DISCUSSION OF TLV'S FOR LEAD

Dr. Bertram W. Carnow
University of Illinois School of Public Health

Thank you. As Dr. Blejer pointed out, virtually everything has been said. I should like to present some of my concerns regarding TLV'S. These relate directly to the advisability of relying on biological monitoring as a measure of health impact of environmental stressors. This concept has come under considerable discussion with some suggestions that it be used as a substitute for environmental monitoring.

The TLV, by definition, is a level of environmental contamination at which workers can carry out job activities for eight hours a day, forty hours a week, presumably over an entire working lifetime, without any adverse health effects. In studies we have carried out over the past ten years, examining the health effects of ambient air pollutants on communities, negative health effects were found at levels considerably lower than are found in occupational settings. For example, in community studies SO_2, which has a TLV of 5.0 ppm, has been shown to cause a sharp increase in acute exacerbation of bronchitis in those with chronic disease and increased cardiac and respiratory deaths at levels between 0.2 and 0.3 ppm.[1-2] These levels are 15 to 25 times lower than those stated to be safe for occupationally exposed workers. This raises the whole question of whether or not the threshold concept is a valid one. I think not.

It must be recognized that humans are in a constant struggle with their environment, which is essentially hostile and the outcome of this host-environment contest, in which all humans and other biological organisms participate, depends on the adaptive capacity of the host as well as the toxicity and level of the environmental stressor. Our studies suggest that there is a high degree of variability in individual response to varying levels of air pollutants. Further, some segments of the population appear to be at much higher risk than others. In fact, it would appear that there is a highly variable dose-response relationship in a heterogeneous population so that at every level of a contaminant, depending on the adaptive capacity of the individual observed, there may be an increase in symptoms, prolongation of illness, and, in some cases, premature death.

The critical question, therefore, is not, what is the magic, albeit mythical, number symbolizing *safe*, but rather how many workers are adversely affected at each level of the pollutant. Other critical questions which must also be asked are: how serious are the effects, how reversible are they, what organs are affected, and how can we best measure the effects: I contend that in order to answer these questions many more studies need to be carried out looking at populations who may

be at higher than normal risk, so that a level may be defined that will really protect all, or at least most people. Many factors which will be briefly discussed, have either not been considered at all or were only superficially considered in examining safe levels. This may, in part, be responsible for a TLV for lead of 200 µg/cu m of air and of 500 µg for arsenic, neither of which, as this conference has shown,is safe.

Multiple Insults of Single Target Organs

Many TLV's are set as though the agent under study was the only one present in the environment. This, of course, is usually not true. There are a number of trace metals, such as cadmium, lead, and mercury, as well as arsenic, which may affect the central nervous system. The effects of these impacting on a single target organ system, the central nervous system, has not been adequately considered, even though some of them frequently appear together in processes like smelting. The problem of multiple insults on a single target organ is certainly real, and must be examined more carefully in relation to arsenic, which is one of many toxic materials etiologically related to lung cancer.

Single Agents Causing Multiple Disease

Another problem relates to the possibility that a single agent may cause multiple diseases and we may be looking at the wrong one in assessing a safe level. Studies reported here by Repko suggest that there are significant negative health effects of lead at levels of 70 to 80 µg/100 ml of blood, a level previously considered by some to be safe. His findings of significant personality changes, sensory loss (hearing), and neuromuscular and psychomotor abnormalities, are quite troubling. These were not the organs or functions that were looked at in determining whether or not lead toxicity was present in a worker. Instead, gross measures, such as anemia, abdominal colic, or wrist drop, were the endpoints, along with a level of 80 µg/100 ml of blood.

Dr. Repko's studies and the studies of others give me very little confidence in the reliability of blood lead levels, even if accurately measured, as a determinant of whether a level is safe. Seppalainen's study of 26 workers, most of whom had less than five years of exposure,[3] revealed that a high percentage had pathophysiologic findings, even though the levels of blood lead, found during very frequent testing over a five-year period, were between 35 and 60 µg/100 ml. Neurologic dysfunction was also found to be present, including decreased maximum motor conduction velocity, sensory conduction velocity, and slow motor fiber conduction velocity.

Dr. Goyer's report on renal pathology suggests more attention should be paid to kidney disease. It also gives one little comfort that workers are remaining healthy, or being protected when the blood lead levels are 80 µg/100 ml.

Multiple Sources of Single Pollutants

The problem of multiple sources of a single agent like lead is also given little consideration in the establishment of a TLV. Cigarette smokers accumulate considerably more lead than other individuals. Lead may also be taken into the body from airborne particles, resulting from automobile exhausts, the leaching from cheap pottery, the consumption of vegetables grown in urban areas, and from other environmental sources. If workers live near the plants in which they work, they may also be exposed to additional elevated air, soil, and dust levels of substances like lead or arsenic. This is particularly true in communities surrounding smelters.

Synergistic Effects

An additional important parameter, which has received little consideration in determining safe levels in the workplace, relates to the synergistic effects of these materials with other stressors. It has been shown in animals that dietary deficiency in chromium,[4] iron,[5] and calcium[6] increase lead absorption and, therefore, toxicity. Gastrointestinal infections, like salmonella,[7] were shown to cause an increase in the release of lead from bone into the blood. Increased alcohol intake and possibly other factors relating to habits or diets may enhance the negative effects of lead on the body.[8]

Greater consideration must also be given to the age of onset of exposure as well as the length of exposure, not merely to blood or air lead levels found. Since there is a strong suggestion from data presented that it is possible to develop asymptomatic lesions, without symptoms, over a long period of time, and some of these effects almost certainly are irreversible. Many smelter and other lead workers, because they are at a low socioeconomic and educational level, cannot easily move from job to job, so they may have such long-term exposures.

