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Opening Remarks:

A Symposium on the Diagnosis and Amelioration of Mass Psychogenic Illness in Industry

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Proceedings from a symposium on the diagnosis and amelioration of industry related mass psychogenic illness (MPI) is presented. Outbreaks of unusual behaviors, sickness and hysteria in manufacturing and assembly facilities and workplaces in the United States, and other countries including England and Singapore are reviewed. Outbreaks of MPI investigated by NIOSH also are included. Social comparisons, complexities, cautions and concepts in research on MPI are discussed. Stress, illness related behaviors, somatization, triggering events, theoretic and psychoanalytic approaches to the study of MPI are described with reference to concepts of convergence, contagion, emergent norms, core variable, sex difference, and coping with stress. Factors that affected symptoms including hysterical contagion, quality of the working environment, mechanization, automation, shift work, extra-large enterprises, anonymity of the workers. Workers and management relations are discussed. The effect of MPI on rapid industrialization in developing countries in described and MPI and chemically induced hypersusceptibility are compared.

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MASS PSYCHOGENIC ILLNESS: SOME CLARIFICATION AND PERSPECTIVES

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The spontaneous outbreak of illness among a group of workers is a dramatic and distressing event. Of primary concern, of course, is the immediate treatment and recovery of the affected workers and the detection and elimination of the illness source. To the extent that the cause can be readily identified and understood, anxiety surrounding the event diminishes. Once the workers have a rational explanation for what happened to them, they can begin to realistically evaluate their symptoms, not only in terms of their immediate significance, but also in terms of their long-range implications. Workers can also monitor efforts to remedy the situation, allowing them to eventually return to work with the assurance that the problem has been corrected and the environment is safe.

Unfortunately not all incidents of mass illness in working settings are so easily resolved. In some cases a physical or chemical cause for the illness may not be obvious, or recurrent episodes may occur long after the initiating cause has been removed, or the expressed symptoms may be too non-specific and transient to permit diagnosis. Under these conditions, the illness remains a mystery and the workplace becomes a breeding ground for anxiety, rumor, confusion and fear. Economic pressures to resume normal operations are counteracted by genuine concerns that the environment is still pathogenic, and workers vacillate between their desire to return to work and their fear that to do so may be hazardous to their health. Having exhausted all possible physical explanations for the illness episode, investigators may turn to a psychological interpretation of the outbreak, explaining it in terms of mass hysteria or mass anxiety. This conclusion is always a tenuous one, being based primarily on the absence of physical evidence rather than the presence of a clearly defined set of precipitating psychosocial conditions. This ambivalence is understandable given the long and controversial history of the mind-body issue in general, and the concept of "hysteria" in particular. Despite these semantic and theoretical pitfalls, the fact remains that the workplace is as much a psychosocial environment as it is a physical one and the effects of psychogenic stressors may be as threatening and debilitating to a workforce as excessive noise levels or inadequate ventilation. There is an urgent need to re-examine the old issue of mass hysteria in terms of contemporary theory and research findings. By what mechanisms do psychological properties and processes become translated into physical symptoms? What conditions promote or limit the contagion of psychogenic symptoms throughout a workforce? Are some individuals or work settings more susceptible to such illness outbreaks than others, and if so what are these critical precipitating conditions? Is it possible that predisposing psychological factors may, in and by themselves, trigger off a cantagion of psychogenic symptoms? To what extent do these same psychological stressors lower worker resistance to sub-standard chemical exposures, making them hypersensitive to mild irritants in the work environment?

What steps can be taken to contain the spread of panic and anxiety once a mass illness outbreak has occurred? Complexity is not a viable excuse for avoiding these issues, and while we might not be in a position to apply any quick and easy solutions to these problems we can at least attempt to develop a conceptual framework for understanding them.

This symposium is part of an effort by the National Institute for Occupational Safety and Health (NIOSH) to elucidate the role of psychosocial factors in the etiology of certain illness episodes in work settings. In this respect, NIOSH psychologists, working in close conjunction with NIOSH industrial hygienists and occupational physicians, have collected data of a psychosocial nature regarding eight distinct illness outbreaks having apparent psychogenic components over the past three years. The general investigation protocol and results of some of these investigations will be presented here today.

In addition to the NIOSH speakers, we are indeed fortunate to have such a distinguished panel of internationally renowned scientists and researchers. Each brings a unique expertise and perspective to the understanding of the major theoretical, methodological, and treatment issues relating to mass psychogenic illness in occupational settings. Our concern for the psychodynamics of certain mass illness episodes does not imply a disregard for the physical and chemical hazards which poetntially exist in the workplace. The materials to be covered in this symposium are meant to complement rather than replace the traditional physical and chemical evaluative process in the investigation of a mass illness outbreak.

Mass Psychogenic Illness: Some Clarification and Perspectives

Our understanding of the conditions influencing individual health and well-being has been greatly enriched by recent advances in fields such as psychosomatic medicine, medical sociology, and health psychology. A basic tenet of this movement is the recognition and affirmation that psychosocial and behavioral processes are important factors in the etiology and exacerbation of a wide range of physical infirmities. Coronary heart disease (e.g., Friedman and Rosenman, 1974; Glass, 1977; House, 1975), hypertension (e.g., Cruz-Coke, 1960; Scotch and Geiger, 1963; Pilowsky et al, 1973), dermatitis (e.g., House et al, 1979), rheumatoid arthritis (e.g., Cobb et al, 1969; Cobb et al, 1959), ulceration (Dunn and Cobb, 1962; Mirsky, 1958), respiratory illness (Jacobs et al, 1970) and a variety of other disorders (e.g., Levi, 1971; Weiner, 1977; Selye, 1956) have been shown to have a psychogenic component. The experience of illness, in such cases, is presumed to result from a depletion of the individual's resources for coping with stress (Selye, 1956; Levi, 1971) and signifies a discordance between perceived environmental demands on the one hand, and individual adaptative capacities on the other (Monat and Lazarus, 1977). This suggests that our understanding of the etiology of psychogenic illness must encompass (at least) three sets of interdependent variables.

The first set consists of psychosocial events and conditions which are inherently stressful and potentially pathogenic to the majority of individuals experiencing them. Bereavement (Lohmann, 1977; Averill and Opton, 1968), social isolation (Bowlby, 1973), abrupt social and cultural change (Gunderson and Rahe, 1974; Dohrenwend and Dohrenwend, 1974), crowding (Levy and Herzog, 1978; Cassel, 1974), and threat or uncertainty (Averill and Rosenn, 1972; Hodges and Spielberger, 1966) are among the factors implicated by previous research as morbific. Behavioral scientists interested in occupational health are now beginning to examine similar psychosocial factors associated with the work environment in an attempt to determine their impact on the safety and health of the individual worker (Caplan et al, 1975; Sleight & Cook, 1974). Job loss (Cobb and Kasl, 1977), machine pacing (Caplan et al, 1975), time pressure and deadlines (Friedman et al, 1958), organizational ambiguity and conflict (Kahn, 1975; Kahn et al, 1965) and overload (Kahn et al, 1965; House et al, 1979) are but a few of the job stressors investigated to date. This research suggests that the psychosocial characteristics of various work practices and organizational styles should be examined and described in terms of their pathogenic potential, much along the same lines as current attempts to identify physical and chemical hazards in the workplace. Such an approach does not imply that a given condition is equally stressful to all parties, but rather calls for an extensive data base describing

the covariation of select work parameters (e.g., overtime, workpace, interpersonal and physical spatial distribution, work/rest ratio, role clarity, supervisory style, job complexity, repetitiveness, etc.) with measures of morbidity and/or well-being in a well-defined worker population. The resulting norms would provide information about the probable impact of specific organizational and psychosocial work practices on the average worker. This is essentially what has been done in the fields of ergonomics and human factors engineering where researchers have focused on "man-machine" interactions in an attempt to tailor the demands of the job task and work environment to the physical, perceptual, and mental capacities for the majority of the worker population.

A second set of variables bearing on the etiology of psychogenic illness involves the psychological processes (e.g., attitudes, expectancies, values) and personality characteristics (e.g., extraversion/introversion) of the individual which shape his or her views of the surrounding world. Individuals do not respond directly to an environmental event, but rather to their perceptions or interpretations of that event.

An upcoming speaking engagement may be perceived as either threatening or challenging depending on the speaker's self-perceived skill, confidence, and orientation toward the audience. A pending move to a new city may be perceived as an opportunity to broaden our experience and develop new friendships, or it may be perceived as an uprooting of the old secure and comfortable social ties which were the source of so much personal stability. Depending on the meaning that we attach to such events (e.g., the speaking engagement or the pending move), one's behavioral and emotional reactions will vary with differing consequences for health and well-being. In fact, Mason (1968), in reviewing research on psychoendocrinology concluded that psychological influences (e.g., novelty, uncertainty, ambiguity) are among the most potent stimuli affecting pituitary-adrenal cortical activity. An understanding of the etiology of psychosomatic illness, then, requires a description of the psychological processes by which an individual comes to experience an event (either real or anticipated) as stressful and its subsequent impact on psychoendrocrine and autonomic reactivity (e.g., Schachter and Singer, 1962.)

The third set of factors underlying psychogenic illness involves coping mechanisms which individuals employ to minimize the impact of experienced stress. The types and range of coping strategies at our disposal are considerable. The most effective means of eliminating stress arising from physical and psychosocial conditions is to act directly on the stress sources such as to produce the desired change. This approach presupposes that the individual has sufficient power and control to effect the change and that the stressor is clearly recognized. Frequently this is not the case. The individual must then

resort to other coping modes. Current interest in physical exercise as a general panacea, and in specific stress-management techniques such as relaxation training (Benson, 1975), biofeedback (e.g., Shapiro et al, 1974), transcendental meditation (Naranjo and Ornstein, 1971), and perceptual/cognitive reappraisal and labeling processes (Folkman, Schaefer, and Lazarus, 1980) indicate the importance that health researchers assign to coping processes in the prevention of stress-related disorders.

The three sets of factors just described in conceptualizing a basis for psychogenic illness represent a gross simplification of what actually occurs "in situ." Indeed, the variables determining one's health status are neither as static or as independent as the preceding discussion implies. Rather, they may interact in complex ways, either reinforcing or modulating the influence of one another. Nevertheless, even this elementary formulation should be sufficient to dispel some popular misconceptions about the nature of psychogenic illness.

For example, it should be recognized that to suggest that an illness has a psychogenic component does not imply that the experienced symptoms are imaginary or illusory. There is a vast literature describing the adverse impact of stress on individual physiological functioning and well-being (e.g., Selye, 1956; Mason, 1968; Levi, 1971; Weiner, 1977). The corresponding symptoms are as distressing and real as those having more traditionally recognized etiologies.

A second misconception is to confuse the term "psychogenic" with "neurotic" or "psychopathologic." As previously discussed, "psychogenic" means "of Psychologic origin" and refers to the fact that certain illnesses are the result of a chronic or traumatic conflict between individual coping capacities and experienced demands. The development of an ulcer in response to prolonged interpersonal conflict (e.g., Weiner, 1977), a heart attack following excessive and repeated deadlines and time pressures (Friedman and Rosenman, 1974), or arthritic attacks subsequent to losing one's job (Cobb and Kasl, 1977) are examples of psychogenic illnesses and reflect a discordance between individual adaptive capacity and experienced stress. Most of us would agree that the pressures, tensions, and conflicts precipitating these illnesses are real and legitimate threats to individual well-being, and that the appearance of symptoms is "reasonable" under the given circumstances.

Neurotic or psychopathologic reactions are a subset of the former (e.e., psychogenic illnesses) in which the individual's experienced stress or anxiety is irrational and excessive, given the objectively defined characteristics of the physical and psychosocial environment. Phobias, anxiety reactions, obsessive-compulsive behaviors reflect abnormally intense

responses to events and conditions (real, anticipated, or imagined) which the average individual would perceive as benign or inconsequential (e.g., Colemen, 1976). Psychogenic illness is a more general category and encompasses not only neurotic reactions, but also those situations in which illness is a more probablistic (i.e., more "normal") response to recognized environmental psychosocial stressors. The term "psychogenic" is not synonymous with "neurotic" or psychologic then, and is equally descriptive of normal and aberrant psychological functioning.

A third misconception dealing with psychogenic illness is that somehow the individual is more to blame, or more at fault for the etiology of this kind of illness than is the case with biogenic or chemogenic illnesses. But, as we have suggested, the etiologic description of psychogenic illness requires conceptualization of (at least) three sets of interactive variables: 1) environmental psychosocial events and conditions; 2) cognitive/perceptual/emotive processes; and 3) coping reserves. In any one case, the relative contributions of the three kinds of factors may differ, and a complete understanding of the illness etiology requires analysis and description of their respective significance. The causal base here should not be confused with degrees of blame or responsibility, however. Illness is a complex process involving host-environment interactions which are not amenable to pure and simple attributions. It is as absurd to blame the victim of an illness of a psychogenic nature as it is to blame the coal miner for his pneumoconioses.

Finally, it should be recognized that illness may be multi-determined, involving the interplay of psychogenic, chemical and physical hazards in our environment and vice versa. A complete understanding of the illness process requires that we give serious consideration to all potential pathogens, be they social, psychological, biological, chemical, or physical.

The present conference represents a preliminary attempt to identify the relevant organizational, psychological, and sociological parameters of mass illness outbreaks having apparent psychogenic undercurrents where a physical basis for accounting for such disorders could not be ascertained. Indeed, the complex nature of the illness process precludes simple solution and those seeking definitive statements will be disillusioned by the papers to follow. The conference should be viewed as a prologue rather than an epilogue, and it is hoped that these proceedings will sensitize us to the role of psychosocial stressors in the etiology of worker illness, and provide us with a tentative framework for future investigations.

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TRENDS IN MENTAL EPIDEMIC DIAGNOSIS AND INVESTIGATION

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Outbreaks of unusual behaviors have been noted in various literatures for at least a thousand years. In the last forty years, however, there have been a number of field investigations and reviews published. The purpose of this report is to review the critical questions which these have raised.

Two independently conducted reviews, one by myself in 1973 (1) and another by Sirois in 1974 (2), together covered 153 mental epidemics. One hundred and four of these occurred in the past century. The review by Sirois, which included a large number of epidemics reported in non-English literature, was limited to episodes related to his concept of epidemic hysteria; my review included other forms of mental epidemics, such as community panics from radio dramas, Swastika epidemics, heroin and cigarette abuse, trauma associated with risk-taking behavior, and transient neurological disturbances. Neither review included epidemics of suicide, homicides, wars nor most other forms of violence. These diverse behaviors may share some of the economic and social determinants of industrial and other community mental epidemics, but have their unique determinants as well. Yet until we know more about them, it may be practical to study them using similar investigative methods.

Of my list of 100 epidemics occurring since 950 A.D., only four (the first in a cotton factory in England in 1787) occurred in industrial settings. Of Sirois' 70 epidemics occurring since 1876, only 8 were industrial. While there have been reports describing several additional industrial mental epidemics (3-9), these cannot reflect the frequency of these outbreaks since there is no agreement on defining outbreaks, nor do we have any systems for detecting them.

Which phenomena to include depends to some extent on the purpose of the investigations, as well as the orientation of the investigator. Economic factors are important in industrial epidemics, because of costs both to employers and employees. Other mass outbreaks are costly in terms of money, life, and comfort to society. At this point we may wish to limit our concern to phenomena appropriate to the epidemiologic model of investigation. Furthermore, as a physician, I prefer to limit investigations to those behaviors in which medical concepts are likely to be helpful.

There are two areas where I feel medicine, particularly epidemiology, neurology and psychiatry, can contribute to understanding mental epidemics. These are clinical diagnosis and epidemic investigation, and it is to these subjects that I wish to address the remainder of this report.

"Clinical diagnosis" implies the medical model of disease. That we are labeling the subjects as sick, and that they are thereby relieved of some of their usual responsibilities at

work and at home, are part of this medical model (10). Conflicts within the medical model, however, particularly in psychiatry, and conflicts between the medical and other investigative and social models, can retard our progress in understanding behavioral phenomena.

Diagnosis implies the existance of an entity termed a disease. The disease concept dates at least as far back as Hippocrates. Over the ages it has sometimes conflicted with, and sometimes included, possession by evil spirits. We usually credit Sydenham, in the 1600's with putting the concept of specific diseases to scientific use in psychiatry (11). Identification of a cluster of signs and symptoms as a specific diagnosis helps the physician select the treatment associated with the best prognosis. The diagnoses that are useful for treatment sometimes, but not always, are also useful for prevention. The latter usually require better identification of etiologic factors.

While diagnosis in this scientific sense is not exclusive to medicine (e.g., mobile and appliance repairmen also use The Term), the social consequences of the medical model are unique. Someone takes over many of the patient's functions because we believe the patient cannot do them for himself, even if he wants to. The question of whether the patient really wants to function is particularly troublesome to psychiatry. Subjects in a behavioral outbreak, for example, cannot, or do not, work, because they and we have labeled them "sick".

By labeling someone a patient we usually imply that he is not responsible for the illness. As we recognize that such behaviors as cigarette and alcohol use are determinants of a wide variety of diagnoses, it becomes difficult to know where a patient's responsibility should begin, and to what extent a patient's illness is of his own making. When is a patient using illness to escape from unpleasant responsibilities? These questions, which psychiatrists deal with more than other medical specialties, are part of the diagnostic problem of behavioral epidemics.

The medical model, in addition to conferring the sick role on the patient, confers a special role on the physician and a special relationship between patient and physician. The model assumes that the patient is seeking help, and that the physician is his helper. The model suggests that the patient can trust his physician and that he can reveal his private thoughts and other parts with assurance of confidentiality. Unfortunately, in psychiatry, in epidemiology, and in occupational medicine, the physician should not be trusted because he wears two hats. In psychiatry we sometimes are helping families and/or society deal with troublesome patients despite the wishes of the patient; in epidemiology, tradition allows

us to quarantine patients despite their wishes; and in occupational medicine we may certify a patient as able or unable to work despite his feelings. We are not, in these circumstances, the advocate of the patient; we are no longer following the traditional medical model, and it seems ethically and conceptually necessary for this to be clear to the patient, physician, family, industry and society.

With this understanding of our diagnostic model, we can examine the diagnosis of mental epidemics. I prefer the term "mental epidemic" to psychogenic epidemics, hysterical epidemics, or behavioral epidemics. In my 1973 review (1,P.359), I described my concern that the meaning of "hysteria" has a tendency to wander; although studies have indicated that persons in some mental epidemics score fairly high on the MMPI hysteria and related scales when tested retrospectively (12), the diagnosis of hysteria in psychiatry is based on additional clinical, historical, and social information. The diagnostic manual which has been used by psychiatrists in the United States since 1968 divides hysteria into hysterical neurosis (conversion type and dissociative type), and hysterical personality depending on how much anxiety is associated with the characteristic symptoms, how enduring the traits have been, and other poorly defined factors (13).

Even if the epidemic behaviors are by people psychiatrists might have called hysterical in the past, they are less likely to call them hysterical in the future.

The third edition of the psychiatrists' diagnostic manual, which will probably be put to use this year, avoids using the term hysteria(14). For example, it classifies what formerly was called hysterical neurosis, conversion type, as Somatoform Disorders, Conversion disorder. By using multiple diagnostic axes, it makes it possible to diagnose concurrently both an acute disorder such as those that are likely to occur in epidemics, and more enduring personality characteristics that might predispose toward epidemic behaviors.

The term "psychogenic" to describe these outbreaks seems somewhat premature, and perhaps misleading. We are most likely to reach a useful understanding of these outbreaks if we assume that they all have significant social, somatic, and psychological determinants. To select a term that focuses their genesis only on the psychogenic determinants, given our current state of knowledge, may simplify at too great an expense of accurancy. "Behavioral epidemics," on the other hand is a term quite similar to my preferred term, "mental epidemics," since it is descriptive and not etiologic. The outbreaks we are studying, and even some of those we are not studying, are all associated with behaviors. The word "mental", however, includes affective and cognitive factors, such as mood changes and thought disturbances, which are as quantifiable

as behaviors and may be at least as important in diagnosis.

Since etiologic diagnosis requires an understanding of etiologic factors, how do we diagnose a condition while we are trying to identify these factors? While the most common expedient has been to use diagnostic labels which merely describe the outstanding disease signs and symptoms, often it is difficult to decide which signs and symptoms cluster together consistently enough to warrant even a descriptive diagnostic label. In my 1973 review, I did not attempt to classify the 100 outbreaks by etiology, but instead described each outbreak, to the extent possible, by the following six different independent axes: (1) behaviors likely to be voluntary, (2) behaviors less likely to be voluntary, (3) observed somatic effects, (4) somatic symptoms, (5) beliefs and mentation, and (6) emotions. It is my impression that describing mental outbreaks with these six axes allows classification without premature etiologic labeling.

