short, this is not "light work" which does not contain hazards! But industry and government continue to perceive this as "light work" and is not taking appropriate corrective action. For that reason this Conference is vitally important and is giving this issue the special attention it requires.

Reproductive Function Both men and women can face workplace hazards that endanger their reproductive organs and federal standards should be promulgated with that keenly in mind. NIOSH has recently admitted that this is not done. However, while both men and women may face these hazards, in the final analysis, it is the woman who bears the child. Given the continuing overall discrimination against women which takes place in the courts of law, in education and training opportunities, receiving credit from banks, attaining health and other insurance, receiving lower wages for comparable work, women face a combination of discriminations which requires special attention. Safety and health protection on the job cannot be separated out from the national disgrace on the treatment of women in the U.S.

Lead Protection for All Workers The often-used example of lead poisoning carries with it conclusions which are often confusing if not incorrect. Here, women are sometimes barred from working as with National Lead Industries. There may be no doubt that women and their reproductive organs are more susceptible to lead poisoning than men and that they should be barred and given another job with no loss in seniority or wages. However, the

workplace should then be cleaned up for the men also so that the dangerous situation taking place in the Prestolite lead battery plant in Los Angeles described in the N.Y. Times Business and Finance Section (June 6, 1976) would not have taken place. In fact, this seemingly enlightened corporate policy of excluding women for their protection may have given male workers the false impression that the workplace was safe for them. This is surely not the case.

Some would draw the conclusion under equal employment laws that women should be placed back into these hazard-laden battery plants with male workers. This is the wrong lesson.

What is the lesson? Whatever federal or state protective legislation or corporate policy which has been justly passed or demanded to protect women workers should be extended to all workers. This would include male workers and men and women workers who are not capable of child-bearing.

According to a 1976 study done by the U.S. Labor Department's Women's Bureau entitled State Labor Laws in Transition: From Protection to Equal Status (a telling title) protective legislation is generally being withdrawn from the books rather than being extended. Extension of these laws to all workers would be taking place if state legislatures had workers uppermost on their legislative minds. The California Welfare Council has recently wiped out such protective legislation which protected women and youth rather than extend as it had power to do.

Recently the Coke Oven Standard Setting process disclosed that women workers are being hired into coke ovens as New Hires, even though it's been known for over 200 years that these ovens are cancer breeders in all humans and more recently that benzo(a)-pyrene, a major coke oven pollutant, is transplacental to the unborn fetus. This cynical steel industry hiring policy which is being done in the name of equal employment must be terminated by immediate government action.

A special OSHA Advisory Committee including trade union women and independent medical/science professionals should be appointed to: 1) Review similar corporate hiring practices and recommend termination for those which are determined to be discriminatory and dangerous; 2) Review all federal safety and health proposed or legal standards to insure that the minimum TLV is established for federal standards which will cover all workers, male and female, and regardless of age or reproductive capability; 3) Take the initiative in developing special women's occupational safety and health mandates which may demand that the OSHA investigate certain overlooked industries which hire mainly women workers; encourage the hiring of women compliance officers and investigate workplaces which have dangerous chemical exposures that are already known to cause dangers to women; 4) Investigate

all claims by industry that government regulation which may call for exclusion of certain workers, men or women, from hazardous jobs constitutes a violation of federal equal employment laws. These industry claims are now going uninvestigated. As a result, all action in this area of job safety and health by the federal government has halted, and it has seemingly inhibited labor to take action.

The establishment and enforcement of existing and proposed laws for equal rights for all Americans is a paramount goal which can only be enhanced by the consistent protection of workers from job hazards as they become known. This may mean the transferring of workers from dangerous jobs with no loss in pay or seniority. These regulations ultimately would then be extended to cover all workers toward a completely hazard-free workplace.

These so-called fears of non-compliance with equal employment laws claims by industry appear to be just another tool in an arsenal of tactics within a broad strategy to weaken and ultimately repeal the OSHA itself. This is the only conclusion which can be drawn for in almost no case has an industry which has issued this claim voluntarily launched a clean-up program for the workplace to protect all workers.

Special SOEH and Conference Role The Society for Occupational and Environmental Health and others attending this Conference are in the best position to separate fact from fiction as women workers demand their equal rights and also demand safer and healthier working conditions for themselves, their families and their communities.

A special continuations committee established by the Conference can follow through on recommendations proposed by the attendees. This Committee would be charged with pressuring the Labor Department's OSHA to establish the aforementioned Advisory Committee including the attached program and immediate activities.

This Committee would investigate the conditions created by management and the scientific information which drove United Auto Workers member Norma James, a General Motors lead battery worker, to sterilize herself to try to insure her continued employment. It would also investigate situations such as the one encountered by Karen Silkwood at the Kerr-McGee plutonium plant and the coverup of the investigation into her death.

This Committee can follow through on other recommendations coming out of this Conference that may include the establishment of special units within NIOSH dealing with reproductive hazards faced by both men and women, and also industries which employ mainly women workers.

In addition, the SOEH newspaper, HAZARD, can begin the

discussions needed on these issues as well as circulating information on other issues as they surface.

NOTES