It is no wonder, that the present TLV is inadequate to protect the health of workers. Even if achieved and adhered to, it will result in blood lead levels in many workers of close to or exceeding 80 µg/100 ml. Since safe implies a "no effect" level plus a safety factor, this would appear to be greatly in excess of such a level. Many other TLV's are in this same category.

I am also concerned about the paucity of epidemiologic data. Dr. Cole suggested that lead is possibly the best documented metal, epidemiologically. We, however, had considerable difficulty during the planning of this meeting, in finding more than a very few, methodologically sound epidemiological studies.

For all of these reasons, therefore, I would suggest that we cannot even remotely consider major dependence on biological monitoring. What is required is many more epidemiologic studies with the generation of considerably more and better data, including better definition of

measurement of body burden, and more sensitive tests to detect early pathology. In addition to examining multiple organs for early pathophysiologic changes and reviewing blood lead *versus* ALA, FEP, and ALAD, to determine the best measurements for assessing body burden, the quality of the instruments used for monitoring air levels of lead needs more attention. There is some evidence that possibly 25 percent or more of airborne lead may be passing through the filters of testing devices, and, since most of the lead present in the air is in submicron particles, this may cause gross under estimation of the air lead burden.

Appropriate consideration of all of the above factors will, I believe, make possible the setting of a level for air lead contamination and body lead at which all, or at least most, workers will be able to work out their entire working lives without adverse health effects. TLV's generally would appear to be based on studies carried out on survival populations, that is those who had not become ill, died, and were not driven out of the industry because of illness. I do not believe that these are appropriate cohorts for study.

In conclusion I would suggest that:

1. TLV's generally, and particularly for materials which are known to cause chronic disease or cancer, be qualitatively evaluated.

2. Biological monitoring alone is not an appropriate way to assure safety and health for workers. Both environment and hosts must be monitored to assure low levels of contaminants for the continued health of workers. This is particularly true for materials which may cause chronic disease, cancer, or genetic or reproductive abnormalities.

3. We develop tests of greater sensitivity which correlate well with early pathophysiologic changes. In the case of lead, ALAD would appear to be such a measurement and is apparently far superior to blood lead levels.

4. We continue to carry out studies to ascertain all those who are at high risk because there are males or females in the reproductive age range and because of other chronic diseases, genetic deficits, or nutritional or other factors so we can quantitatively define the level of risk by matching good environmental measurements with sensitive biological endpoints.

5. We then promulgate standards which will protect all workers and not seek to exclude those who might be at higher risk, such as pregnant women, and others.

6. We carefully examine the question of whether current levels of technology will adequately protect workers by assuring low levels of environmental contamination in industry and, where these are not available, to make this a research priority.

It is only in this way that the workers will, indeed, be able to carry out work activities eight hours a day, forty hours a week, for a working lifetime without seriously affecting their health.

REFERENCES

1. Carnow, B.W., Lepper, M.H., Shekelle, R.B., and Stamler, J. The Chicago air pollution study: SO_2 levels and acute illness in patients with chronic bronchopulmonary disease, Arch Environ Hlth 18:768, 1969

2. Carnow, B.W. and Carnow, V., Air pollution and the concept of no threshold, chapter in Advances in Environmental Science and Technology, Vol 3, 1974

3. Seppalainen, A.M., Sakari, T., Hernberg, S., and Kock, B., Neuropathy at "safe" levels of lead exposure, Arch Environ Hlth 30:180-183, 1975

4. Schroeder, H.S., The role of chromium in mammalian nutrition, Amer J Clin Nutr, 21:230, 1968

5. Six. K.M., Goyer, R.A., and Haseman, J.K., Dose-response to lead ingestion in rats fed low dietary calcium, J Lab Clin Med 82:100, 1973

6. Background information, Proposed National Emission Standards for Hazardous Air Pollutants: Asbestos, Beryllium, Mercury, EPA Office of Air Programs Publication No. APTD-0753, December, 1971

7. Hemphill, F.E., Kaeberle, M.L., and Buck, W.B., Lead suppression of mouse resistance to salmonella typhimurim, Science 172:1031-1032, 1971

8. Mahaffey, K.R., Nutrition factors and susceptibility to lead toxicity, Environ Hlth Persp, 107-112, May, 1974

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

HEALTH EFFECTS OF *OCCUPATIONAL LEAD and ARSENIC EXPOSURE* A SYMPOSIUM

HEALTH EFFECTS OF OCCUPATIONAL LEAD AND ARSENIC EXPOSURE

A SYMPOSIUM

Edited By

Bertram W. Carnow, M.D.
University of Illinois School of Public Health
Contract Number 210-75-0026

Chicago, Illinois
February 24-25, 1975

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
Division of Surveillance, Hazard Evaluations and Field Studies

February 1976

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402

SPONSORED BY: Society for Occupational and Environmental Health
National Institute for Occupational Safety and Health
United Church Board of Homeland Ministries
University of Illinois School of Public Health
Chicago Lung Association

SUPPORTED BY: National Institute for Occupational Safety and Health
United Church Board of Homeland Ministries

PLANNING COMMITTEE:

Bertram W. Carnow, Chairman
Edward J. Calabrese
Richard A. Lemen
Vaun A. Newill
John Zalusky

HEW PUBLICATION NO. (NIOSH) 76-134

The Editor or his staff prepared abstracts of the presentation when they did not accompany the author's manuscript.