Investigations of epidemics have implicitly made diagnoses on two levels, the individual and the group. For the individual, the necessary psychiatric interviews need not be mystical or unsystematic, since recent efforts have standardized techniques both for data collection (15-16) and research diagnosis (17). While these efforts still do not match the reliability and simplicity of standard psychological tests, they are an important new route through which the medical model of disease is being translated to science in the field of psychiatry.

The medical model, applied to epidemics, requires that individuals in the same epidemic have the same diagnosis. An outbreak in which patients display a variety of symptoms and behaviors can contribute to information on psychiatric diagnosis, since the occurrence of different symptoms concurrently in different individuals is most easily explained by their being different forms of the same diagnosis.

Analogy with infectious disease epidemics suggests that individual diagnoses can be used to characterize the group outbreak, but that additional group descriptors will be necessary. Just as an epidemic of typhoid fever (individual diagnosis) should also be described on the basis of group and environmental characteristics (e.g., milkborne, explosive onset, institutional setting, etc.), so we need to consider group factors in mental epidemics. Whether person to person or common source, whether in communities or institutions, are group descriptors appropriate for outbreaks of both behavioral and somatic diseases.

Probably new group descriptors will be needed for adequate mental epidemic description. Studies of group dynamics, for example, suggest that groups tend to pass through different emotional phases. Bion has labelled these phases by the

basic assumptions prevailing at the time in the group (18). His fight-flight phase, during which group members unconsciously act out hostile impulses through aggressive or flight behavior, resembles in some ways the flight behavior of some mental epidemics. Monitoring Bion's variables through longitudinal studies of occupational groups might discover characteristics useful for diagnosis at the group level which could help predict and prevent behavioral outbreaks.

Since we are concerned about the relationship between enduring individual or group traits and transient behaviors or states, our diagnostic labeling must also distinguish transient from enduring characteristics of both individuals and groups, as is the intent of the two proposed diagnostic axes of the new diagnostic manual, DSM-III. For psychiatric conditions which were transient and not associated with recognizable mental conditions, the older diagnostic scheme, DSM-II, included a heading "transient situational disturbances," a noncommital term which would accurately but unhelpfully describe most individuals in mental epidemics. In DSM-III the most comparable category will probably be "adjustment disorders," which includes subcategories for disorders with depressed mood, anxious mood, physical symptoms, and disturbance of conduct.

One of the traditional uses of epidemiology has been for completing the clinical picture by describing disease prior to the time people would generally present themselves to physicians for help with symptoms. Epidemiologic studies in psychiatry, for example, indicate that most of the general population has at least some emotional impairment. The Mid-Town Manhattan Study estimated that 81.5% of that urban population had at lease mild psychiatric symptoms (19). What is not clear is how psychiatric judgments of impairment in nonpatients from the general population relate to psychiatric diagnoses in patients. Is the disease or medical model appropriate for nonpatients? It is possible, for example, that the traits and symptoms in mental epidemics do not cluster sufficiently and are so multiply determined that major modification would be needed in traditional medical diagnostic technique?

The medical model may be with us in investigating mental epidemics in part because mental symptoms are known to result from toxic effects of certain environmental agents. Lead, mercury, arsenic, many volatile hydrocarbons, organophosphates, and an ever increasing list of suspected new chemical agents may produce a variety of psychiatric symptoms. The experience and training of a psychiatrist or neurologist may be necessary to diagnose these conditions as well as treat them. Mental epidemics, particularly in industry, have usually been diagnosed on the basis of excluding possible chemical causes. Because it is never possible to be sure all agents have been excluded, the diagnosis of most mental epidemics is never definite, and rarely early in the epidemic's course. This is

dangerous: serious toxic exposures may be labelled psychogenic even after extensive environmental and toxicity studies, and studies aimed at understanding the origins of mental epidemics suffer the inherent weaknesses of retrospective research designs. A need therefore, exists for techniques to diagnose mental epidemics, toxic as well as psychogenic, "on positive grounds" (20).

There are several research areas which may eventually provide us with positive diagnostic techniques. One is suggested by areas of agreement among retrospective investigations of mental epidemics indicating that those affected were different from those unaffected on a number of measureable characteristics. These include response patterns on the MMPI and other standard psychological tests, sickness absenteeism, and selected demographic characteristics (12). It seems unwarranted, however, to accept the results retrospectively of psychological tests, which might be influenced by the outbreak experience. Despite serious practical difficulties, prospective investigations are necessary to determine whether psychological findings can predict who is susceptible to psychogenic illness. If they can, their use on a universal basis in epidemic-susceptible industries might be warranted.

Data on absenteeism and demographic patterns of affected subjects, since they are based on preexisting records, are not likely to be distorted in retrospective studies. The data suggest the possiblility that mental epidemics and absenteeism in industry and perhaps in schools as well, are closely related phenomena. The two phenomena share a number of epidemiologic characteristics. Both seem to be more common in women(21), in low socio-economic groups(22), and in those dissatisfied for a variety of reasons with their jobs(23). There are ethnic, cultural and/or racial differences in both mental epidemics and absenteeism, (21,23), although data are not available to determine whether the patterns are similar in the two conditions. Factors which have been associated with sickness absenteeism which should be examined for mental epidemics include complexity (but not time duration or distance) of transportation to and from work(23); prevailing unemployment rates(23,24); availability of sickness benefits (23,24); association with mortality patterns(24); national and geographic patterns (24,25); season of the year; such habits as alcohol and cigarette use and abuse; and perhaps even episodes of air pollution(25). Knowledge of the individual characteristics of high risk subjects and about the seasonal, geographic, and socioenvironmental patterns of outbreaks would contribute toward earlier positive diagnosis of psychogenic illness.

Some additional diagnostic help is likely to come from two other areas of psychiatric research. In the last few years there has been confirmation and extension of genetic studies originally published decades ago(26). These make it likely

that there are major biological determinants to most forms of schizophrenia(27), many forms of depression (28), many kinds of sociopathic behavior(29), alcoholism(28), and selected other mental disorders.

Biochemical measurements may offer a second source of diagnostic help for psychiatric conditions. Specific syndromes of schizophrenia and affective illnesses are being associated with alterations in levels of neurotransmitter metabolities in the blood, urine, and spinal fluid and in levels of selected cell enzymes. These biochemical alterations are likely to be useful for diagnosis and treatment.

Schildkraut et al., for example, proposed a biochemical classification of depressive disorders based on measurement of urinary excretion of 3-methoxy-4-hydroxy-phenylglycol, epinephrine and metanephrine, together with platelet monamine oxidase activity(30-31). It seems quite possible that biochemical measurements may soon be available for estimating susceptibility to syndromes associated with mental epidemics, and possible even for distinguishing these syndromes from others caused by environmental toxins. Furthermore, toxicological studies of the effects of heavy metals and organic compounds on neurotransmitters, enzymes, and metabolites may provide techniques for earlier and more complete detection of outbreaks of mental symptoms associated with these chemicals. Since the need for positive diagnostic techniques is so critical, wherever possible mental epidemic investigations should include studies of neurotransmitter metabolites and platelet monamine oxidase activity in both subjects and controls. Available research diagnostic techniques should be applied to individuals affected by mental epidemics as early in the course of these outbreaks as possible so that clinical and biochemical features that might distinguish symptoms resulting from toxic exposures and those from emotional stresses can be identified.

Mental epidemic diagnosis requires knowledge of the determinants, or etiologies, of these outbreaks; useful diagnoses are those which use a minimum of signs and symptoms to indicate etiology and/or predict outcome with and without treatment. Research into causes and treatment of disease provides information which modifies and improves our diagnoses. Ironically, our success with the treatment and prevention of infectious disease resulted in almost a century of over-simplistic medical thinking about etiology. Infectious diseases were labelled with the name of their toxic agent or parasitic organism. This was considered their etiology, and other social,psychological, and biochemical determinants were often ignored. Knowledge about the complexity of chronic disease, together with greater understanding of acute and infectious diseases, has created a greater appreciation of multiple causality in medicine. Smallpox was caused not only by a virus, but also by

a great variety of factors which contributed to both susceptibility and exposure. Human rabies requires penetration of human skin by a warm-blooded infected animal, and those factors which lead to such penetration are as essential to development of the disease as the virus that must be introduced; so also are those factors that might be responsible for neglect of preventive immunization following the viral introduction. Since there are multiple etiologies to all diseases and all phenomena, our task in prevention is to discover etiologic factors which can be modified or eliminated.

Over the centuries, there have been many theories proposed on the determinants of mental epidemics, but relatively little information available to test them. To translate intellectual theories into practical diagnoses and health programs requires that data from reproducible studies be systematically reported and classified.

Suspected etiologic factors can be classified along many axes. By duration of actions, for example, we have, at one extreme, enduring traits of an individual, such as intelligence and personality, or enduring traits of the epidemic environment, such as noise or psychological stress; at the other extreme are such epidemic precipitants as an index case fainting(35) or the radio broadcasting of a threatening message(36). Some of the general rules from infectious disease epidemiology, such as the relationship between slope of epidemic curve and time of exposure to the precipitating agent, probably pertain to most mental epidemics as well(37).

Another axis for classifying determinants or etiologic factors in epidemics is location in the host or in the environment. Suspected mental epidemic determinants in the host include age and sex, and in the environment, nature of industry, season of year, and toxic exposures.

Using both the duration-of-action and host-environment axes together, we can describe enduring host factors, such as sex, youth and history of absenteeism(12). An example of a precipitating host factor, which is difficult to distinguish from the epidemic phenomenon itself, would be the fainting of an "index case."(35) Enduring environmental factors that have been suggested include noise(12) and various forms of psychological stress (4,38). Environmental precipitants include toxic chemicals known and suspected of producing mental symptoms, as well as threatened and real changes in the work environment. One of the reasons that diagnosis of mental epidemics by exclusion is unsatisfactory is that the list of possible environmental toxic precipitants is increasing because of both the rapid development of new industrial chemicals and increased suspicion about possible effects of chemical and exposures which have long been with us. While epidemiologic evidence is lacking, some clinicians suggest that the list of environ-

mental agents precipitating mental symptoms should include common hydrocarbon fuels, refrigerants, rubber, plastics, textiles, perfumes, tobacco smoke, and many other substances quite common in both industrial and domestic environments(39).

In additon to duration-of-action and host-environment, such traditional etiologic axes as the biochemical or biological class of an environmental agent are important contributors to developing practical mental epidemic diagnoses.

At present these diagnoses do not exist. Since the medical approach to disease control depends on them, it is unclear whether medicine can make a significant contribution to mental epidemic investigation.

How the data were selected and how they were recorded have had major influences on the scientific value of investigations of mental epidemics. Selection of data depends partly on the investigator's hypotheses, and partly on the availability of scientific methods for research design, measurement, and date analysis. Based on the latter, the first era of investigation can be called prescientific. Until quite recently most descriptions of mental epidemics were unsystematic. While Hecker, in his 1859 treatise(33), placed bacterial epidemic disease on the same investigative basis as the dancing manias, contemporary descriptions of bacterial epidemics were far more objective and productive than those of mental epidemics. John Snow's 19th century analyses of cholera in London(40) still match in objectivity any investigations yet made of mental epidemics.

One of the earliest scientific studies of a mental epidemic was the investigation published by Leake et al in 1934 of an epidemic of poliomyelitis in California(41). While solidly diagnosed cases of paralytic poliomyelitis did occur in this population, there were a large number with mental symptoms who did not develop paralysis. Twenty-six years later McEvedy and Beard(42) suggested that the disease in this subgroup had been psychogenic.

While it is difficult to settle the nature of this outbreak retrospectively, the objective and quantitive descriptions of clinical and epidemiologic features make this 1934 investigation an important milestone. While more recent mental epidemics are still initially investigated as outbreaks of organic disease, the time between epidemic and investigation for psychogenic factors has shortened below the 26 year interval of the 1934 outbreak.

Of those phenomena I consider mental epidemics, a few were never considered organic diseases and yet received systematic investigation. Perhaps the earliest of these was the panic reaction to the October 30, 1938 radio program, Invasions

from Mars. Cantril, a social psychologist, conducted extensive investigations, which were published in 1940(36). His field work applied research techniques which are similar to those in a number of subsequent epidemic investigations.

The first systematic investigation in the United States of an industrial mental epidemic with which I am familiar occurred in June 1962 in Spartanburg, South Carolina. Psychological investigations began after a two-month delay while organic causes were first investigated. The results of the investigation were published three years later(43). While I am not aware of an industrial mental epidemic which was investigated from onset as psychogenic in origin, an outbreak among school children in Louisiana in 1962 received an early psychogenic diagnosis, with organic and psychological investigations conducted concurrently (44). Precedence has therefore been established for systematic mental epidemic investigation in both closed and open population groups, and for such investigation to be initiated early enough to permit concurrent psychological diagnostic investigation.

Another trend in mental epidemic investigation relates to administration, and to the speed with which an investigation can be initiated. In the United States, acute infectious disease investigation is usually the responsibility of local and state health departments, with consultative assistance available from the federal Center for Disease Control. With this network it is possible to investigate outbreaks soon after they are reported, using protocols developed in advance.

This network can be used to investigate mental epidemics, although the absence of a detection or reporting system for mental outbreaks leaves us without information on their frequency or distribution. For this reason, while directing the Center for Epidemiologic Studies of NIMH during 1969-1974, I made several efforts to develop surveillance programs for detecting short term variations in community mental health. Portions of these programs may also be applicable to monitoring mental health in occupational settings.

We piloted three different kinds of community monitoring systems: Continuous community surveys, continuous analysis of existing community behavioral data and the monitoring of mortality relevent to mental health. The community surveys monitored fluctuations in selected psychological variables using weekly samples of the general populations of Kansas City, MO., and Washington Co., MD. (45). Weekly household samples of 20 to 30 interviews were conducted for more than a year in each community. While we examined the weekly fluctuation in scores on a variety of depression and psychological scales, preliminary analyses found no significant deviations from mean levels that might have suggested the presence in these communities of psychological "epidemics." Unfortunately,

these unusual data have never, to my knowledge, been throughly analyzed or published. The extra cost of including independent weekly sampling in a community survey was difficult to justify when there are so little data available on the frequency of epidemic phenomena. The lack of a significant finding, therefore, may be the result of inadequate sample sizes, insensitive psychological scales, infrequency of abrupt psychological changes in the communities under study, or incomplete analysis of the data.

A second program initiated by the Center for Epidemiologic Studies at the University of Texas School of Public Health to examine the frequency of mental epidemics in the community was a collaborative effort to examine changes over time in what we termed existing community data. This project collected and examined the frequency over time of selected events which reflected human behaviors in the city of Houston. These events included violent deaths, false fire alarms, emergency ambulance dispatches, emergency room visits, visits to psychiatric hospitals and clinics, calls to several hotlines, and police data on several different crimes. Available data on possible determinants of these behaviors such as weather, air pollution, economic indices, unemployment, and unusual community events, were also collected. A number of unusual occurrances were noted over the 2-1/2 year period of data collection (July 1, 1971 - January 15, 1974). These included several days with unusual numbers of homicides, of suicides, of psychiatric hospital admissions, of police broadcasts, emergency ambulance runs, and of accidental deaths. Funds were inadequate to investigate these occurrances individually, although for several of them the investigators were able to look at relationships among the data sets they were routinely collecting. A day with an unusual number of homicides, for example, was associated with a community threat of a hurricane and with reduced police activity, low total mortality and mortality from suicide, and low number of false fire alarms and arson suspects. The analysis of these data is still incomplete. While the financial needs of this project were considerably less than those of the two-city community studies, funding was difficult to obtain because these community data do not seem so directly relevent to community mental health.

The third program for surveillance of community mental health compared the frequency of emotional problems in people who had died with that in the living population. Any excess frequency of emotional problems among decedants is a quantitative measure of community mortality associated with emotional problems. Monitoring fluctuations in this association might detect mental epidemics associated with excess mortality. While the field work was piloted in 1970-1974 in Alachua, Co., FLA.(46), the data collected were based on too small a sample to warrant analysis for short-term fluctuations.

None of these three pilot efforts to develop data systems which might determine the true frequency of community mental epidemics was prepared to investigate any epidemics that it might detect, or that might come to the attention of state or federal health agencies. While CDC expressed an interest at the time in developing an investigative plan for behavioral outbreaks, I left the Institute before any such plans were developed.

Mental epidemics in communities may have greater potential public health significance than those in industry, since they can affect a greater number of people. Yet it is also possible that they are less frequent and less serious. The greater number of community epidemics described in the literature, in contrast to industrial epidemics, is no indicator of greater frequency. Furthermore, the closed nature of industrial epidemics the more amenable to investigation. We, in fact, see that NIOSH has succeeded in developing investigative plans for psychogenic illness where no such plans exist for community outbreaks.

While the existence of investigative plans provides the potential for early investigation of outbreaks and the possibility of early positive diagnosis, the existence of these plans does not address the problem of epidemic frequency. From a public health point of view, I believe our most pressing need is to determine the frequency of mental outbreaks both in industry and in the community. The convening of this symposium suggests that efforts in industry might be more successful than those in the general community.

I am assuming that epidemics are occurring that never appear in the literature, and that those that come to our attention are likely to be nonrepresentative. They may be the more serious or they may be occurring where administrators are more concerned, more curious, more brave, more enlightened, or perhaps under more employee pressure. Several mental epidemic investigations have described the power of suggestion (1), and we must be aware that it affects industrial hygienists and epidemiologists as well as employees. The existence of the NIOSH investigative team has created an outbreak of epidemic investigations. It may even create, through widespread publicity, an actual increase in outbreaks. It is essential to determine the frequency of outbreaks not only to evaluate their public health importance, but to evaluate the possible effects of our investigative efforts themselves.

Although our efforts at developing community surveillance techniques have as yet not been successful, there are characteristics of industry that suggest a more promising outlook. One factor is the possible relationship between absenteeism and mental outbreaks. Analysis of absenteeism records is unobtrusive and relatively inexpensive. The data needs to be examined to determine whether absenteeism clusters in time and

and place and kind of work. Should clusters exist, we need to describe the nature of the symptoms. A study of absenteeism might avoid some of the resistence both employers and employees have to studies suggesting psychogenic origins to physical complaints.

In addition to a monitoring system to determining frequency and distribution of outbreaks, priority should be given to prospective studies in selected high-risk industries. Since it may not be evident which industries are at high risk, these studies may need to await the analysis of absenteeism. The purpose of these prospective studies would be to identify and measure, prior to the outbreak, characteristics of those affected during mental epidemics. It is possible that some of this could again be done with existing medical records as part of the above absenteeism study; the major limitation to use of existing records is the desirability of psychological and biochemical tests which are unlikely to be routinely available.

We have no knowledge of the frequency of mental epidemics in communities and no plans I am aware of for developing monitoring systems to detect them. While CDC may be available to investigate outbreaks for which their consultation is requested, we should be prepared to investigate promptly any epidemics that come to attention. The experience NIOSH gains from investigations in industry may help develop protocols for community investigations so that the necessary data can be obtained as early as possible in the course of an outbreak.

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ILLNESS AS AN EMERGENT NORM

OR

DOING WHAT COMES NATURALLY

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The sudden occurrence of outbreaks of sociogenic illness has only recently begun to receive scientific attention. Few reports have been published in the literature despite the apparent relatively high prevalence of such events. This paper is an attempt to use social psychological factors and theory to understand such occurrences and to provide a set of theoretical explanations which can be generalized to other such events. To this end, Labeling Theory, Emergent Norms and Coping Theories are used. Examples from a variety of such events are used to integrate the explanation.

The intent of this manuscript is to describe the occurrence of an outbreak of illness among a group of workers in a data processing center of a university and to attempt to explaine this outbreak in social psychological terms. The use of social factors for an explanation is critical since no apparent biophysical agent could be found for the occurrence of the event. In addition, I hope to begin the development of a theoretical model for the explanation of such phenomenon which have a prevalence apparently greater than reported in the scientific literature. To present this explanation, I will attempt to draw on several such occurrences already reported in the literature and to weave a theoretical fabric which can be used in additional such outbreaks. The specific event in the Data Center has been published elsewhere (Stahl and Lebedun, 1974) and will be briefly reviewed below.

At the outset, let me place a caveat of this attempt at explanation. The orientation of my discipline, medical sociology, is one of explanation or understanding rather than amelioration. Hence, while I hope that this work is "relevant" to the problem at hand, I do not normally do my research in an industrial setting and do not have to concern myself with those problems which you psychologists, environmental scientists, physicians and managers must adjust to. I'll try to be of help but you must realize that my concern is more with explanation rather than cure. In this work, I am more concerned with explaining the sociogenic nature of the events than with deriving a solution for helping the worker or the management of the industry solve the problem.

The event to be described happened in the Data Processing Center of a major midwestern university. The town's only industry is education, thus limiting other employment opportunities of the Data Center workers. The sort of work done in the three story building consists of key punching, and verifying records of all university salaries, purchase vouchers, student records and grades, and other routine university business. Because of the nature of the Data Center's work, few outside persons entered the building and the work was highly routinized. All affected workers were housed on the building's first floor in a single large room with about 30

old and noisy keypunches and an equal number of verifying machines.

The organizational structure of the keypunch operation is important to the event. The Data Center supervisor is housed on the second floor with the computers and other managerial personnel while the person directly responsible for the keypunch operation and its 35 workers sits in a glass enclosed office above and in the corner of the large and open keypunch room. From this vantage, she can observe all activity in the room. Directly under her supervision are two assistant supervisors who do some keypunching and verifying, the assigning of tasks and are expected to report any problems among the women who work in the room to the supervisor. One assistant supervisor is responsible for the keypunching operation, the other for the verifying operation.

Also important is the fact that the workers are expected to not converse during working hours about non-work-related matters except at two 15 minute coffee breaks during the day and at lunch time. The coffee breaks are pre-assigned and staggered thus precluding interaction between friends except for those accidental periods during which two or more friends are assigned the same break. Even trips to the toilet were a subject of some speculation on the part of some of the workers; one worker expressed the feeling that the supervisor was "aware" of the frequency of use.

While there was no contact with the public on the part of the workers, the building supervisor dictated a rather strict dress code for his female workers. This was mentioned frequently by the workers who felt that the poor pay did not warrant the purchase of "business clothes" and that they could do their job just as well in any reasonable form of attire.

The occurrence of the "Mystery Gas," as it was headlined in the community's newspaper, took place on a Wednesday and Thursday in the early spring. At about mid-morning on Wednesday, a majority (exact prevalence figures are only estimates) of the 35 workers in the keypunch room became ill with symptoms which included headaches, smelling a "strange and burning odor", hyperventilating, nausea, vomiting, burning and tearing eyes and fainting. Ten of the workers were taken to the university hospital"s emergency room either by ambulance, by other workers or by supervisory personnel. The Data Center was closed for the day and environmental scientists began the task of taking multiple air samplings.

On Thursday, the workers reported back to work and within one hour of opening the symptoms recurred. The building was again closed, workers returned to the emergency room or went home and the team of environmental scientists again performed air sampling through the day and well into the night.

The building was reopened on Friday. Prior to the beginning of work the supervisory personnel, a physician from the emergency room and the professor in charge of the environmental studies met with the workers to assure them that whatever the problem was, it had now passed. They did not specify precisely what had caused the strange series of events, but suggested that an "atmospheric inversion" had caused smoke from the adjacent university power plant to accumulate and descend upon the Data Center. It is critical to note that they did not tell the workers that the physiological evidence from the blood and urine testing and the air sampling produced no evidence of the effects of the hypothesized "atmospheric inversion". The workers accepted this explanation and work resumed in the Data Center with only one worker experiencing symptom recurrence later in the day. Incidently, the one worker who began to hyperventilate was quickly isolated and sent home before the others could react to her symptoms.

It was on this day that we were able to interview the workers who returned to work. Unfortunately, about one third of the workers absented themselves on this day; however, from reports of other workers, the symptomatology of those absent was no more or less severe than those who did return to work.

The work of Colligan and Murphy (in press), that of Kerckhoff and Back (1968), Knight et al. (1965) and Levine (1977) all indicate that outbreaks of such events tend to follow some stressor event. This was clearly the case in the event which we observed. The university was in the process of building an addition to the adjacent power plant. Thus, heavy machinery was constantly being used and there were unanticipated (at least to the workers) dynamite blast of bed rock throughout the day for a two week period prior to the event's occurrence. During the time which we were in the building conducting this aspect of the study, at least four such blasts were heard and felt; the experience was at best disconcerting. One worker expressed the idea that she should be receiving a salary bonus for "hazardous duty." Furthermore, workers were being asked to work overtime, which many expressed as being a burden since most were married, several had small children and few liked the job.

The most immediate event which seemed to trigger the outbreak was the moving of a large diesel engine under one of the open windows of the Data Center to run several pneumatic hammers. While those women initially affected by the "mystery gas" were distributed throughout the room and not necessarily near the open window, the majority of the workers mentioned the presence of the engine. The environmental studies, however, were unable to detect noxious fumes from this source.

It was during this Friday that a then graduate student of mine (now Dr. Lebedun) spent the entire day in the Data Center

interviewing privately the workers and supervisors. The interviewing was both open-ended and by means of some quickly constructed interview schedules which had face valiadity. We were interested in testing several hypotheses, including the relationship between work satisfaction and symptom severity (we suspected an inverse relationship), the empathy between the supervisor and the worker, and the worker's level of job satisfaction (a positive relationship was expected, and found), and an exploratory hypotheses regarding the manner in which the symptomatology spread throughout the building. Our interviewing lasted from the beginning of the work day until about four P.M., when the building supervisor confronted us with the statement that he thought what we were doing was not advantageous to the morale of the workers (although workers were almost too anxious to talk with us), that our interviewing (which lasted for about 15 to 30 minutes per worker) was disrupting his ability to complete the day's work, and that we had probably collected "enough information".

We found that those workers who were most dissatisfied with their jobs were the ones most likely to report the most severe symptomatology. Those who fainted and vomited were invariably the persons who had attempted to look for other work or expressed the highest levels of dissatisfaction with the work conditions, the nature of the relationship between the supervisor and the workers, and the restrictive dress code. These were also the persons who made statements such as suspecting that the supervisors kept track of the number of times the toilet was used and were too punitive regarding the use of the pay telephone in the building. It is interesting to note that one of these workers had mentioned to her immediate supervisor that she was looking for another job and was verbally chastised by the first floor supervisor.

In addition, there was a strong association between the supervisor's perception of the worker's personality and the worker's level of job satisfaction. The supervisor was adequately "reading" the worker, at least in this regard, and the worker knew it. Fascinating was the fact that at no time was the worker's competence as a keypunch operator ever mentioned by the supervisor. All judgements seemed to be based on the worker's personality.

Perhaps the most interesting finding is the fact that persons who considered each other friends in the Data Center, shared symptoms. The construction of a sociogram, that is, asking each worker to select other workers who they considered as friends, or to select those other workers whom they respected or talked with most frequently, indicated that the four women in one of the sociometric networks all fainted and that no other worker in the Center fainted. Similarly, the five workers in another sociometric network were the only ones who vomited;

no other workers shared this expression of the "mystery gas." In the same manner, two women, one a foreign person who spoke little English but had prior keypunching experience, said they became agitated when they saw the events occurring around them but experienced no symptomatology. They mentioned no friends in the Data Center and no one mentioned them in their sociograms.

Further elaborating this point, it is interesting to note that the workers were highly aware of the temporal order of events. Statements such as "first Sally started breathing funny and Mildred's eyes started watering," or "Jane vomited and then I noticed that Ann also vomited and then I had to vomit," were made. The symptoms were essentially "managed" in such a manner that many workers were able to verify who got what, when and where, suggesting that the idea of "social contagion" (Milgram and Toch, 1969) was clearly operative in this network.

Several of the above findings require elaboration and explanation. My intent here is to attempt to explain in theoretical terms those events which were most significant and which should have some utility for understanding future outbreaks of such "mass" epidemics.

1. Sex: One of the most persistent variables in our study as well as in other studies is that only the women in the Data Center were affected by the "mystery gas". Explanations for this, at least in the incident which we studied, might be that only women were on the first floor of the building and that the men working in the building had both more varied and potentially interesting jobs. Thus, in the case under study here, we do not have an adequate control group.

However, in virtually every study of similar occurrences, the vast proportion of persons affected are women. Levine (1977) reports that 57% of those fainting in high school band were girls; Knight, et al. (1965) reports that all but one student who fainted in a southern school were girls; Kerckhoff and Back (1968) report almost all of the affecteds were women in the Southern textile plant which they studied. In fact, in Colligan and Murphy's (in press) review of 16 separate incidents which they found in the literature or for which they had access to the data, 93 percent of the affecteds were women. There are several potential explanations for the preponderance of females found in the affected category. While somewhat interesting, the two most frequently found in the literature seem to beg the question.

The first explanation indicates that men are more goal oriented than women (Colligan and Murphy, in press) and despite psychological strain, men are more likely to view the work task as a means to an economic end and therefore will ignore the

strain and will not express that strain in terms of a stressor reaction, such as illness. In this rather sexist explanation, women are said to be more "emotional" or to better express their feeling states whereas men are better able to control their emotional releases. The explanation strikes me as being not only sexist, but as being unsupported by empirical evidence. The goal orientation supposedly more frequently found in men does not explain why women can run a sewing maching as well as men (Kerckhoff and Back, 1978) or play a wind instrument in a band (Levine, 1977) as well as their male peers. Goal orientation, by definition, is related to the task at hand, not some nebulous, ill-defined component of the environment.

A second explanation often posited is that for women, the work role is secondary to the wife/mother role. This supposedly provides a rationale for women seeking an easy role release from for-profit work roles. If you have read the brilliant sociological analysis by Study Terkel in his book Working (1972) or have read the work of Smelser (1963) and Bendix(1956), you quickly discover that working is not all that it is cracked up to be for the average person on the assembly line. Work is a place to be for eight hours a day and both males and females seem to dislike repetitive tasks equally well. This is further supported by the fact that hourly employees miss more work due to illness than do highly salaried professionals and that lower income adults use health care services more frequently than higher income adults (Monteior, 1973). The fact that some females have another role to fulfill outside the work force does not mean that they are more likely to report illness than men to seek secondary advantages.

Perhaps more to the point regarding the female preponderance in the prevalance of affecteds is the fact that women have transitionally known how to use the health care delivery system in more appropriate ways than men. The latest data available from the National Center for Health Statistics (1979) indicate that 47 percent of the women and 38 percent of the men in the United States had a "general checkup" within the last year; 80 percent of the females and 70 percent of the males saw a physician within the last year. While use patterns are higher for women than for men, death rates in the two groups, even controlling for age and especially among those still in the work force are now and consistently have been lower for women.

Thus, women have been more adequately socialized in the use of the health care delivery system and it is more socially appropriate for women to use the system whereas for men, use of the system for complaints other than those perceived to have low commonality or thought to be serious is not a socially sanctioned behavior. It is not surprising then, that in light of a stressor event, we find women more frequently somatocizing the stress than we do among men.

Phillips and Segal (1969) make this point and conclude that women are more willing to discuss illness than are men. This of course, leads to the greater prevalence of women in data on the number of physician visits per year and suggests the appropiateness of women using illness as a socially legimate role release. Since the avenue of illness is appropriate, then it is more likely that they will use that avenue to provide perfectly legitimate releases from various social roles, such as the work role, which they find, for whatever reason, to be situationally problematic.

Phillips (1964) makes the same point in a different manner. He states that males are more stigmatized, or negatively sanctioned, than are women for using various forms of behavior, such as illness, as a role release. Thus, again, the social system sanctions the use of illness as a role release for women, whereas for men, a similar role release is more negatively viewed.

2. Socioeconomic Status: Like the variable sex, it is no random accident that most of the studies reported in the literature involve persons of lower and lower middle socio-economic status. Kadushin (1964) reports that illness prevalence in general is probably about equal in all social classes, with some obvious exceptions, but that the lower status individual "feels" more sick than does his/her more middle class counterpart. He postulates that in the lower classes anxiety is more frequently expressed in physical terms than in emotional terms. This may in fact be because the shared vocabulary of psychological distress is found predominantly in the middle class; psychiatry, much to its collective chagrin,has become a middle class service phenomenon.

Crandel and Dohrenwend (1967) share this perspective in their excellent study by relating that lower class individuals express the same types of psychological distress in somatic terms while middle class individuals express the same types of psychological distress in psychological terms. This conclusion is also shared by McBroom (1970). Thus, it is possible that in the Data Center, those individuals who were more highly educated, one indicator of socioeconomic position, and who had the more prestigious jobs, such as the men responsible for computer programming and the coordination of all Data Center Work, may have experienced the same stressors but were able to verbalize them. The lower status workers, who were all women, reacted in a pattern of somatocizing their distress.

3. Illness as Response to Strain: In the "mystery gas" experience described earlier as well as in virtually all events reported by Colligan and Murphy (in press), some form of precipitating event triggered the outbreak of the illness. The event in the Data Center consisted of the moving of a diesel engine under one of the windows following a long

period of intense noise, dynamite blasts, the strain of imposed overtime and a very unpopular management style. One has the impression from this situation as well as from many of the other situations studied, that those persons in the affected category felt trapped by an intolerable structure and that something had to give. Lest you get the impression that this is a Freudian interpretation, let me explain.

There is in the area of social epidemiology, a long tradition of research and theory attempting to relate social factors to the onset of physiological symptomatology and disease. Some of these studies include the following: change observed in serum uric acid and cholesterol levels in men undergoing job loss (Kasl, et al., 1968); coronary heart disease attributable to occupational stress (House, 1974); normal life events seen as stressors leading to a catalogue of morbidities (Dohrenwend, 1973); spouseless motherhood and its relationship to elevated levels of physical and psychological incapacity (Berkman, 1969); the relationship between various psychosocial factors in the prevalence of such disparate signs of hypertension and lower back pain (Backus and Dudley, 1974); and social change leading to self-perceived disease in a Dutch population (Aakster, 1974).

These empirical observations exist within a broader theoretical underpinning although the various theoretical structures used to explain them are as varied as are the findings themselves. Most critical among the explanations are those by Hans Selye (1956), Moss's brilliant statement on Illness, Immunity and Social Interaction (1973) and the work of the Dohrenwends (1974). Generally, these statements postulate that the extent to which the social environment is seen as stressful will lead to the occurrence of both illness and disease events. Thus, a positive relationship can be expected between the amount of strain in the social environment and the production of a stress, which in Sely's terms is expressed physiologically.

If, as in the "mystery gas" experience, other avenues of amelioration are blocked, illness may become an appropriate stress reaction to the environmental strain. Furthermore, and independently of the blockage of legitimate avenues of change, the studies relating social factors precipitating illness and disease indicate that a physiological reaction is a normal outcome of the presence of a stressor; that is Selye's major thesis (1956). Hence, fainting, vomiting, hyperventilating, and the group of nonspecific symptoms reported in the "mystery gas" experience as well as in other reported events, are perfectly appropriate and socially legitimate ways of excusing oneself from a bad situation.

Mechanic and Volkart (1960) further elaborate this point by finding that illnesses and symptoms which are common, relatively familiar to the group and have predictable and non-life

threatening outcomes are much more likely to be used in an attempt to gain access to the sick role. Certainly the list of symptoms reported in the various studies reviewed by Colligan and Murphy (in press) fall in this common, non-dangerous and predictable category. Hence, the sick role (Parsons and Fox, 1952) using these symptoms, is an appropriate role release. Illness is, essentially, a coping mechinism.

4. Selective coping: If we accept the proposition that illness and/or symptomatology is a copy mechanism, then it is appropriate to ask why it is that not all persons react to an obviously negative set of stressors. Some persons in the "mystery gas" experience developed no clinical signs and in fact did not even smell the "mystery gas" while others were taken unconscious to the hospital's emergency room. The same variability of reaction is found in virtually all of the events which Colligan and Murphy (in press) reviewed.

There are probably two basic reasons for this less than perfect relationship. One will be elaborated later in a discussion of the meaning of social support systems. The other explanation of the non-perfect nature of the relationship between stressor and reaction is the fact, that while on the surface, the stressor events are less than ideal situations within which to work, not all individuals perceive them as being equally problematic. The classic epidemiological model postulates that there is a linear relationship between the stressor event, or the social structure, and the disease outcome (Stahl, et al., 1975). The search for a direct relationship between agent and outcome used so frequently in our research comes from the monoetiological model of disease etiology. That is, disease can be explained by looking only at those persons exposed to a given infectious agent. While this may be a necessary relationship, it ignores the sufficiency explanation in the philosphy of science (Nagel, 1961). In some recent work on hypertension (Stahl et al., 1975) it is postulated that those individuals who perceive the structural events as inhibiting life goals are the persons who will eventually contract the disease. Translated in terms of the "mystery gas" experience and applied to a number of other studies reviewed by Colligan and Murphy (in press), it appears that only those persons who perceived that the stressors were severe, and/or socially disruptive, were the ones to react by developing some form of symptomatology. This explanation is isomorphic with the data in that those persons who were most dissatisfied with the work situation at the Data Center were also the ones to develop the most severe symptomatology. Those who used illness as a legitimate role release were the ones who perceived the structural situation to be so severe that they had looked for another job within the last year, felt that the Supervisors were grossly unfair, and generally disliked the work situation. These were also the individuals who felt that they simply had

a "job" and that their work prevented them from spending time at other important social obligations, such as their families.

Richard Lazarus (1978) expands on this thesis by applying it directly to hypertension. He postulates that coping is based on the specific environmental and psychological events and that without an understanding of the individual's ability and willingness to cope with these events, it is impossible to understand the selective reaction of the various participants following the stressor event.

5. Social Support System: Perhaps the most fascinating aspect of the "mystery gas" experience is the manner in which the illness was "transmitted" throughout the group. Transmission relates directly to the selective coping process just elaborated. Mentioned above was the fact that not all persons experienced the illness and that this might be due to the selective perception of the severity of the potentially stressor events. The lack of a perfect relationship between the stressor events and the onset of the illness may also be due to the fact that persons are less than perfectly integrated into the social support system or are isolates in the information network within the work site (Moss, 1973). In the "mystery gas" experience, it was noted that those persons who were isolated from the friendship and communication patterns within the Data Center experienced no symptoms. On the other hand, those persons involved in specific informal information and friendship patterns shared a common set of symptoms. The intent here is to explain the nature and meaning of these friendship patterns for the occurrence of the outbreak of the sociogenic illness.

Social support systems are those networks of friends and/or primary relationships which provide role models for one's behavior and which lend social credibility and positive sanctions to the behavior of the individual. Thus, a social support system might be a family, one's colleague group, or the work group within which one operates for eight hours each day. Kaplan, et al. (1977) provide a good framework for the study of the effects of social support networks. While research in this area is relatively new, it does seem to indicate that the existence of social support networks is significant in moderating the effects of stressful environmental stimulae as well as providing the appropriate types of cues for responding to those stressors (Gore, 1978).

It is this latter point that will be argued here: the availability of role models for illness provides the basic mechanism through which sociogenic illness can "spread". That is, because of the availability of social support networks among peers, the Data Center workers essentially "learned" how to behave in the stressful situation.

Turner and Killian (1975) develop the concept of "emergent norms" which can be used to explain the phenomenon of the spread or contagion of the series of symptoms observed in the "mystery gas" experience as well as in a number of the other outbreaks reviewed by Colligan and Murphy (in press). That is, in any collective behavior, whether it is an anti-war demonstration or a number of workers in a common location, milling behavior must precede any collective action. Milling behavior is such that persons circulate among one another and share their perceptions of the events which are occurring. From these shared perceptions, definitions of the situation arise which are communicated to others in the environment. These shared communicated perceptions then emerge as the basis for norms regarding how to cope or handle the situation at hand. In this way, the affected group shares a common definition of the situation and establishes a series of normative behavior patterns which are unique to the given situation. Since the situation is itself rather abnormal, the emergent norms are not necessarily congruent with the norms which exist for the larger social system. Hence, the norms which emerged in the "mystery gas" incident might have been negatively sanctioned by the supervisors but were considered appropriate and useful by those individuals trapped in the situation. A shared, collective response was established on how to react to a situation which consisted of the accumulation of a long-standing grievance addressed to the social structure and exacerbated by the intolerable (as perceived by the workers) work conditions.

Stating that a norm for behavior emerged from the situation partially begs the question of why specific illness symptomatology was chosen by the participants to express the strain rather than coping in more legitimate means (as defined by the larger social system). Colligan and Murphy's review of the various studies indicates a remarkable similarity of symptomatology. These include: headache, dizziness, weakness, nausea, burning eyes, shortness of breath, vomiting, fainting, headache, hyperventilation and skin rash. Every one of these symptoms, or more appropriately clinical signs, has either been experienced by virtually every human or if not experienced, virtually everyone knows how to imitate or recognize the specific sign. In short, there is a high commonality of symptomatology of this nature. Nothing is terribly unique in any of the situations.

To explain this commonality, it is necessary to use part of a theory called "Labeling Theory." While this theoretical construct has generally been applied to criminal behavior and mental illness, I want to be perfectly clear that there is no intent in its use to draw any analogy between those social behaviors and the occurrence of a sociogenic outbreak of illness.

In Scheff's classic work on mental illness (1966) a series of propositions supported by empirical evidence are presented. First, the various signs of mental illness, and for our purposes, the various signs repeatedly found in the outbreaks of sociogenic illness, are clearly quite common and easily learned. There is a sterotypic swooning pattern; everyone knows what being nauseated feels like and the mere suggestion of nausea triggers that same perception in others (remember dissecting your first laboratory frog in high school?). Thus, everyone learns these clinical signs. The nature of the immediate situation, being anxiety laden and very confusing to the participants forces them to look for an explanation of the immediate events. Because of the need for an explanation, the reaction of the immediate peer group becomes critical in determining both the duration and the outcome of this initially ambiguous set of stimulae. The reaction can turn to an elaboration of these easily learned and frequently previously experienced sets of symptoms.

Another proposition suggested by Scheff is that as in mental illness, there may be a reward for playing out these stereotyped behaviors. The victim now shares the experience with others thus making the behavior a collective one and reinforcing the actor's own perception of the reality of the event.

The victim now has an explanation for her own feelings of anxiety which is shared by other participants. The attention given the affected workers, while absolutely justified in such situations, further reinforces the behaviors since others in the situation are acting as though the situation were "real." It is critical here to emphasize that this statement is meant to be "value free"; a lack of reaction by others in the situation, such as the supervisors who took workers to the hospital, would be both unethical and immoral.

In this way group participants develop a collective definition of the situation where few,if any,clues to what is happening are present. General dissatisfaction, triggered by some event such as the diesel engine and dynamite blasts, produces a vague and amorphous feeling state. This feeling state requires some definition since all people understand their social world by placing events and feelings into categories. Thus, a collectively shared and understood norm emerges to define the situation and persons proceed to do what comes more or less naturally. Illness is a perfect reaction and a quite legitimate manner of defining the situation and reacting to it. Illness provides a set of parameters, easily shared by others, which can provide immediate relief from the collectively experienced situational difficulty. Furthermore, McEvedy and Beard (1973) report that affecteds report more sick days from work and a greater prevalence of hospital admissions than do non-affected workers. This implies, then,

that affecteds already know how to use the system and how to "experience" illness in a manner differently than do non-affected workers.

These explanations are further strengthed by looking again at the sociometric analysis which emerged from the "mystery gas" experience. Using the sociogram technique which asks the workers to specify other workers in the Data Center considered to be friends or persons with whom they spent most of the time, a pattern emerged which further explained the spread of the sociogenic illness. All persons within a given sociometric network shared almost identical symptoms and few workers in other networks had those symptoms. Thus, there was network of fainters, a network of hyperventilators, etc. Where one individual had multiple symptomatology, the most severe of the symptoms (usually fainting) was used as the indicator.

It is hypothesized that even within these small sociometric networks, the victims "learned" the appropriate response to the noxious situation. The norm for behavior emerged by individuals taking cues from others with whom they normally shared interaction. In this way, a study of the social relationships showed a high prevalence of "appropriate" symptomatology. Furthermore, workers within each network generally knew the temporal pattern of events which preceded their own contracting of the appropriate symptomatology. Generally, the person most frequently selected as the center of the sociometric network (called the "sociometric star") preceded other members of the network in developing the "appropriate" symptomatology, which was subsequently "shared" with other sociometric members.

This pattern more closely follows that of contagion theory rather than convergence theory (Milgram and Toch, 1969). In convergence theory, affecteds seem to develop symptoms or responses more or less simultaneously. A detailed questioning of the affected in the "mystery gas" experience indicates that the workers were quite aware of the pattern of events. Those persons who developed a response to the situation communicated these responses, during the milling phase of the collective behavior and thus a norm for the specific illness sign emerged within each sociometric group. The question remains, of course, as to why one symptom was used in one sociometric group and why another emerged in a different group. These innovations were adapted by the leaders in the situation and seemed to spread to others in the group (Coleman, et al., 1966).

The sociogenic outbreak of illness was stopped at the Data Center when sources external to the workers and with socially legitimate expertise provided an explanation for the series of events. The environmental scientists and physicians essentially ended the outbreak by declaring that the "atmos-

pheric inversion" was responsible, despite a lack of clear evidence that this phenomenon either occurred, or if it did, had any detectable physiological effect on the workers. Thus, the "mystery gas" was given a label and the behavior of the workers was legitimated.

It is rewarding to know that the supervisors of the Data Center subsequently reevaluated their management policies and instituted changes in the organization of both the work and the severity of supervision. To my knowledge, no additional problems have occurred.

Several points must be mentioned in concluding. One of these points bears on the ethical nature of such research. Without a sympathetic audience among the management of the Data Center, the results for the workers might have been disastrous. This is, since the workers were not unionized and keypunch operators in a university population are abundant, there is the possibility that these people might have been fired for being disruptive to the ongoing operation of the Data Center. In making reports exclusively to management, which Colligan and Smith (1978) state frequently happened in such situations, the investigators should remain highly sensitive to the implications of their findings. To "blame" the workers for hysteria or malingering because no toxic agent can be found is both unwarranted and unethical.

Hopefully, you have noticed that I have avoided using the term "psychogenic" illness and have instead used "sociogenic" in describing the occurrence of events. This is meant to be more than a bit of chauvinism from a sociologist. The events explained here, as well as those analyzed in other social settings, have as much group or peer input as they have psychological explanations. In fact, one of the central theses presented here is that the norms of behavior emerge from the group and that to explain the event requires an understanding of both psychological characteristics of the participants as well as an interpretation of the group dynamics which is after all, the immediate environment for the fostering of the event.

Finally, and related to the above issue, is one of the major drawbacks in all of the data reported in the literature. Looking for sociogenic or more broadly put, social psychological pathogens, in the occurrence of these events has consistently been relegated to a residual category of research. That is, if standard biochemical techniques fail to explain the event, it is only then that the social scientists are called upon. This probably results from two factors: those "in residence" who study such phenomenon are in fact, biologically or physically trained scientist and secondly, outbreaks of these events call for the immediate amelioration of the problem, not the more complicated, long-term understanding and explana-

tion of the situation which can be more readily provided by the social scientists. Dr. Colligan and his colleagues (Smith et al., 1978) are highly aware of this problem and have developed a series of social psychological instruments which can be used cojointly with biophysical techniques usually used in these situations. This development is critical for a better understanding of the occurrence of such events.

The convergence of the social and biophysical sciences in this area, as in other areas such as clinical medicine, will represent the benchmark for a scientific future which can better approach an understanding of humankind. The models presented here and the ongoing research in this area should better explain why it is that people, faced with a given situation,do what comes naturally.

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A SOCIAL PSYCHOLOGICAL VIEW OF MASS PSYCHOGENIC ILLNESS

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The perspective described here is most applicable to epidemics in closed populations. such as schools or factories. It postulates a situation in which there is a high level of collective tension, the introduction of one or more cases of dramatic individual illness events, and the resulting increase of tension and associated physiological symptoms. Those who become ill can be differentiated from those who do not according to level of tension, personality characteristics and position (spatial and social) in the situation. The importance of a credible explanation of the illness is emphasized, and the actions of experts who seek to understand the problem are seen as sources of validation of the credibility of the danger. These actions are also seen as contributing to a tendency for later cases to be less severely ill and to the ultimate dampening of the contagious response. Reasons for the preponderance of female victims are discussed, and several kinds of issues future investigations might emphasize are suggested.

Although reports of mass psychogenic illness have been available for centuries, only during the past few decades has there been any significant scientific interest in them. The increased scientific interest has resulted in part from the recognition that such cases are frequent in occurrence and "expensive", both socially and economically. Perhaps even more important in explaining the increased interest, however, is the greater acceptance of psychogenic explanations of illness behavior. This had led investigators to go beyond identifying such epidemics by the exclusion of other sources of illness and to attempt to specify the dynamics of the process by which they develop. This paper discusses one view of such epidemics of mass psychogenic illness.

The critical feature of the social psychological perspective used in this analysis is its focus on the interpersonal and cultural context within which mass psychogenic illness develops. It recognizes that those examples which have received the most attention recently all occur in relatively constrained social settings in which the participants are limited in the degree to which they can determine on their own the kinds of behaviors they will engage in. This structured context is especially important in understanding what generates such psychogenic epidemics. Equally important in understanding them is a set of cultural definitions which specify appropriate methods by which individuals may cope with their personal problems and their social relations with others in those settings. The definitions that prove to be important include both those which pre-exist in the group involved and those that evolve during the period of disruption.

The view discussed here may be summarized as follows: Mass psychogenic illness tends to occur in a situation in which a number of people find themselves exposed to sources of strain

from which no fully acceptable means of escape is availible. Those who are in the situation may know that some actions would serve this purpose, but the actions would also lead to negative outcomes, and they are thus not used. The combination of the original strain and the difficulty of using any known means of coping generates a general state of tension whose level is likely to be raised through interstimulation among those present. The state of unresolved tension leads at least some people to experience physiologocal symptoms. These symptoms come to be associated in the minds of at least some of those present with some credible, though factually incorrect, "cause" in the setting. Such an external "cause" objectifies the source of the symptoms and adds a further source of strain - the fear of the threatening agent. The belief in the threatening agent, the increasing number of victims, and the actions of outside experts which tend to validate the credibility of the threat all serve to increase the probability that others will experience similar symptoms which may be defined as being due to the presumed source of the spreading sickness. (See Kerckhoff 1970 for a more general statment of this perspective.)

In the process of spelling out the details of this social psychological perspective, the paper cites examples of the kinds of processes discussed. It then turns to the prospects for furhter work on the problem, and asks: What kinds of research appear to be most promising to increase our understanding of cases of this kind?

Although it is possible to analyze epidemics of mass psychogenic illness within a broader framework than will be used here (see Markush 1973, Sirois 1974), the present discussion will be limited to examples occurring in relatively closed populations. Most of the examples appearing in recent writings have occurred in institutional settings, such as in a school or a factory. It seems likely that the present discussion could be extended to apply to other less circumscribed settings, but to do so would require us to discuss situations about which little empirical evidence exists. As will become apparent in what follows, theoretical leaps are needed even to discuss the more circumscribed settings; much greater gaps exist in our knowledge of more open situations.

There are two immediately obvious characteristics of such settings as schools and factories: (1) They involve relatively large numbers of people, all of whom are in roughly the same position in the social structure. Both schools and factories have large numbers of relatively low status persons (students, workers) who are defined as a homogenious population by their superiors (teachers, supervisors). (2) In order to solve the functional problem of continuous and orderly operations, a number of limiting rules are established which restrict the amount of independent action on the part of all such low status persons. Workers can only take schedule "breaks,"

students need a "hall pass", etc. Even the most enlightened management or administration must restrict free movement to a considerable degree, and it is impossible to accommodate to the full range of individual differences. This is the normal state of affairs in a school or a factory.

Such restrictions are, of course, variable, both from place to place and from time to time in a given institutional setting. Many of the studies of mass psychogenic illness have suggested that the epidemics occur under conditions of additional restriction or stress. For instance, pressure from management to increase production, or the introduction of a new procedure, or the requirement that workers work overtime can make the normally tolerable conditions more stressful. It is also true that most institutions have mechanisms for dealing with the normal tensions associated with the necessity of restricted individual autonomy. In the school, the teacher attempts to be sensitive to the individual needs of the students, and counselors and others serve as a further source of amelioration. In a factory, supervisors will attempt to balance the need for production with a humane concern for the workers, and unions and other worker organizations provide channels through which difficulties can often be resolved. Yet, these mechanisms do not always function satisfactorily from the perspective of the worker or student. Such organizations can become disorganized, individual supervisors or teachers can fail to meet others' needs, or external conditions (e.g., the pressure of production schedules, the upcoming examination) can limit what can be done to ease the problems experienced by the workers or students.

Many of the epidemics studied seem to have occurred in settings in which the pressures on the low status persons were greater than usual (Colligan and Murphy, 1979). A general sense of tension was experienced by the workers. A further condition also seems necessary if some form of mass psychogenic illness is to develop out of such tense situations. This is the inability of the workers who experience the tension to take appropriate action to relieve it. In some examples, for instance, inadequate avenues of redress within the organization may become important (Kerckhoff and Back 1978). However, one obvious method of reducing the stress is always available - one can simply leave the situation. Clearly, in those situations in which a mass psychogenic illness epidemic occurs, that method has not been used. Workers get sick on the job, students get sick in school. There seem to be two kinds of possible explanation for the fact that those who experience stress in such situations do not leave. First, in many such situations,especially for workers, there are obvious rewards associated with staying, and a loss of those rewards is associated with leaving. Absenteeism leads to loss of pay, and it can even lead to dismissal. Many workers remain on the job out of fear of such loss, thereby increasing the

sense of strain. Second, if at least part of the job-related stress is due to unsatisfactory channels of amelioration on the job, any attempt to seek approval of a lessening of stress may even worsen the workers' relations with supervisors. There is thus likely to be an uneasy balance between the need to do something about the problem that is faced and the need to avoid the kinds of action which seem most suitable for coping with the problem.

The discussion thus far appears to describe a situation in which workers or students can analyze the pros and cons of taking action to resolve a problem but are caught in a no-win situation. This really oversimplifies the matter. Actually, the source(s) of tension may not be clearly understood by those who experience it. Especially in well-established situations, the requirements may seem so natural and appropriate that it is not clear to those who experience them just what the problem is. There is often a pervading sense of tension without an understanding of its source. Most workers recognize that limitations, rules, specified procedures are needed if the factory is to function adequately. They may not like it, but they will probably see such restrictions as something they have to accept if they are to hold that job. That may not reduce the tension, but it may reduce their ability to conceive of a way of lessening it. It is also important to recognize that if mass psychogenic illness is to result, tension must be relatively widespread in the situation. This does not mean that everyone must be equally distressed, but it does mean that the shared conditions must result in a shared sense of tension.

A situation in which many people working in the same setting share a sense of tension which they do not understand and/or know how to reduce is undoubtedly fairly common. What we find is that in some such settings, one or more cases of illness appear, and these are explained (either by the victims or by others) as being due to some toxic element in the situation. The element may be a gas, a chemical, a poisonous insect, etc. The presumed toxic element must have two important characteristics, however. First, it must be in the situation. The early cases of illness must be attributable to something to which other workers are also presumably exposed. Second, it must be a credible threat. That is, it must be consistent with the general cultural beliefs regarding how people get sick (e.g., a toxic chemical is more credible for most Americans than witchcraft.) Often, there is either clear evidence to support the idea of the threat or the presumed threat is the kind for which such evidence is difficult to produce. In some epidemics, a strange smell is defined as a poisonous gas. In others, a tiny insect or an irritant in the materials being handled or an unknown contagious disease is blamed for the illnesses.

This last characteristic, the kind of evidential basis for the explanation, is the most problematic. The epidemics reported in the press and even in the professional literature are often unclear about how the affected population explains the outbreak. It seems likely that where the reports are silent on this matter it is because of limited information rather than a lack of an explanation in the affected population, but we cannot be sure about that. It is clear, however, in those cases where an explanation is reported, that it may either be available before the first cases of illness or be provided after they occur. For instance, in some cases, it is clear that someone identified a "strange smell" or other foreign element in the situation prior to the first cases, and those cases were immediately attributed to that element (Roth 1965). In others, the cases are initially attributed to a "mysterious malady" and only later does a coherent explanation evolve (Levine, et al 1974). It is difficult to generalize from present evidence beyond indicating that once a number of cases of illness occur, they generally come to be linked together as having been due to the same threatening force, a force that is in the school or work setting and which continues to threaten others in that setting. In most cases, some kind of "cause" of the cases comes to be agreed upon by many people in or associated with the situation, but even where the cases remain mysterious, they are seen as all being caused by "it" - whatever "it" is.

The most common, and obviously necessary, action taken by officials when the first case or cases of illness occur is to remove the individuals from the setting to where they can receive medical attention. Though this is certainly appropriate, it has an effect which goes beyond the simple medical treatment provided for the stricken individual; it calls attention to the cases, and it provides confirmation that the individuals "really are" ill. In the wake of a series of cases, other official actions are usually taken which even more dramatically validate the significance of the threat. The plant or the school may be closed, experts are brought in to conduct various tests which seek to specify the source of the difficulty. If a gas is suspected, air samples are taken. If a toxic chemical is suspected, all kinds of tests may be conducted. If a poisonous insect is suspected, exterminators may be brought in. Such actions also tend to confirm the possibility of a serious threat.

This is where the credibility of the "cause" that has been suggested within the affected population becomes critical. If the kind of illness experienced by the early cases is a possible outcome of the kind of threat reported, officials will take the reports more seriously and will mobilize every visible and technically adequate means of seeking evidence of the threat. Even if the evidence is not found, the search itself increases the general credibility of the threat and

makes it likely that some of those in the affected population who were at first skeptical will have second thoughts.

There is also a reciprocal reinforcement process involved in the validation of the threat. Although one or two cases of illness might not impress many of those present, as the number of cases increases, it is difficult to avoid believing that "something" must be making those people sick. Again, if a credible explanation involving a toxic element in the situation has been suggested, the more cases that occur, the more difficult it will be to discount that particular explanation. Also, the more an individual believes in the reality of the threat, the greater the tension (s)he is likely to experience, and the more likely (s)he is to experience symptoms associated with tension. Such symptoms are then very likely to be associated with the suggested threat, and the individual may well become a "case."

The general view of mass psychogenic illness adopted here sees the individual cases as exhibiting symptoms that are associated with unresolved tension. It is assumed that many circumscribed settings, such as those found in schools and factories, impose sufficient restrictions on the free activities of individuals that many persons in those settings will be particularly tension-filled. The introduction into such a situation of one or more cases of illness, especially if the illness is dramatic (e.g., fainting), will suggest that a tangible source of threat is present. This would be especially likely in a population whose members do not recognize the possiblity of a psychogenic contribution to discomfort and who, therefore, tend to "somatize their difficulties" (Bart 1978). Those who are experiencing unresolved tension will be likely to associate their physical experiences with the presumed source of threat and attribute their discomfort to it. Belief in the presumed threat will increase the level of discomfort they experience and make it likely that other cases will occur and that they will be explained as being due to the same threatening element.

The actions of officials, though necessary, will tend to validate the seriousness of the threat, thereby adding to the tension and increasing the likelihood that symptoms will be experienced and they will be attributed to "it," whatever "it" is.

If the general pattern of evolution of mass psychogenic illness just outlined is at least partially correct, it leads us to look for certain patterns in the development of such epidemics. It is also true that numerous studies of such epidemics have uncovered some patterns which can be used to raise further questions about the perspective just outlined. For instance, in no known example of mass psychogenic illness have all persons in the population involved become ill. This

raises questions about channels of "contagion" and the possibility of "immunity". Also, if the explanations of the threat must be "credible," this raises questions about differences in the social definitions of what is credible and how they might effect the development of an epidemic. And, if the first (few) cases are presumed to be almost random events which act as initiators of a complex process in which a belief in a threatening force evolves, and if that belief provides an explanation of the symptoms others experience and leads them to become more seriously affected, this suggests that the early cases are different from the later ones. Finally, there has been a strikingly consistent pattern reported that suggests that women are much more likely than men to be victims of mass psychogenic illness. Some attempt must be made to asses the validity and significance of that sex difference.

Who becomes a case? While the question that heads this section is central to understanding and attempting to deal with cases of mass psychogenic illness no simple answer can be given at this time. However, the perspective proposed in this paper suggests some directions in which future investigations might go in pursuing an answer. The general conceptualization presented earlier points to several factors which increase the likelihood of an epidemic and therefore increase the likelihood that any given individual will become ill. These factors are: tension experienced in the work (or school) situation, the experience of symptoms resulting from that tension, the belief that those symptoms and serious illness can result from a threatening element in the situation, and the failure to take adequate coping action to relieve the symptoms. Although all of these elements are included in the general theory of mass psychogenic illness proposed here, each of them is a variable rather than a constant, and thus various individuals in the situation will exhibit them to differing degrees. The probability of any given individual becoming a "case" in an epidemic of this kind should thus vary with the extent to which each of these factors characterizes him or her. There is evidence that variation on each of them is important in becoming ill.

The kind of tension relevant to mass psychogenic illness which has been emphasized in the earlier discussion is the tension generated by the conditions experienced in the setting in which the epidemic occurs. Since the setting is not wholly uniform, undoubtedly people in different groups vary in their experiences. Yet, it is difficult to assess the significance of situational sources of tension without also considering experiences outside that setting. For instance, although required overtime work may be stressful for all workers, this undoubtedly varies according to the other kinds of demands on a worker's time imposed by outside groups - such as family obligations. For some, overtime simply means additional energy

expended for a higher rate of pay. For others, overtime means very complicated arrangements for child care, club or church obligations, car pool schedules, and so on. Also, of course, there are variations in the kinds of stress experienced by workers outside the work place. For some, the tensions at work are simply added to many others experienced elsewhere. Without attempting to propose a "calculus of tension", therefore, it seems reasonable to assume that individuals in the same setting will actually be experiencing different levels of tension. An adequate understanding of the differential response to epidemics of mass psychogenic illness must certainly take that variation into account.

Many studies have attempted to assess the significance of personality characteristics in the distribution of cases within a population (see Colligan and Murphy, 1979). Basically, in the context of the present discussion, this suggests that individuals may vary in their ability to cope with tension or stress. Even holding the level of tension constant, individual responses to it may vary. For instance, the tendency to attribute one's own control, or the tendency to seek assistance rather than attempt to cope on one's own, seems likely to affect how an individual will react to a sense of tension in a work or school situation. In particular, how an individual responds to physical symptoms (fatigue, headaches, nausea, feeling faint, etc.), in fact the willingness to acknowledge that "something is wrong", may influence how (s)he will behave in such a situation. One very tangible way in which such differences can affect the development of an epidemic of mass psychogenic illness is to determine whether individuals will remain in a situation which is stressful to them. The view developed in this paper suggests that those who are able to acknowledge that they have a problem, who can assess it as an externally caused problem, and who see medical experts as possible sources of amelioration, will leave the situation and seek aid before becoming a "case" in the dramatic way that will contribute to an epidemic. In that way, some individuals are probably "immune" to some of the forces which spread the epidemic (see Kerckhoff and Back, 1978).

Other factors besides personality undoubtedly affect the probability of an individual's becoming a "case", however. One of these is the position the individual occupies in the social setting. The position can be defined in both spatial and social terms. In spatial terms, the closer the individual is to the early cases, the more impressive they are likely to be. It is difficult to believe that the victim was "faking" or wasn't "really sick" if you were standing next to them when it happened. Also, if the explanation of the early cases of illness is made in terms of a toxic substance in the situation, anyone who was close to them can more easily believe that (s)he has been "infected".

Social closeness may be at least as important as spatial closeness. If one's best friend has been stricken, it is very difficult to argue that only "a bunch of crazy women" have been involved. Of course, if the epidemic continues over a period of days, as many have done, being personally close to an earlier victim is probably going to lead to visits to the hospital, discussions among mutual friends, and attempts to follow the progress of the case. In other words, the experience is going to be more intense and continuing than if one does not know the earlier victims personally. The importance of interpersonal relations in the transmission of mass psychogenic illness has been strongly suggested by the research to date (Kerckhoff and Back, 1968). The same social psychological perspective suggests, however, that resistance to the transmission may also depend on interpersonal relations. Being "immune" may thus depend not only on personal characteristics but also on one's social position. If none of one's acquaintances has been stricken, it is easier to reject the entire epidemic as something "out there" and not personally relevant. We might thus expect that non-victims would be socially connected, just as we expect that victims would be socially connected.

What is a "credible" explanation? In a detailed case study of a case of mass psychogenic illness, Kurt Back and I (Kerckhoff and Back 1968) argued that the explanation of the illness needed to be reasonable within the particular setting. In that case, the bite of a poisonous insect was a reasonable explanation since the epidemic occurred in June in the South where ticks and black widow spiders were known to be found. We pointed out that: "Although almost any kind of threat might have provided the basis for such an epidemic of symptoms (e.g., a vengeful spirit whose 'home' was disturbed by the building of the plant), the credibility of some kinds of threat will be greater than others" (pp. 158-9). In the context of the U.S.A. in the 1960's, that was a reasonable statement. Yet, we know that in another time and place the idea that illness might be caused by a vengeful spirit would be quite credible indeed.

An interesting example of the importance of the social and cultural context is provided by an epidemic in Singapore discribed by Chew, et al (1976). They reported a series of cases of illness (what they call "spells") in a TV assembly plant. Many of the cases involved violent behavior, screaming and fainting. In several cases, the affected women claimed to be possessed, to be "spirits" (the "jin"). A local medicine man was brought in to help solve the mystery and to calm the workers. This kind of explanation of a series of cases and the action taken by management would hardly be credible in this country, but it was quite credible within that cultural context. The case in Singapore also indicates the importance of social relations in the affected population.

Although only two-thirds of the workers in the plant were Malays (others were Chinese and Indians), all but one of the affected cases were Malays. Whether this concentration within the one cultural group was a function of shared cultural beliefs (which made the claims of being possessed more credible to the Malays than to the others) or due to closeness of personal relations (which made the early cases more significant to friends within the single cultural group) cannot be determined from the data available. But the seeming "immunity" of the non-Malays is very striking. Back and I noted a similar difference between black and white workers' susceptibility to the epidemic of insect bites but in that case the social segregation of the two groups seemed to be the obvious explanation rather than differences in cultural beliefs.

It is interesting to note that in the Singapore case, management called in the medicine man even though they presumably did not believe the explanation of the epidemic that such an action confirmed. The action was taken to attempt to reassure the affected population that everything possible was being done to "cure" the illness. It seems likely that some epidemics closer to home have also involved such palliative actions by those outside the affected population. In order to reassure those affected, experts or managers may carry out procedures which they view as wholly unnecessary but which are used to prove the invalidity of a possible explanation of the illness or to prevent further cases attributed to that presumed cause. No generalization is possible, but it is at least reasonable to question whether such actions are always well-advised. Although a negative finding from such procedures may "prove" to the experts that the suggested explanation was incorrect, the very fact that the experts took that action tends to confirm the credibility of the explanation. More often, of course, the experts are at least originally unsure whether a truly toxic element is responsible for the illness, and thus they must carry out a series of procedures to search for such an element. Any such action tends to confirm that there could be toxic elements in the situation which could account for the illness. There seems no way, short of an initial confident diagnosis of mass psychogenic illness, to avoid presenting that possibility to the affected population. It may be wise, however, to avoid offering an unnecessarily long list of possible explanations, including explanations the experts view as not actually credible.

The view proposed here is based on the assumption that the initial cases in most epidemics of mass psychogenic illness are in the nature of random events which then set off a kind of "chain reaction" of similar illnesses within the population. It may well be, of course, that the initial cases are due to the kinds of distress in the work situation which has been discussed earlier. These individuals may be under greater tension or be less able than others to cope with it. However,

their illness may be due to entirely different causes. The first cases may actually be responding to a toxic element, a virus, food poisoning, etc. The important thing, though, is that their illness becomes defined as being due to something in the circumscribed setting and that others then associate any symptoms they may experience with this presumed source of threat. According to this view, the early cases provide evidence of a threat in the setting, and they thus both increase the level of tension experienced by others and provide a ready explanation for any symptoms others may develop.

The early cases are likely to be rather dramatic. The worker who feels ill and quietly goes to the infirmary or gets permission to leave work is not likely to stimulate the kinds of epidemics discussed here. Screaming, vomiting, and fainting are the kinds of behavior which attract attention and suggest that a serious problem may be involved. Even then, however, the idea that there is an imminent threat will probably not immediately be widely accepted. But it is only necessary for one or two others to accept the idea, to respond overtly to the fear and to exhibit illness behavior, in order to increase the level of tension and raise the level of physical discomfort many others will experience. As the number of cases grows, the belief in the threat will increase, and overt responses to the threat will spread.

The responses to the threat will be of many different kinds. Those of us who study such epidemics focus on the "cases," those who are known to have become ill. Those individuals represent only one kind of response, however. Others may respond by withdrawing from the situation, by attempting to reinforce each other's skepticism about the threat, by mounting a corrective program (helping those stricken, complaining to management, etc.), or in some other way. It may well be, in fact, that many situations which might have become epidemics of mass psychogenic illness are reduced in severity by such responses.

In the epidemics that evolve sufficiently to be studied carefully, there seems to be a pattern of legitimation involved that both increases the number of cases reported and reduces the level of severity of the symptoms in the later cases. As the number of cases increase, the explanation for them which evolves becomes increasingly convincing. At least it becomes increasingly difficult to deny that "something" is causing the illness. Since medical treatment is sought for those who become seriously ill, seeking medical attention is the reasonable thing for anyone to do if they believe they are becoming ill. In the later period of the epidemic, therefore, individuals in the population who experience even minor symptoms are likely to attribute them to the threatening element in the situation, and they are likely to seek medical aid before becoming seriously ill. The concerned response of

the physicians tends to legitimize the reporting of even mild symptoms. As the number of cases grows, therefore, the severity of the symptoms reported to physicians will decline.

I noted earlier that when the experts take the illness behavior of the early cases very seriously and launch ameliorative actions and a search for causes, this tends to validate the belief in a threatening force and to increase the tension in the situation. Yet, one can argue that the continued involvement of experts can ultimately dampen the spread of the illness. By making it possible for those with mild symptoms to receive medical attention, physicians reduce the incidence of severe cases and lower the level of fear in the situation. Also, as the level of severity of the symptoms declines, even those who firmly believe in the reality of the threatening force are able to see the threat as one that is "going away". Whatever "it" was, it does not seem to be as bad as before. It may be true in some cases that "almost everyone" comes to believe that the illness was "only in their heads", and thus the epidemic ends in embarrassment. However, the general dampening effect due to expert attention and declining symptom severity seems a more reasonable explanation for the termination of most epidemics of mass psychogenic illness.

It has been repeatedly noted that women are much more likely than men to be the victims in such epidemics. The evidence is too strong to deny the sex-linked nature of the phenomenon. Yet, this only poses the further question of why this should be so. No one is likely to accept the facile explanation that "women are just like that" without raising additional questions.

Colligan and Murphy (1979) have suggested that a consideration of the nature of sex roles in our society may help explain these observations. They note that "females have been encouraged to by sympathetic, loyal, submissive and emotionally expressive" and that these characteristics should lead women to be more active in an expressive social process such as found in these epidemics. They also point out that women in the labor force often face some serious role conflicts. Their work obligations may make it difficult for them to carry out their familial obligations in the way they would like to. Thus, women may experience more stress on the job than men because of these dual and sometimes conflicting obligations. The combination of greater tension and a more expressive personality can thus be viewed as a reasonable basis for explaining the preponderance of female victims of mass psychogenic illness.

Although I think that the Colligan and Murphy discussion has considerable merit, I would like to suggest another possible reason for the preponderance of female victims. When we review the work situations in which the best documented cases of mass psychogenic illness have occurred, we find them to be

strikingly similar. A room filled with rows of sewing machines operated by women, a series of long benches at which women assemble TV sets, a large room in which many women punch and verify computer data cards, and so on. These are highly regimented work situations in which the workers are all carrying out very boring, repetitive tasks. Hour after hour, they do the same thing over and over again. In most of the cases reported, the degree of regimentation seems to be very high and the workers are permitted only infrequent breaks between which they are restricted in both physical space and their ability to vary their activities. It would be difficult to find situations which would be more conductive to a sense of tension and frustration.

Without claiming to have detailed knowledge of the job distributions of men and women, I would like to suggest that women are more often found in such repetitive, boring, restrictive work settings than men are. It seems part of the folklore of industry that women are "good at" such tasks. They are supposed to tolerate the lack of large muscle movement and the constant repetition of small-part assembly tasks. Thus, we seldom find large numbers of men in such situations. It may be true that women tolerate those work conditions better than men, but it seems likely that those work conditions are as much responsible for the high proportion of female victims of mass psychogenic illness as is the fact that women are expressive and suffer from role conflict.

Since employers are certainly interested in preventing the kind of epidemics discussed here, it is worth suggesting that one of the ways they may be prevented is to reduce the strain workers experience on the job. It is not reasonable to expect that women are so "different" that they can easily tolerate conditions which would be expected to drive men to rebellion. At least the work conditions are under the control of management, whereas role conflict and the presumed expressive nature of the female personality are not. Thus, although all of these factors may help explain the preponderance of female victims, only one of them is controllable.

An important distinction between two kinds of cases of mass psychogenic illness is suggested by the differentiation made by Turner (1964) between convergence and contagion as the basis of similarity of behaviors in a collectivity. This distinction has been discussed by Milgram and Tock (1969), among others, and has more recently been included in a discussion of epidemics of mass psychogenic illness by Colligan and Murphy (1979). Turner was particularly concerned with the emergence of "new" or non-traditional forms of behavior which are not easily predicted in advance. The distinction he made was between cases in which the similar behaviors are relatively independent individual responses to a similar

stimulus (due to the "convergence" of similar persons) and the effects of one person's behavior on the tendency of another to behave similarly (through a "contagion" of the behavioral form. In fact, Turner argued that neither of these processes adequately explained many situations of collective behavior. Rather, he pointed to the importance of an "emergent norm" in such situations, a new definition which legitimized, often even encouraged, a form of behavior not previously deemed appropriate. He suggested that the behavior of the index cases in many such situations did not so much "contage" as they helped to define what was a possible, useful, or a satisfying way of coping with the situation. Others adopt such forms of behavior, then, because they provide an acceptable way of behaving in an ill-defined situation. Turner's emergent norm theory was thus intended to counteract what he and others viewed as the magical quality of the idea of behavioral contagion.

I would agree with Turner's basic idea that the spread of similar forms of behavior is seldom due to a simple process of contagion if contagion is viewed as direct imitation without purpose. I would prefer to use the term contagion to refer to a process by which one person's behavior elicits a similar response from another due to the meaning that the behavior has for the other person. In some cases, that meaning may be goal-directed; the behavior is seen as a good way to achieve a desirable goal. In the epidemics described here, the behavior of those who seek medical assistance when they begin to feel ill would be of that type. In other cases, the original behavior may serve to define the conditions in which the population finds itself. The early cases in an epidemic serve to define the setting as one that contains a dangerous element. If another person then becomes ill, it is not because (s)he "caught" the illness expressed by the other's behavior but because (s)he believed in the threat and experienced physiological symptoms of increased tension.

Even if we use the term "contagion" in this special or restricted way, however, it is clear that not all epidemics of mass psychogenic illness involve much contagion. In many epidemics, a number of cases occur almost simultaneously in response to a suddenly discovered (or suspected) toxic element. Someone smells a strange odor, and a number of people faint in response to this "gas". Others vacate the area, and the epidemic is over. It all happens at once and is quickly finished. It seems likely that such epidemics are the result of the convergence rather than the contagion process. By that is meant that the individuals who become victims are responding more in parallel than in sequence, their personal qualities are much more fully responsible for the behavior than are the kinds of worker-worker and expert-worker relations that have been discussed here.

Undoubtedly there are no really pure examples of convergence or contagion. In the present discussion, for instance, we assume that one or more early cases initiate the contagion process. In most epidemics, it seems reasonable to view several of the early cases as relatively individual events involving little inter-individual influence. And, even in the most rapid response epidemics, all cases do not occur simultaneously and thus it is at least possible that later cases have been influenced by the existance of earlier cases. But, the two kinds of situations are very different, and it does not seem reasonable to expect the same generalizations to apply to both. At the time we were engaged in the analysis of the June Bug data, we investigated another epidemic which followed a pattern expected from the convergence view of these epidemics (Roth 1965). We were unable to find any pattern of interpersonal relations among the victims, even though such a pattern was a prominent feature of the June Bug analysis. Personality characteristics seem to be a much more central feature in cases where the convergence perspective is appropriate. Future research should attempt to differentiate between these two kinds of epidemics and to assess the extent to which different kinds of explanation are needed for them. For instance, is the convergence type more likely to occur in populations having a high proportion of a particular kind of personality, and/or an overall higher level of tension? Is it possible that the contagion type can occur in any population having a basic set of structural characteristics, only requiring a single or small number of initial cases which are defined appropriately to heighten the level of fear?

This last question leads to another set of issues which the earlier discussion has made salient. Basically, these are issues dealing with the structure of the situations in which the epidemics occur. Are some structures especially likely to generate tension as I have suggested earlier? If so, are there ways in which the tension may be relieved so as to avoid the kinds of epidemics we have observed? Although this is presented as an important research question, it is obviously an important management question as well. It may be possible to observe various kinds of structured situations and to assess their tension-producing potential without waiting for one of the explosive events to occur. This would require a better set of measures than I am aware exists for indexing collective tension, but it would be well worth the investment to attempt to develop such measures.

The situation changes, of course, when the early cases occur. A set of actions, both official and unofficial, are initiated at that time. We are beginning to have available information about a sufficient number of cases that it ought to be possible to begin to estimate what kinds of actions tend to increase and what kinds tend to decrease the probability of additional cases of illness. With regard to official actions,

the earlier discussion has suggested two opposite patterns. It has suggested that the intense and serious involvement of experts in the situation may serve to increase the credibility of a serious threat thereby raising the level of tension and increasing the probability of additional cases. Yet, it has also suggested that the involvement of experts should ultimately lead to a reduction in the incidence of cases with severe symptoms and will help convince even firm believers that, whatever the problem might have been, it has been removed. It is possible, of course, that both of these statements are correct. An initial increase in tension may be the price that must be paid for the ultimate solution to the problem.

It seems unlikely, in any event, that the involvement of experts can be avoided. Unless we are able to specify for industrial and school physicians what symptoms are found only in cases of mass psychogenic illness, these epidemics will continue to require the serious and intensive involvement of medical and other kinds of experts. In one of the more widely-read discussions of such epidemics (Roueche 1978), it is suggested that if the attending physician or other authority figure takes a firm stand, (s)he can stop an epidemic before it becomes more serious. If (s)he simply states authoritatively that there is no toxic element in the situation and that the symptoms are due to tension and fear, it is possible to return the situation to normal in short order. Yet, in that same discussion, the physician who accomplished this transformation is quoted as having said, "But, thank God, I was lucky". It isn't likely that many people would want them to do so. Yet, more careful study of the immediate symptoms and the pattern of spread of the early cases may make it possible to make such decisions with greater confidence. One way to do this would be to record as carefully as possible the sequence of events in epidemics that do occur, even if this must be done retrospectively. A systematic review of a series of such records could then make it possible to determine when one could make a confident determination that the epidemic is one of mass psychogenic illness and not some other form.

It should be emphasized in closing, though, that in order to approach these epidemics in a way that is most promising for diagnosis and definitive action, something besides the usual medical perspective must be adopted. In the present discussion I have adopted a terminology I assume most physicians would have used, at least in that I have referred to individual "cases". The "case" in this and most discussions is an individual who comes to the attention of a physician, is examined and, possible, given some form of treatment. To make the kinds of analysis suggested here, and to be able to take the kind of definitive action that might effectively dampen an epidemic with strong psychogenic components, it is necessary to see the whole series of individual cases as the

focus of analysis. The clues to the overall diagnosis will be found as much in the pattern of cases and in the overall distribution of symptoms as it will in the individual cases. The physician may not always be able to "step back" and take such a broader view - if nothing else, he may be too busy coping with the flood of individual cases - but someone needs to do it.

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AN EPIDEMIC IN A TEXTILE PLANT IN THE ENGLISH MIDLANDS

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An account is given of an apparant psychogenic epidemic that occurred among female operatives in a textile factory in the English Midlands. In the course of one morning, 22 of the factory's 161 employees - all of whom were females - were afflicted with short-lived symptoms that included dizziness, blurring of vision, nausea and gastro-intestinal upsets.

The epidemic began among the younger women and was largely confined to them, the attack rates being 26% in the under 20's against 6% in the over 20's. It appears that an episode the previous day in which one of the women soiled herself in public may have been the factor that raised the general anxiety level, with a rather dramatic faint by another employee triggering the actual outbreak.

In May 1968, one of the British national newspapers ran a report on a peculiar epidemic at a clothing factory in a town in Midlands. Under the banner headline 'Mystery Bug Hits Girls at Factory', it recounted how the girls had started collapsing soon after arriving at work. The symptoms were varied: dizziness, blurred sight and stomach aches - to which a local newspaper reporter added "sick and jelly-like legs". On the theory that the illness could have been caused by fumes, the Fire Brigade had been summoned but when they arrived,the firemen found nothing to support this idea and they ended up giving the building a completely clean bill of health. The ambulance men found more to occupy them: they had been sent for by the manager when he realized that the morning's trouble was going to be more than a matter of just one or two cases. By the time they arrived, the total had, in fact, risen to 15 and they were sufficiently impressed by what they saw to take all these 15 to the hospital. During the later part of the morning, another six employees were affected, these cases apparently being less severe because they were merely sent home, not taken to the hospital. The number of people employed at the factory was given as 170, all of them, apart from the manager, being female. 1.2

The doctors at the hospital the more seriously ill women were taken to couldn't find anything wrong with them: they were obviously upset and frightened by clinical examinations were entirely negative. Nor could the City Medical Officers find anything to go on when they visited the factory in the early afternoon. The talk of sickness suggested the possibility of food poisoning but the outbreak had taken place within an hour of the start of work, before any food had been consumed on the premises and the episode had built up so quickly that it is difficult to believe that food eaten in the canteen the previous day could have been responsible. Moreover, it was soon established that only about half the women involved in the epidemic ever eat in the canteen at all - many of them lived close enough to go home for their meals. Nevertheless, a meticulous examination of the kitchen equipment was under-

taken, while faecal specimens were collected from the kitchen staff, the affected women and any members of their families who volunteered that they had been less than well. None of these investigations yielded anything which could be plausibly linked to the epidemic.

During the Medical Officers' visit to the factory, one more member of the staff, a woman of 38, was taken ill. Her symptoms didn't seem to be the same as the others: to quote the doctor's report:

> "She was shaking violently and had apparently lost partial use of her limbs. She was taken to the rest room and (later) appeared to be recovering well."

She was the last person to be affected.[3]

My own part in the investigation that followed this epidemic was minor. I got in touch with the City's Department of Health, stating my interest in psychogenic epidemics. I was given permission to visit the factory and collect some basic data on the population at risk. This could be regarded as a single entity because the women all worked in one large room. This was well lit and had no internal divisions, so everyone could easily see everyone else and any disturbance would immediately be apparent to all. The question was: were the younger women disproportionately involved in the epidemic? If they were, it would indicate that at least part of the trouble had been emotional.

The analysis was soon done. The factory management supplied me with a list of their employees - 161 in all - and their dates of birth. Sixty-one of them were under 20 years of age and of these women, 16 (or more than a quarter) had been affected. The remaining 100, who were 20 years of age or more, had produced only 6 cases between them. The difference between these attack rates - 26% in the younger group and 6% in the older - seemed to me significant.

Everything now suggested that the psychogenic element had been an important, indeed maybe dominating, factor in the epidemic. Understandably enough the result was that both the City Health Authorities and the Factory Management decided that the right course of action was to play down the whole affair. The sooner it was forgotten, the less likely they were to have another outbreak. This is not only understandable, it is entirely correct. Too active a scrutiny can easily keep this sort of trouble going and even cause it to flare up worse than ever. Nevertheless, the decision was sad from my point of view because it meant my being asked to stop my investigation just when things were getting interesting.

At the end of the year, the City Authorities sent me their

Annual Report. This contained the following paragraph on the epidemic under the rubric ' An Outbreak of Doubtful Aetiology."

> In May there occurred among factory employees engaged in clothing manufacture, an exposure outbreak of illness characterised by prostration, nausea, abdominal pains and diarrhea with loss of consciousness in a few cases. Out of a total of 165 female employees, 22 were affected and some hysterical elements appeared to be present. There was no common food factor and despite extensive investigations, no infectious or toxic cause of the outbreak could be traced.[4]

And that was that - or at least it was until the officers of NIOSH were kind enough to ask me to speak at this symposium.

Although I have been involved - nearly always retrospectively - in investigating putatively psychogenic epidemics in various schools and hospitals, I have only had the one experience of this type of thing in an industrial setting. Clearly, if I was going to come to Chicago at all, I would have to do my best to discover a bit more about the how and why of it. I got in touch with the Medical Officer of Health for the area- a new man- and put my case to him. He came up with the welcome news that the factory was still in operation, that there were people there who remembered the epidemic to make it worthwhile for me to visit, and that neither he nor the Management had any objection to my doing so. So, armed with a tape recorder, off I went.

Eleven years had seen some changes, of course. The factory premises were the same but the contraction in the British textile industry had taken its toll: the number of employees now was nearer 60 than the 160 it had been before. The work was exactly the same, though: the women were machining brassieres for a British multiple store which, though it doesn't own the factory, provides it with near enough 100% of its orders. The Manager was now a woman, quite right too, in these days of Women's Lib - and a piece of luck for me, she had been one of the six Floor Supervisors on duty at the time of the 1968 epidemic. It was largely due to her that I was able to piece together the following story.

Work began quite normally on the day of the epidemic, which was a Thursday. This means that all of the 150 or so operatives were at their benches within a few minutes of 8 a.m. These benches all faced the same way, towards what we can call the front wall. This wall contains two emergency exits, one on either side of a large central window (Fig. 1). The normal way in and out is through a door on the right. Off the short corridor between this door and the main factory entrance are the medical room, Manager's office, Records office and rest room.

The majority of the women - some 120 of them - were working sewing machines. Their benches occupied the rear two thirds of the factory floor. Immediately in front of them were 16 women working trimming machines, in front of them 12 'examiners' and, in front of them at a bench directly under the central window of the front wall, two packers.

Some time after work had begun - possibly as much as an hour later, but certainly no more - a woman in the trimming section fainted. She was seated at A5 on the plan: a young woman of 18, unmarried and with no medical or obstetric history. She remembers her head feeling muzzy, her arms feeling light and her fingers tingling: then she went right out. Her Supervisor was called and found it impossible to bring her round enough to walk her out: she was going to need carrying. The Manager arrived, picked her up, took her out through the left hand fire exit and went round the outside of the building and back through the entrance corridor to the medical room. He obviously felt that this would be quicker than trying to negotiate his way between the work benches. The Supervisor went with him. While the two of them were trying to revive their patient,the second case arrived at the medical room door. She was a 16 year old who had been sitting diagonally behind the first at B6. She was able to walk with assistance, this being provided by one of the other supervisors. The first girl was still flat out, a second had arrived and soon a third came on the scene. The manager decided to call for outside help.

Before either the ambulance or the Fire Brigade appeared on the scene - a matter of 10 minutes at most - a whole batch of women reported to, or were brought to, the medical room. As there was no space for them there, they were put in the corridor outside. This group eventually grew to number about a dozen.

We know where three of these women had been working. One was from position C6 in the trimming section where the trouble had started. Two others came from C11 and D8 in the block of machinists immediately behind. There is a probability that three other women came from this block and a general agreement that at this stage the epidemic hadn't spread any further towards the back of the room than this. The rearward block of machinists was either completely or almost completely unaffected.

Among those who had been taken ill by now was one of the six supervisors. She wasn't one of the two supervisors involved in caring for the sick: both of these were still in the medical room trying to cope with the two initial cases who showed no signs of recovery at all. One of these supervisors - my main informant - told me how daunted she felt: 'I honestly thought they were going to die'.

At this stage the ambulance men arrived. And at this stage one gets the first undoubted cases in the back of the room. One was an older women, 52 years of age, married and, according to people who knew her well, 'entirely sensible'. She was sitting at F19 and saw a young woman somewhere ahead of her show signs of becoming distressed. She said 'Oh look, there's another one' and then she herself slid off her chair and fell to the floor unconscious.

The woman behind her, who told me this story, admitted that she now got nervous herself. "I thought'it's that close to me I'll be next' and if I had let myself go I could have gone too. But Mr. Brigstock (the Manager) said 'Elsie, you're not going to faint, you're ALRIGHT.' And I was."

The epidemic now began to peter out. There was one more locatable case, also at the back of the room. This woman, 36 years old and married, was sitting at C18. By lunch time four others had reported sick and been sent home and then there was the final case, already mentioned, who became disturbed during the doctors' visit in the early afternoon. The interesting thing about these later cases is their higher average age. As the histogram shows (Fig. 2), the epidemic in its rapidly propagating phase had been concentrated in the younger age band. During its declining phase it struck fairly evenly across the age groups. The difference is, I am told, highly significant statistically, which is surprising because one very rarely finds any statistically significant differences in this sort of work. I'd like to think that it was a reflection of the more sluggish reactivity of the older age groups because I have long had a theory that the time-base of affective excitation and decay lengthens as one grows older.[5] Unfortunately, it seems clear that the older women tend to work in the back half of the room (Fig. 3) so it would be just as reasonable to say that these women reacted later because the stimulus reached them later. But what is true is that none of them were taken to the hospital, whereas all of the first lot were.

This small epidemic clearly bears the hallmarks of a psychogenic affliction. Fainting and feeling faint were important features. So, according to several observers, was an element of panic. There is a suggestion that the initiator had been overbreathing and an implication - in the visiting Medical Officers' report - that the final case in the series was more emotionally upset than physically ill. Nevertheless, there is one atypical element in the symptomatology: the report of gastro-intestinal upsets. This doesn't seem to fit the general picture: indeed, given the time scale of the epidemic, it doesn't seem to make any sense at all. How did it come to figure so prominently in the story?

A possible reason appeared when some of those who had been

present at the time reminisced for my benefit. The day before the epidemic, there had been an unusual and upsetting incident. As the women were clocking off work, one of them lost control of her bowels and soiled the passage way. A superviosr took her to the toilet and helped her to clean up and everything was restored to normal as soon as possible. But there was no way of concealing this public incident and it must have become a topic of conversation among the employees.

Nothing like this incident had occurred before, so far as anyone can remember, nor, for that matter, has anything like it occurred since. It must have been still in people's minds the next morning. So it is no wonder that, when some sort of sickness broke out among the women, several people simultaneously thought of faecal contamination as a possible cause. The idea seemed all the more plausible in that the employee who had had the problem the day before - a 17 year old - was working in the area where the epidemic originated - at C6, in the trimming section - and was herself soon involved in it. Moreover, she would obviously have said that he had had diarrhea if questioned by a doctor. Against this background, it is not difficult to imagine other affected women agreeing that they had stomach trouble too.

This seems to me to clear up the mystery of the gastro-intestinal complaints. It also suggests a possible reason why this particular epidemic occurred at all. A woman faints at this factory about once a month on average and yet there is only this one instance of the disturbance propagating. Perhaps the soiling incident the day before raised the employees' emotional tension to the threshold for propogation and, by bad luck, a faint occurred the very next day, before the tension had time to decay. The coincidence of the two elements, one so unusual as to be effectively unique, and one less unusual, but a bit alarming all the same, combined to produce an upsurge of anxiety in the susceptible members of the population- those who were younger, or not so well, or not so stable, or maybe just a bit more anxious than usual on that particular day.

A few final points. The index case was still employed at the factory at the time of my second visit - to be exact , she was back there after an absence of a few years during which she had had her family - and I was able to interview her. What seemed clear both from the interview and the general opinion of her held by her colleagues was that she was a very sensible, straightforward woman with no hysterical tendencies at all. This is not surprising: an epidemic occurs when the threshold for spontaneous propagation of anxiety is reached. The events that push anxiety up to this level must, of course, be anxiety-provoking, but they needn't in themselves be more psycho than somatic. Indeed, they needn't be of psychological inspiration at all. Doubtless if this young lady had dropped over with a subarachnoid hemorrhage instead of, as I suspect, a combined

vasovagal and overbreathing attack, the train of events on the factory floor would have been just the same. So, in looking at the process of psychogenic spread, there may not be all that much to learn from an inspection of the initial case. There may - as I think there has been in some instances - but there may not - as I think is clear in others. [6,7]

On the other hand, I think a study of the geography of an epidemic can be very useful. One of the less obvious but still critical contributions the index case made to this epidemic was to faint in the part of the room where the younger employees were concentrated and where the threshold for propagation was lowest. Factories with known seating plans clearly provide a good framework for studying the spread of epidemics and, hopefully, isolating factors of this type. I am especially sorry that I wasn't able to pursue this approach at the time this particular epidemic occurred: it would clearly be interesting to have a complete version of the seating plan and the case to case spread.

What I have learned is that one should never be discouraged from making a detailed examination just because people say that the cases occurred anywhere, might be any age, and get ill in any order. In every epidemic I have investigated, people have said things like this - and I have invariably found such statements to be misleading. Psychogenic epidemics are interesting in themselves and in my experience, detailed study of them makes them more so.

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OUTBREAKS OF MASS HYSTERIA AT WORKPLACES IN SINGAPORE- SOME PATTERNS AND MODES OF PRESENTATION

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Outbreaks of mass hysteria or "epidemic" hysteria have been described mainly in schools in various countries (E.S. Tan, 1963; Knight et al, 1965; Ebrahim, 1968; Gwee et al, 1969). But in recent years, this phenomenon has been noted to occur not infrequently in factories and other places of work, where its effects and implications may be much more serious in terms of economic considerations and work morale.

This paper summaries seven outbreaks of what may be termed "occupational" mass hysteria in the island Republic of Singapore, in the period 1973 to 1978, and discusses certain interesting features.

Three factories, situated near each other in a light industrial estate comprising blocks of flatted factories, were affected in the first series of outbreaks.

The first, and the main factory affected, was involved in assembling television sets. It employed 802 female factory workers (of whom about 65% were Malays- the rest were mainly Chinese and some Indians), and 97 male workers, working 2 shifts, from 7 a.m. to 3 p.m., and from 3 p.m. to 11 p.m. On Saturday, the 13th of January, 1973, at about 7 p.m., a female worker suddenly started screaming and then fainted. When the supervisor and some of her colleagues carried her to the clinic on another floor, she started screaming again and struggled with them. Subsequently other women were affected. Because of these incidents, the factory closed earlier that night.

The next 2 days were holidays, being a Sunday and a public holiday. As all the women affected were Malays, the management engaged a "bomeh",or native medicine man,to exorcise any evil spirits as it was believed that the incidents were a manifestation of spirit possession. But when the factory reopened on Tuesday, 16 Jan., more women were affected from 2:30 p.m. onwards.

On 17 Jan., the hysteria outbreak spread to a nearby factory manufacturing electrolytic capacitors, which also employed a predominantly female labor force. A few days later, a third factory reported one hysterical case.

The whole outbreak lasted from Jan. 13 to 27, and a total of 97 persons were affected, giving altogether 107 "spells" (or attacks), some having repeat attacks. (Chew et al, 1976).

	No. of Spells			
Factory	Hysteria	Frightened	Total	No. of Subjects
A	39	51	90	84
B	16	0	16	12
C	1	0	1	1
Total	56	51	107	97

There were 3 main modes of presentation of the cases:

1. Hysterical Seisures: These were characterized by screaming, struggling fiercly and general violence. A very violent woman would require 4 men to hold her down. Intramuscular injection of chlorpromazine or other tranquilizers e.g. valium (diazepam) did not seem effective. The affected worker would ignore questions or attempts at reassurances and might even scream more. Some would scream that they saw a dark figure coming to strangle them. Some would laugh in between screams.

2. Trance States: This was a "subgroup" of the hysterical presentation. While being violent, or before becoming violent, a worker would suddenly speak as though she was someone else, and claim that a "jin" or spirit was speaking through her. Four of the hysterical cases developed such a state, which lasted only a few minutes.

3. Frightened Spells: The affected workers only expressed a feeling of unexplained fear, and might complain of feeling cold, numb, or dizzy.

It was noted that 3 of the workers affected on the first day, had repeat attacks on the second day when the factory re-opened after 2 days of public holidays.

All were females, except one Malay male, who experienced only fear. Except for one Indian, all the cases were Malays.

The second major outbreak involved a feed factory. The factory produced dried cuttlefish. It occupied a 4-story building with an employment of about 350, of whom 300 were females, and about 70% were Malays. Working hours were from 7 a.m. to 5 p.m. (with staggered starting and finishing time), Monday to Friday, with work on alternate Saturdays. There was no shift work or night work.

The outbreak started on Saturday, 29 Sept. 1973, when a female worker suddenly started screaming and crying. She then became violent, requiring a few men to restrain her. This was followed by two other girls getting the attack. It was said that the girls saw a black person with hairy arms trying to frighten them.

The next day being a Sunday, the factory was closed. Nothing happened on the next 3 working days until Thursday, 4 Oct., when cases of hysteria started at 8 a.m. A total of 10 women were affected that morning, including 3 who had been affected on the first day. As there was great confusion, the management closed the factory in the afternoon.

A "bomeh", or medicine man, was called to exorcise evil spirits. A repeat exorcism was carried out a few days later,

as cases of hysteria continued to occur. A total of 56 "spells" (17 violent and 39 frightened) involving 28 persons occurred between 29 Sept. and 15 Oct. 1973.

Those affected were all Malay females, with two Malay males. (One of the Malay males was violent, but the other only experienced fear).

A feature of this outbreak was the high rate of recurrence. (Please see Table 2).

No. of Attacks (Spells) Per Person	No. of Persons
1	12
2	10
3	2
4	2
5	2
Total	28

Table 2: Recurrent Attacks (Spells) in those Affected.

It was interesting to note that all 3 of the women affected on the first day of the outbreak had repeat violent attacks on the second day when cases occurred again, although there were 3 working days in between with no attack.

Another series of episodes involved two adjacent factories. The first manufactured dry cadmium-nickel batteries for use in cameras, calculators, etc. It occupied 4 floors of a modern factory building, and employed about 500 workers, mostly young females in their teens and twenties, about 70% of whom were Malays. There was generally only one shift, from 7:30 a.m. to 5:00 p.m., Monday to Friday, with occasional overtime on Saturdays, but this was on a voluntary basis.

On Friday 23 July., 1976, at about 2:40 p.m., a female worker in the Final Assembly Section suddenly screamed and collapsed on to the floor, and lay there quietly. A few male workers helped carry her to a rest area in the canteen on another floor. On the way down, she started screaming and became violent. About 10 minutes later, another female worker started to scream. Soon afterwards, others followed. Because of this, that section of the factory was closed early, and overtime work scheduled for the next day (a Saturday) was called off.

On Monday, 25 Jul., at about 3:10 p.m., hysterical cases recurred. Altogether 6 workers were affected, of whom 4 were repeats, occurring in those workers who had the attack on

Friday, 23 July while 6 other workers were frightened.

A total of 28 spells occurred in 18 persons, between 23 to 30 July. A summary of the cases is given in the following table:

Date of Occurrence	No. of Spells		
	Hysterical	Frightened	Total
Fri. 7-23-76	6	1	7
Mon. 7-26-76	6(4R)*	6(1R)	12(5R)
Tues. 7-27-76	0	2	2
Wed. 7-28-76	0	1(1R)	1(1R)
Thurs. 7-29-76	2(2R)	2(1R)	4(3R)
Fri. 7-30-76	0	2(1R)	2(1R)
Total	14 (6R)	14(4R)	28 (10R)

Table 3: Summary of Cases in Outbreak (R= Repeat)
*R= Repeat Cases

All those affected were females,16 of the 18 were of Malay origin, one Chinese, and one Pakistani. The latter two experienced only fear.

On July 29, 1976, cases appeared in a neighboring factory manufacturing cameras, and employing 3,800 persons, 50% of whom were Malays. A female Malay worker complained of a headache, and suddenly fell down, screaming. (When interviewed after the incident, she denied seeing any spirit or figure, but said she had felt frightened). Three female operators and male supervisor then helped her to the office to rest. Two of these operators, while in the office, also experienced fear, and started to cry and scream.

The outbreaks in this factory lasted from 29 July to 4 Aug.'76. there were a total of 30 spells with 20 hysterical seizures and 10 frightened occurrences, affecting 23 persons of whom 7 had more than one spell each. All except one were females. The sole male involved only had a feeling of fear. Of the 23 affected, 22 were Malays, and one an Indian.

This episode also illustrates the great likelihood of recurrence of an attack in an affected person, especially among the first few cases. The first person to be affected on the first day was also the first case on the second day, after the weekend. And out of the 6 hysterical cases on the second day, 4 were repeats of those affected on the first day.

In this outbreak, urine samples from a few of the hysterical girls were taken for analysis for morphine, codeine and methaqualone. None was detected.

The hysteria outbreak affected, not a factory, but a large company engaged in providing telecommunications services. The outbreak occurred in a branch telephone exchange with 527 persons, all females, 203 or 38.5% being Malays. They worked shifts.

On 20 Oct., 1979, at about 4:30 p.m., one of the Malay telephone operators fainted, and suddenly started to scream. This was followed a little later, by a number of other women. Altogether 17 women were affected between 4:30 and 7:00 p.m. A doctor was called and he gave injections of tranquilizers and called the ambulance to take them to the hospital. A number of cases became violent and had to be physically restrained.

Altogether, between 20 Oct. and 8 Nov., 22 telephone operators were affected, giving 24 cases (12 violent and 12 frightened). Thirteen were Malays, 5 Chinese, 2 Indians and 2 of other races.

In 1978, there continued to be sporadic attacks among some of the telephone operators affected in the outbreak in Oct./Nov. 1977. One of the 2 women who had 2 attacks in that outbreak, (SA) had her second attack on 8 Nov. 1977 at another branch telephone exchange to which she was transferred a few days after she had her first attack. She was subsequently transferred again to another exchange where she had her third attack on 20 April 1978. On 26 Oct., 1978, she had her fourth attack at yet another branch exchange to which she was transferred.

The other woman who had 2 attacks in Oct./Nov., 1977 (H Y), had her third attack on 4 Jul.1978 at another telephone exchange.

A third girl (A M), after having an attack in Oct./Nov., 1977, was later transferred to another exchange, where on 18 April, 1978, she had her second attack, after having been told that morning that she would be transferred again to another exchange (which was far from her home). On 18 May 1978, while still at her previous place of work, she had her third attack. (She was at that time, in the third month of her first pregnancy).

Yet another episode involved a factory engaged in the manufacturing of ballbearings. It occupied a modern single story building, and employed 800 workers, 50% of whom were Malays. The incident occurred in one section of the factory, called the "Clean Room" where 107 female workers, working shifts, were engaged in "sealing" the ballbearings. Adjoining the Clean Room was a "Washing Room" where ballbearings were degreased in ultrasonic tanks using freon.

On Sunday, 30 Jul.1978, there were about 20 female workers doing overtime work in the Clean Room, commencing work at 8 a.m. At 9 a.m. some workers complained of an "acidic smell."

At 9:15 a.m., a worker (A) complained of dizziness, nausea, tearing and a blocked nose. She then visited the toilet, and while at the toilet she fell down. On return to the Clean Room, she fainted. At about the same time, another worker (Rh) also had similar symptoms. She visited the toilet, and when she returned to the Clean Room, she saw that A had fainted. With the help of two other workers,she carried her to the office. She then also fainted.

Ro, another worker, had symptoms of headache, nausea and tearing. When A fainted, all the workers in the Clean Room were evacuated, at about 9:30 a.m. On reaching the reception hall, Ro also fainted. A fourth worker (S), together with the rest of the workers, left the Clean Room. On reaching the reception hall, she also became ill.

The Company doctor was called. He found all 4 girls conscious, but "agitated". One of them (A), became very hysterical, crying, screaming, and hyperventilating; she developed carpal spasms. The doctor gave all 4 injections of valium (diazepam) and sent them to the hospital. Three of them were warded for observation. On examination, nothing abnormal was found, but A was very "upset" and crying. The next day, A developed hysterical hyperventilation, which subsided with an injection of valium.

The Management's explanation for the incident was that one of the workers (in another section) had forgotten to switch on the cooling condensing coils of the ultrasonic tanks in the Washing Room. Because of this, the vapor arising from the volatile freon was excessive. This was taken out by the local exhaust ventilation system at the mouth of the tanks, but as it travelled along the ducting of the system, because of the excessive vapor "overload", some of it leaked out of the system. As the ducting passed through the toilets in the women's restroom, the girls must have been affected when they went there.

This explanation was initially thought plausible, but interviews with the 4 affected workers revealed that they developed symptoms while in the Clean Room, and 2 of them did not even visit the toilet that morning. If, on the other hand, there had been leakage of freon vapor from the Washing Room into the Clean Room, (as a complaint of "aoidic smell" was made), then it was rather surprising that the other women in the room did not seem affected. The seating arrangement of the employees was such that the 4 affected workers were seated in various parts of the room. Also, the hysterical presentation was rather suspicious. This suspicion, however, was only strengthened when another similar incident occurred in December 1978 (see below).

On Monday, 18 Dec. 1978, soon after reporting for the afternoon

shift at 3 p.m., few workers complained of a chemical smell in the Clean Room. At about 3:30 p.m., the supervisor saw 2 Malay women seated at their tables, with their heads in their hands. They complained of giddiness. They were asked to go to the locker room just outside, to rest a while. A little later, a Malay man, in charge of the Washing Room, reported that the 2 women had apparently fainted in the locker room and he asked the supervisor to evacuate all the workers from the Clean Room. There was a lot of commotion as the employees left the room. Outside the room, some female workers fainted, and were carried to the Conference Room to recover. Oxygen was administered.

There were a total of 8 women, all Malays, being resuscitated in the Conference Room, including the first 2 workers who had fainted in the locker room. The doctor was called. Two of the women became violently hysterical, screaming and struggling, and had to be physically restrained. (One of these was A, who was also the first worker who had complained of giddiness and later fainted in the locker room).

After injecting them with valium (diazepam), the doctor called the ambulance to send them to the hospital. Seven were warded for observation, while the eighth (A) was observed for an hour and sent home with one day's sick leave. The doctors found nothing abnormal on physical examination of the 7 workers, although they complained of dyspnea and a "suffocating" sensation. Chest X-rays, and blood gases done on a few of them, were also normal. All were discharged the next day.

Of the 8 women affected, 3 of them had been affected in the earlier incident in July. The first victim in the December incident was not only among the 4 affected earlier, but she was also one of the 2 persons who became "violently hysterical". In the hospital the next day, she also developed "hysterical hyperventilation".

The outbreaks of mass hysteria described presented, with some exceptions, a general pattern:

1. A sudden onset (without warning).

2. A high degree of "infectiousness" or communicability, spreading not only from person to person, but also from one department to another, and even from one factory to another.

3. A high rate of "repeats", i.e., the tendency for a person, after having experienced one attack, to get another.

4. A likelihood for those affected on the first day of an outbreak to be involved in other attacks on the second and subsequent days.

5. Although a psychological phenomenon, psychological counter-measures did not prove effective, e.g., the calling in of a "bomeh",or medicine-man, to exorcise spirits.

6. An outbreak would subside on its own, usually within a week or so.

There were 2 main modes of presentation of cases:
1. The violently hysterical - presenting with screaming, crying and struggling. The violence might be aggravated by attempts to restrain them. During such attacks, they were usually incoherent and ignored questions and attempts to calm them. Injection of tranquilizers did not seem particularly effective. They might or might not have visual hallucinations.

2. The frightened - presenting with an unexplained feeling of fear. They might also complain of feeling cold, numb or giddy.

In addition to the above, another presentation, which is more a "subgroup" of the violently hysterical, was that of a "trance state", during which the person acted as though she were somebody else, with a spirit of "jin" speaking through her. This presentation, lasting only a few minutes, was uncommon, and was observed only during the Jan. 1973 out-break.

This interesting phenomenon could be partly explained by the cultural background and traditional belief in spirits by the Malays. According to this belief, living things have a "vital force", which they call "semangat", which plays an important part in maintaining health. Its presence in the body is be-lieved to exert a force externally, preventing evil spirits ("hantus") from acting on the body. The hantus are believed to be ubiquitous but invisible, and it is only when the force of the "semangat" is weakened, that the spirits can gain entry into the body. Sudden fright or shock is believed to drive the "semangat" out of the body, and hence render the person vulnerable to the evil spirits. Females are believed to have weaker "semangats". (Children with fits are thought to be possessed by a hantu. When a child's eyes roll upwards during a fit, he is believed to be looking at a hantu on the ceiling about to attack it). (KassR, 1961/62).

Malays generally also believe in "bemehs", some of whom have the "jin" (or genie). These bemehs are able to make charms for good or evil.

In spite of such beliefs, however, it was noted in several outbreaks, that the calling in of a bemeh to exorcise evil spirits did not stop the outbreak, which subsided on its own. Although an outbreak usually lasted about a week, with daily recurrences, there may be a lapse of a few days with no attacks,

and this may give rise to a "false sense of security". Thus, vigilance must be maintained for at least a few days after the last case has occurred.

All the outbreaks occurred in large modern factories or work places. The general work environment was clean, airconditioned, with good lighting and ventilation. In most cases, little or no chemicals were present or handled. It thus seemed that the physical work environment might not have been a factor in such outbreaks. Shift work and night work also did not seem to be a factor as outbreaks occurred on all shifts both day and night. But it was noted that all the factories involved "assembly line" type of work, which was monotonous and repetitive. Some factories in the U.S.S.R. and Sweden have, in fact, introduced modifications in the assembly line type of work, in an attempt to reduce the boredom of repetitive work.

Almost all the cases involved females, but the phenomenon was not confined to the young or any particular age group, as the age distribution of the affected workers reflected the age composition of the general worker population in those factories. (It was perhaps fortunate that few men were affected, as those affected seemed to possess extraordinary strength!)

Most of the persons affected were Malays. (However, in few outbreaks, statistical evaluation of the racial composition did not seem to find this a significant factor. But as the numbers involved in each outbreak were relatively small, this must be taken with caution.) It was noted that such outbreaks usually occurred in factories which had a predominantly Malay worker population.

In general, workers who were affected on the first day of an outbreak were more likely to get repeat attacks on the second and subsequent days. It should therefore be considered in any outbreak, to remove those affected (especially on the first day) and keep them away from the workplace for at least a week (or until the outbreak has subsided). This is important in view of the fact that one attack can so easily trigger off an attack in another person. Thus persons involved in one attack should be considered "highly infectious", to be strictly segregated and isolated. (Chew, 1978)

To find a "triggering" event that initiated an outbreak was very difficult. But in several outbreaks, there were, as far as we could ascertain, no recent change in management or supervisory personnel, or in conditions or arrangements of work. In the 1978 outbreak in the ballbearing factory, however, several of the affected workers, when interviewed discreetly, confided some complaints about the management, and excessive work pressure, including excessive overtime. These complaints were greatest in the clean Room, where the outbreak occurred.

This particular section had a higher labor turnover than the rest of the factory.

This set of circumstances may partially explain why the cases occurred only in that particular part of the plant and did not spread to the rest of the factory, as in other outbreaks.

The presentation of cases in the ballbearing factory was also rather interesting and unusual. Unlike the usual presentation of sudden screaming and fainting, the affected workers complained of headache, giddiness, etc. and then fainted. This had initially been attributed to leakage of freon vapor, and it seemed a likely explanation. However, in retrospect, after 2 episodes of a similar nature had occurred, affecting, among others, the same workers, while other workers were uneffected on all occasions, others seemed relatively unaffected, it would seem unlikely that freon exposure alone could account for all illness occurrences. Clinical and laboratory investigations at the hospital were negative.

It is thus important to bear in mind that an outbreak of mass hysteria may mimic some other condition (and vice versa), and it may be in retrospect, in the light of accumulated circumstantial evidence, that the actual cause is discovered.

The outbreak at the telecommunications workplace involving telephone operators was also interesting. Recurrence of attacks in a few of these persons occurred as isolated incidents and did not spread to other people. Frequent transfers and change of workplaces seemed to be a factor in these attacks.

A subsequent study (Chan et al, in press) found that the telephone operators affected in the 1977 outbreak, had a slightly higher Neuroticism score than the controls, but this was not statistically significant. They also had a much higher Extroversion score and a lower Intelligence score, but $p > 0.05$.

Having looked at the patterns of various mass hysteria outbreaks, we would next have to identify what factors would "trigger off" an outbreak, and what factors would make certain persons more "susceptible" to an attack than others.

With regards to the latter, besides studying such demographic data as age, sex, race, religion, duration of employment, educational levels, etc., possible areas of study would include the psychological makeup and personality, intelligence level and level of job satisfaction.

An explanation for such outbreaks affecting mainly Malay female workers has been advanced by Ackerman (1978). The traditional upbringing of Malay females is very conservative.

Thus the social norm expected of them is to be submissive, obedient and "well controlled". Self-expression is not a characteristic to be encouraged,it being preferred that girls remained "malu" (i.e., shy and retiring).

Malay females, especially if from the rural areas, may encounter psychological stress in adapting to an industrial setting. Added to this, they may also have dissatisfaction over certain work conditions and styles of management (e.g., strict discipline). But because of their upbringing, it is against their nature to openly rebel. Direct confrontation with authorities would bring about social disapproval. It is also possible that monotonous work would leave them much time to "brood over" any unhappiness they may experience.

Traditional Malay culture includes strong beliefs in spirits and spirit-possession. It is thus postulated that, because of internal conflict generated by unhappiness and the inability to self-express or vocalize their unhappiness, for fear of social disapproval, the Malay women react by mass hysteria, i.e., it is used as a "safety valve". Because of the traditional belief in spirit possession, such behavior, far from being condemned, is accepted, and there is therefore no social disapproval. On the contrary, it generates concern and sympathy on the part of parents, colleagues and perhaps even managements.

However, much more study is required into the various aspects of the phenomenon.

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PANEL PRESENTATION

OCCUPATIONAL PHYSICIANS:

Elva Elesh
Mitchell Singal

INDUSTRIAL HYGIENISTS

Paul Pryor
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Mr. Moseley: I would like to open our contribution to the symposium on mass psychogenic illness in industry with some background information about the National Institute of Occupational Safety and Health, or NIOSH, for anyone in the audience who may not be familiar with this federal agency.

In order to assure safe and healthful working conditions for men and women, Congress enacted the Occupational Safety and Health Administration Act on December 29, 1970. The responsibility for the enforcement of the provisions of this Act belongs to the Department of Labor's Occupational Safety and Health Administration. However, included in this Act was a provision for the Secretary of Health, Education and Welfare "to conduct research and related activities, experiments and demonstrations relating to occupational safety and health, including studies into the motivational and behavioral factors, and innovative methods, techniques and approaches for dealing with occupational safety and health problems." Sections 20 and 21 of the Act outline these provisions and provide the basis for the formation of the National Institute for Occupational Safety and Health.

Included in the variety of research and service functions NIOSH performs, is the Health Hazard Evaluation Program, which was developed to implement the provisions of Section 20 (a) (6) of the Act; specifically "to determine, following a written request by any employer or authorized representative of employees, whether any substance normally found in the place of employemnt has potentially toxic effects in such concentrations as used or found". "Substance" in this case has been defined as chemical substances only. Safety and physical hazards are not currently covered by the Health Hazard Evaluation Program.

Health Hazard Evaluation studies are terminated with the issuance of a Determination Report, which includes an assessment of the subject hazard and recommendations to remedy or reduce the exposure to the hazard. The Health Hazard Evaluation Program is responsive not only to requests regarding substances for which employee exposure may not be regulated and for which there may be no presently known effects of exposure. Possible results of these studies are NIOSH current intelligence bulletins, Department of Labor emergency temporary standards, and perhaps promulgation of new federal standards.

A regulation, published in the Federal Register, (42 CFR Park 85) describes the conditions and procedures whereby NIOSH conducts the Health Hazard Evaluation Program. The Hazard Evaluation and Technical Assistance Branch of NIOSH conducts these health hazard evaluations.

As one may gather from the title of the Branch, a service known as Technical Assistance is also provided; the regula-

tions governing this program are given in 42 CFR part 85 (a). The Technical Assistance portion of the program was designed primarily to provide industrial hygiene and medical expertise to State and other Federal Agencies who were not capable of providing their own in-house technical expertise. However, Federal Executive Order #11807 and Section 19 of the Act have excluded State and Federal Agencies from the provisions of the Act and have charged them with the responsibility of providing their own comprehensive occupational safety and health programs. In practice, however, technical assistance is still provided to State and Federal agencies on a limited basis. Although it is not specifically mandated, technical assistance is provided, when requested, to employers other than State and Federal agencies. Also in practice, the technical assistance program has provided a mechanism for the Branch to evaluate physical hazards.

Copies of the rules and regulations governing occupational safety and health investigations of places of employment, that is Part 85, the Health Hazard Evaluation Program, and Part 85 (a), the Technical Assistance Program, are available at the NIOSH Information Booth, and I believe that's No. 107 and 108.

The Hazard Evaluation and Technical Assistance Branch currently consists of industrial hygienists, physicians and other health professionals. The Behavioral and Motivational Factors Branch of the Division of Biomedical and Behavioral Science is relied upon for the development of the behavioral portion of those studies where MPI is suspected. A detailed discussion of this Branch's input will be presented later.

Before any field visit is arranged, a decision is made, based on the available information, whether or not one, all, or some combination of the three groups -- industrial hygiene, medical and behavioral-- should be initially involved. This decision is based, among other things, on the amount of environmental or medical data previously gathered and available, the number and severity of the reported symptoms, the potential for toxic substance exposure, and the time interval between the illness episode and subsequent visit by NIOSH.

Since the program's inception in 1972, the Health Hazard Evaluation and Technical Assistance Branch has received over 1,100 health hazard evaluation requests. Of these, over 500 final reports have been disseminated. Currently, the Branch is involved in approximately 130 active studies.

In at least 19 of these 1,100 studies, it was determined at that time or, in retrospect, that the problem was probably mass psychogenic illness. However, there has been an increase in the number of cases we've investigated over the last few years, although as Dr. Markish has indicated, MPI is not a

new phenomenon. Perhaps this is due to the increased publicity attending such situations or to the increasing amount of coping necessary to function in today's society.

The dates for the initial episode of illness, in each plant investigated (some investigations involved multiple episodes of illness) were distributed as follows: 1972-2; 1973-1; 1974-2; 1975-1; 1976-1; 1977-5; 1978-7.

The settings for these episodes involved; offices and similar non-industrial settings - 9; electronics assembly plants -4; shoe factories - 2; other light industrial operations, manufacturing and packaging/distribution - 2 each.

Our first speaker, Dr. Singal, will discuss some common epidemiological and medical characteristics often found in mass psychogenic illness, and certain considerations in the diagnosis of mass psychogenic illness.

DR. SINGAL: I will first discuss some general concepts concerning mass psychogenic illness and then I will summarize the findings in 19 NIOSH investigations.

The diagnosis of mass psychogenic illness is not merely one of exclusion. That is, it is not based merely on the absence of significant environmental and medical findings. Nor does the finding of an environmental contaminant necessarily exclude the diagnosis of mass psychogenic illness. For example, the hyperventilation syndrome is fairly characteristic clinical picture, and the presence of this syndrome in several people is highly suggestive of psychogenic illness. I will therefore, discuss it in some detail as an <u>example</u> of psychogenic illness.

"Hyperventilation" is a physiologically unnecessary increase in the frequency and/or depth of breathing. While it may be due to various physical or physiologic disorders, certain drugs, or carbon dioxide levels in excess of 1% (that's twice the current TLV), the usual cause is acute or chronic anxiety. The term "hyperventilation syndrome" refers to a cluster of symptoms often seen in someone who is hyperventilating. These include such non-specific symptoms as dizziness or lightheadedness, fainting, fatigue, and weakness; and more specific symptoms such as chest tightness, shortness of breath, rapid heartbeat, palpitations, dry mouth, numbness or tingling of hands, feet, and more characteristically, about the mouth; and in severe cases, carpopedal spasm (those spasms of the hands and feet which may look to a layman like convulsions). Symptoms may persist for hours or days, and recurrences are not uncommon.

While hyperventilation lowers the arterial pCO_2 and increases the pH, these changes alone do not seem to cause the typical symptoms, at least in healthy volunteers. Thus, the physiologic

basis for some of the symptoms is not clear.

In this presentation, the terms "hyperventilation" and "hyperventilation syndrome: will be used interchangeably. Let me emphasize again, however, that the term "psychogenic illness" will not be limited to hyperventilation.

A feature that suggests mass psychogenic illness is the mode of transmission. A characteristic of mass psychogenic illness is that one is affected not necessarily because of being in proximity to the source of an alleged exposure, but rather only after having knowledge of either the alleged exposure or the fact that someone else was exhibiting signs of illness. Cafeterias, rest areas, etc., may serve as the setting for transmission of such information in an institutional setting. An even better sign, although not always easy to specifically document, is the association of illness with actually seeing someone else exhibiting signs of illness.

Thus, when questioning people involved in what may be an episode of mass psychogenic illness, crucial questions to ask are: (1) Before you became ill were you aware of illness in others? and (2) Before you became ill did you actually see someone else ill? Please note that it is important to obtain this information from both affected and unaffected people. It is not merely positive responses to these questions that help document mass psychogenic illness, but the <u>differences</u> between affected and unaffected people in the positive response <u>rates</u>.

Another factor in deciding whether the episode of mass illness is primarily psychogenic is the pattern of onset and severity of symptoms. If there are a number of people with relatively severe acute symptoms, it is unlikely that the illness is due to a low level of a contaminant. This is particularly true if the symptoms are of sudden onset and begin after the people have left the source of exposure.

Another clue is the presence of moderate to severe symptoms without the usual symptoms caused by the alleged contaminant. For example, if a substance were ordinarily a respiratory tract irritant and caused coughing, it would be unlikely that exposure to this substance would cause a variety of respiratory and other symptoms--but not coughing.

It is sometimes hypothesized that the symptoms occurring during outbreaks labeled "mass psychogenic illness" may be due to unusual susceptibility to low, perhaps even undetectable, levels of environmental contaminants. In order to discuss this, it is necessary to differentiate the three types of hypersusceptibility.

First, there is immunologic hypersusceptibility, commonly called

allergy or hypersensitivity. A period of time is needed for sensitization to the allergen before symptoms are manifested. This may be several days to several years, depending, among other things, on the frequency and level of exposure. It would thus be so unlikely that several people would first manifest symptoms within minutes of each other that this explanation can be essentially dismissed as a cause of mass psychogenic illness.

Second, there are idiosyncratic reactions. These are, by definition, uncommon, and are symptoms and signs that differ markedly from those typical of exposure to the particular substance. Most people will not be so affected regardless of dose. Thus, this type of unusual susceptibility cannot readily account for more than a small proportion of people being affected after a single exposure, nor for several people being simultaneously affected after chronic exposure.

Third, there is the hypersusceptibility due to individual variation in the dose needed to produce a given effect. Occupational exposure standards take this phenomenon into account and are ordinarily set to prevent at least the overt adverse effects from occuring in most people. Therefore, if the exposures are well below such standards, it would be unlikely that more than a very small proportion of people would be affected. Now admittedly, when an investigation of an outbreak of this type takes place after the fact, exposures cannot be directly measured if the circumstances at the time of the outbreak were atypical. However, reasonable estimates can be made by extrapolating available data.

Another proposed explanation of mass psychogenic illness is that it represents neurobehavioral effects of environmental contaminants. While this could conceivably account for symptoms in some individuals, the initial case in an outbreak, for example, it does not readily explain the phenomenon in general. If the neurobehavioral hypotheses were true, then presumably (1) mass psychogenic illness should occur more frequently in work places where exposures to putative etiologic substances are greater, and (2) attack rates in any given outbreak should be higher among groups with greater exposure. As the following discussion will illustrate, neither has been the case in NIOSH's experience.

The population at risk in the studies NIOSH has been involved in, that is, the number of employees working in the plant or the specific area where the outbreak occurred, has ranged from fewer than 10 people to 1,000 people. Typically, however, these episodes occurred in establishments employing 200 to 500 people. The attack rates have ranged from 5% to 83%. However, there were differences from investigation to investigation in the definition of what constituted a case, in the methods used in determining the number of people affected,

in the definition of the population of risk, and finally, in the professional scope of the investigation, that is, industrial hygiene, medical, behavioral, or some combination.

Furthermore, the attack rates were sometimes estimated on the basis on nonquantitative data, data of questionable validity, and results of questionnaires in which there was a low response rate.

Typically, but not exclusively, episodes of mass psychogenic illness, in NIOSH's experience, have occurred at plants engaged in manufacturing or other activities where the work involved repetitious tasks and high-volume output. The work forces at these plants were composed primarily of women. Thus, the people affected by the episodes NIOSH has investigated have been primarily women. However, there is insufficient data to determine whether attack rates are greater in women when both men and women are exposed to the same socio-economic and occupational environment.

In three of the 19 investigations, the problem was limited to "bites" and/or itching, and the alleged hazard was insects. In the other 16 investigations, the alleged hazard was presumed to be some chemical substance in the environment. In three of these, chemicals were used in the area where the episodes of illness occurred, or potentially toxic substances were generated by the production processes or other activities in the plant. However, in 7 of the 8 other investigations, there were no apparent sources of chemical contamination of the plant environment. In view of this, the source of the alleged chemical hazard was sometimes thought by the employees to be outside the plant. In the remaining investigation, which will be discussed later in the report, there was actually evidence of a transient external source of contamination.

In most outbreaks of mass psychogenic illness, again in NIOSH's experience, there has been a readily identifiable precipitating event, what will be referred to later in this report as a trigger mechanism; that is, some unusual event to which the symptoms are attributed. In 14 investigations, the precipitating event was apparently an odor, either an unusual odor or an odor ordinarily present but not as strong. In two of these cases and one other, there was also visible vapor or smoke preceding the incidents. In the remaining four cases, there was either no apparent precipitating event or none recorded in the report of the investigation.

For the 16 investigations in which the epidemic disease was not considered to be insect bites, the most commonly reported symptoms were: headache (14); dizziness/syncope (12); nausea (11); drymouth/throat (10); eye/nose/throat irritation (9); sleepiness/drowsiness(7); weakness(7); and numbness/tingling(6).*

*The number in parenthesis indicate the number of plants investigated in which this symptom was a major complaint.

In nine of these 16 investigations, the combination of symptoms were not suggestive of hyperventilation or there was insufficient data to conclude that the epidemic illness was hyperventilation.

In only two investigations was actual observation of another ill person documented as the mode of transmission. In the others, such an association was either not sought or not found, in some cases, because of the lack of data concerning unaffected persons.

A major problem with compiling this summary has been the lack of uniformity in collecting the data at the time of the investigation. In some investigations, the possiblility of mass psychogenic illness was not recognized at the time of the investigation and the conclusion was reached afterward. Clearly, in these cases, the pertinent epidemiologic questions were likely not to have been asked.

Similarly, information obtained from medical records and other secondhand reports is generally limited to the data that was sought by that particular physician or investigator. In other investigations, data was obtained only from people who sought medical attention. This may underestimate the number of affected people by excluding those who may have been symptomatic but did not seek medical attention.

Finally, there were investigations in which little or no data was obtained from people who were not affected by the illness. Without this information, the mode of transmission cannot be adequately documented, nor can the perceptions of the precipitating events and other conditions by the unaffected people be compared with those of the affected people.

Dr. Elesh will now discuss some historical aspects of mass psychogenic illness and present the first case study.

DR. ELESH: Although, as Dr. Singal mentioned just now, in industry outbreaks of mass psychogenic illness have been associated with a predominantly female workforce, of limited formal education engaged in repetitive tasks involving the use of chemicals, the nature of which is unknown to the workers, this is not universally so. In the public at large, the hyperventilation syndrome has been documented as occurring almost equally between the two sexes.

There have been scores of epidemics of the hyperventilation syndrome in the past in schools, hospitals and other institutions, and they were the subject of a symposium at the Royal Society of Medicine in London last spring. One of the expressed aims of the symposium was to discuss the question of whether an epidemic of this kind "is organic, psychogenic or hysterical in origin." It is barely credible that nobody

was invited to present the case for either hysteria or psychogenesis. To admit the possiblility of a psychogenic origin would be to let the medical side down.

An epidemic of this nature occurred in the Royal Free Hospital Group in 1955. It affected nearly 300 people with malicious selectivity; almost all the victims were members of the staff! Attending physicians and residents were naturally considered to be immune once a diagnosis of mass hysteria had been considered, until some of the internists who had been looking after many of the cases had the illness most severely. A new clinical entity had, therefore, to be summoned, and "epidemic benign myalgic encephalomyelitis" was born.

This emphasizes the point that MPI is no respecter of persons and that it may affect white-collar workers as well as blue-collar.

Other ways in which MPI may manifest itself include symptoms mimicking organic heart disease, dermatitis and nerve-muscle disorders. During World War I, the terms "effort syndrome" or "Soldier's heart" (otherwise known as DeCosta's syndrome), gained recognition. The diagnosis was a common one in the trenches. Symptoms included chest pain, difficulty in breathing, faintness, generalized weakness, and prolonged anxiety. Considering the conditions of trench warfare, it is not surprising that the "effort syndrome" was, at times, epidemic.

There have also been a number of reports in which groups of workers have developed skin lesions which were difficult to differentiate from organic disease. In one such episode, which Dr. Markish referred to earlier, one worker with an allergic skin disorder persuaded her colleagues that this condition was caused by exposure to substances in the work environment. Her fellow workers then developed various forms of skin rashes. I should like to stress the word "various" as, in outbreaks of mass psychogenic illness, one positive clue to the diagnosis is that the symptoms and signs are heterogeneous, compared with exposure to toxic substances and/or agent, where the workers'reaction is homogeneous, to a greater or lesser degree.

Nevertheless, when investigating these outbreaks, environmental factors must first be ruled out. A dermatologist patch-tested all the affected workers with all chemicals to which they were exposed, with negative results, which "cured" this epidemic. It may be significant that the originator of the outbreak was initially reluctant to be patch-tested, and, when informed of the negative results, was the only worker who would not accept these findings.

A similar outbreak occurred in an all male work force. A dermatologist was asked urgently to see eight men with "occupa-

tional dermatitis." The men intended to stop work unless some action was taken. They were aged between 18 and 53; the average age was 30. They were tool operators and were in contact with cutting oils. Some weeks previously the oil had been changed from the type which they had used for years. At the time the oil was changed, one of the workers who had had atopic aczema for a long time had an exacerbation on the upper trunk for no obvious reason, and he took time off work. His fellow workers then examined their own skin and found, on examination, spots, mildly itchy at times, on their backs, and less so on their chests. Apart from skin blemishes common to the public at large, none of these workers had any evidence whatsoever of occupational dermatitis. The employees attributed their alleged skin problems to the new oil, but there was no evidence for this.

By the rapid attention to the problem and by frank discussion with management, and with the men as individuals, as well as in a unit, closure of the plant was avoided and the men were relieved to learn that they had no occupational dermatitis.

These examples demonstrate, as will be noted in the following five NIOSH investigations into mass psychogenic illness, a loss of a sense of individuality -- employees working,as it were,in a unit.

Since joining NIOSH in 1977, I have been involved in the investigation of three outbreaks of mass psychogenic illness, one of which I shall now describe. This occurred in an electronics plant which has been in operation for approximately two years, and employes a non-unionized work force consisting of approximately 350 female and 50 male workers. The plant had operated on a single day shift, extending from 7:00 a.m. to 3:30 p.m., for approximately five months. Prior to this, the plant operated both a morning and afternoon shift. The introduction of a new automated conveyor-paced system was responsible for the move to the single shift schedule, in that production has more than trebled with no change in the size of the over-all work force.

Production takes place in one large room, beginning with the cut and form department where wires for individual electronic components are snipped and bent by machine for delivery to the manual insertion line. Adjacent to the cut and form department are automatic insertion machines, where circuits are stamped into pre-cut circuit boards. From here the boards move to manual insertion lines which run parallel to the automatic insertion machines. Here the components from the cut and form department are manually inserted into the circuit boards as they move along a U-shaped conveyor. There are four such manual insertion lines running parellel to each other. On each line, workers face one another across the conveyor, the speed of which is set by the line foremen. Insertion line

employees are expected to work two hours continuously and, although relief personnel are available, the employees are encouraged not to leave their stations unnecessarily. After the components have been inserted, the circuit boards are transported to the soldering and degreasing stations where the circuits are maching-soldered, and dipped into a cleaning solvent.

Running at a right angle to the manual insertion lines is the in-process inspection line. Rejected units are sent to an adjoining re-solder line, where they are manually re-soldered and sent back for inspection. Following inspection, each unit undergoes a final degreasing.

The plant is modern, well lit and well ventilated, with very good housekeeping.

In late 1977 on a Friday moring, approximately 40 female workers complained of feeling unwell with a variety of symptoms referable to the respiratory, gastro-intestinal and central nervous systems. Headache, lightheadedness, dry mouth, sleepiness, weakness and nausea were complained of most frequently, both singly and in combination. The workers attended the local hospital's out-patient department, where they were examined, and blood was drawn for chemical analysis. Most were released, but a few workers were admitted for observation, as hospital personnel did not know to what chemicals, if any, the women had been exposed.

The solvent used in the line degreasers is a halogenated hydrocarbon, the Freon R TMS, a mixture of Freon R TF blended with methanol. Other chemicals in use were a solvent blend flux-thinner and resin foaming flux in the soldering machines.

Central nervous system depression may occur in cases of exposure to very high concentrations of fluorocarbons, and symptoms from over-exposure may manifest themselves as lightheadedness, dizziness and chemical "drunkenness".

Methanol is virtually non-irritating to the eyes and upper respiratory tract at levels below 2,000 parts per million. Ethanol is irritating to the eyes and nose at extremely high concentrations.

Management immediately contacted OSHA, and a variety of other federal and state agencies, as well as private consulting firms. In repeated environmental sampling, no substances and/or agents capable of causing the workers' symptoms could be detected in significant amounts.

The plant closed, as usual, over the weekend. The workday on the following Monday was essentially uneventful.

On the Tuesday morning, a member of management ran a "burn test" on a piece of plastic used as housing for the electronic components. On detecting the odor, another member of management, not usually present on the floor, ordered the evacuation of the plant. He himself complained of dizziness. This occasioned some anxiety and panic among the work force, a number of whom reported, or were taken, to the same local hospital. The plant immediately closed down, and management sought advice from 19 public and private agencies.

Again, in repeated environmental sampling,including analysis of drinking water and investigation of the cafeteria facilities, no substances and/or agents capable of causing the workers' symptoms could be detected. The chemical nature of the solvents and fluxes used have been described. Considering the toxicity of each, the amounts likely to be encountered were not sufficient to have caused the reported symptoms. There was no report of spillage(s) or mechanical failure coinciding with any of the illness episodes.

Being aware of NIOSH's experience in incidents of a similar nature, later that day, one of the private consultants suggested that the company request NIOSH's assistance.

On the following day, NIOSH regional hygienists visited the plant, conducted an extensive walk-through survey, evaluating all phases of the operation, and acquired a list of chemicals used and material safety data sheets. Ventilation and climate control systems and general operating conditions were reviewed with plant personnel.

Arrangements were then made for me to visit the closed plant the next day, to discuss all medical findings with a physician who had been retained by the firm to investigate the two episodes.

Prior to my arrival at the company, I telephoned a doctor who had examined some of the employees at the local hospital. He affirmed that no abnormalities were detected in the workers, either on physical examination, or in laboratory testing of blood samples, which could relate to occupational exposure to toxic substances and/or agents.

The following morning, in company with two NIOSH industrial hygienists, I visited the local hospital, and with the physician referred to earlier, reviewed the medical records of 85 workers who had been seen at the hospital.

I was informed that six workers showed some elevation of blood cyanide levels. As blood cyanide levels are not accurate measurements of cyanide absorption, the laboratory which had reported this finding was to carry out the more specific determination of blood thiocyanate levels, the results to be

available within a few days. Unfortunately, the physician retained by the firm, whose laboratory had reported the elevated blood cyanide levels, saw fit to give a press hand-out to the effect that workers at the plant had been poisoned by cyanide, which generated much publicity and worker anxiety. Since there was no cyanide usage at this plant, the only assumption one could make, at the time, was that these workers were smokers, but no smoking histories had been taken by the consulting physician. Therefore, no correlations could be made regarding smoking as the cause of the elevated cyanide levels.

When I eventually managed to contact the toxicologist at this laboratory to ascertain the blood thiocyante levels, I was informed that this test had not been performed. The explanation offered to me was that, due to the large number of samples being analyzed, the one lab worker who had reported all the six elevated cyanide levels, had improperly performed the test, thus leading to inaccurately high readings. It is unfortunate that so much publicity was given to alleged cyanide exposure without correction when the error was discovered.

Also, to compound any management/labor frictions, there were newspaper reports to the effect that management had stated that the workers were imagining the incidents, and that a psychiatrist would be brought in to evaluate the problem. This generated considerable hostility in the work force.

To resume the report on NIOSH's investigation: after leaving the local hospital, the three NIOSH officials visited the plant. The physician with whom I was to discuss all medical and toxicological findings--the one who had circulated the cyanide poisoning diagnosis--had departed the previous day and I was therefore unable to resolve this with him.

During the plant walk-through, I asked the plant manager what, if anything, of an unusual nature had occurred prior to, or coincidental with, the first incident. He informed us that the previous evening the climate control system was modified from summer to winter conditions which entailed introducing steam heat into the upper area of the plant.

As you well know, warm air,being lighter than cold air, circulation of the warm air downward would initially be delayed, possibly allowing chemical odors to accumulate in the region of the workers' breathing zones, i.e., an indoor inversion. As time passed, of course, this situation would rectify itself.

During a later meeting with management, the NIOSH investigators were informed that a meeting was to be held that evening (a Friday) with workers to inform them that no hazardous substances and/or agents had been detected, in concentrations which could

pose a potential hazard to health. Thw workers would also be told that additional ventilation equipment had been added to make the plant even safer.

The theory of the indoor inversion was propounded to, and accepted by, management.

Realizing that the workers would be genuinely, if unjustifiably, concerned about possible hazards to their health, I requested of the management that, apart from NIOSH personnel, no one should be in the work area who was not normally present at start-up on the following Monday, as this could well undermine the credibility of managements' assurance that no toxic agents were present in significant amounts in the work environment. Other investigors agreed.

With the same consideration in mind, the NIOSH hygienists decided that instrumentation would be on hand to identify any causative agents, should another "incident" occur, but to allay any possible fears in the work force, no sampling would be conducted.

On Monday, the three NIOSH officials were in the plant prior to start-up. Management informed the workers that a government physician, with two government inspectors, was present in the work area for private consultation.

During the work period I spoke to the supervisors who, incidentally,had experienced nothing untoward during either incident, and to many of the workers, reassuring them that, in my professional opinion, there would be no long-term hazards to their health associated with either incident as, even if there had been a transient increase in the environmental concentrations of chemicals used in this plant, the effects, if any, would be temporary and would disappear when exposure ceased. The first aid room was put at my disposal and several workers sought private consultation.

At the beginning of the shift, I encountered some hostility among the workers, who asked if I were a psychiatrist. On being informed that I was just a "common or garden variety" doctor, they became more communicative.

Work practices and general conditions were observed throughout the work periods, without a show of environmental measuring devices, as mentioned earlier.

Other than for employees having existing medical conditions, unrelated to work exposure, the day was uneventful, with no workers becoming ill. At the end of the work period, it was requested of me that I go to one of the directors' office. Upon entering, I was faced with a blaze of lights, television cameras, microphones, and a room full of news reporters.

It was interesting to me to note the different approaches to the subject of the two TV reporters. One was obviously interested in my views as to the health hazards, if any, present in the plant; the other was unwilling to accept that the workers were not being poisoned--specifically with cyanide. These attitudes were obvious in the coverage of the interview given in the news on two channels. One gave a factual, unbiased report, whilst the other resorted to sensationalism, showing headlines such as "Mystery Unsolved in Poison Factory". It was feared that this might rekindle the workers' apprehension and jeopardize the rapport which had been established with the work force.

During the morning, I introduced the team from the Division of Biomedical and Behavioral Sciences to the Shop Safety Committee, and to some of the workers. They were greeted with overt hostility. This was obviously not the time to bring in a group of psychologists.

Again, no illnesses were reported other than scattered complaints of random worker ailments, such as sore throats, colds, etc.

I trust that this description illustrates the point that many workers are genuinely,(though sometimes unjustifiably), concerned about the potentially adverse effects upon their health, of exposure to substances, and/or agents, in their working environment.

In this particular instance, it could well be that the workers noted an increased, though non-toxic, accumulation of odors, which caused an understandable alarm. Increased production without an increase in work force and variable line speeds could be stressful. In this operation, the line supervisors determined the speed of their respective lines. Some workers approved of this arrangement, while others did not.

The symptoms of which they complained are consistent with severe anxiety, which, if not allayed, may lead to the continuance of complaints of ill health, and possibly precipitate another "incident".

It was recommended to management that an explanation, in readily-understood terms, be given to the work force as to what could have triggered the outbreak. Unless some explanation is given to them, the workers fear that whatever caused the "incident" has not been detected and the scene remains set for another "incident".

The workers would also be informed as to the nature of the chemicals used in the plant, the potential health hazards, and good hygiene practices.

The hiring of a full-time nurse was also recommended and approved by management.

Although none of the numerous hygienists who investigated this plant had detected any levels of chemicals which could pose a hazard to health, nevertheless, a recommendation was made to monitor the work environment for possible exposure to chemicals in use, for example, halogenated hydrocarbons and alcohols, on a regular basis, and record all measurements. This should especially be performed during any process changes, or heating/cooling modification.

There have been no further incidents in this plant.

Our next speaker is Dr. Singal, who will present a review of one incident he was involved in.

DR. SINGAL: This example of mass psychogenic illness occurred in a warehouse work force which handled "permanent press" clothing. Clothing from approximately 400 different manufacturers, both domestic and foreign, was handled in this operation. The work process involved receiving boxed or hanging clothing directly from the manufacturer, sizing and ticketing the merchandise, and then processing it for distribution to retail stores.

There were no chemicals used in the workplace, with the exception of small amounts of solvent in spray cans used to clean ticket stamping machines. However, permanent press fabrics continue to off-gas certain organic compounds, primarily formaldehyde, for some time after treatment with the resins used to impart the permanent press characteristics. No alterations of clothing and no manufacturing was performed in this warehouse.

The operation was housed in a 110,000 square foot warehouse. There was a partial second level consisting of steel grate walkways. Clothing was moved about the warehouse manually with the aid of overhead conveyors. The warehouse was not air conditioned and it was reportedly not uncommon for summertime indoor temperatures to exceed 90 degrees. The warehouse was heated in the winter by a gas-fired furnace and ventilated year around by one centrally-located 86,000 cubic foot per minute supply fan and by whatever air exchange took place when the 10 loading dock doors were opened. There were also four 10,000 cfm exhaust fans located along one side of the roof to help relieve the positive pressure buildup caused by the supply fans. There were no windows in the warehouse.

The warehouse work force consisted of 150 first shift employees and 75 second shift employees; 85% of the employees were women. Many of the employees were part-time, and the company had a liberal policy of allowing employees to determine their own

schedules with respect to the number of hours per day, days per week and time of day for reporting to work. Consequently, there were quite a few high school age students and women with children at home who were working part time.

In early April 1978, some employees began complaining of work associated symptoms; headache, nausea, tiredness and weakness. No clustering of cases was seen, so this was not considered unusual by management. However, when a number of employees reported to the nurse's station at the same time in mid-April complaining of similar symptoms, the company became alarmed. A private consulting firm, the state Occupational Health Division and the Federal OSHA were requested to evaluate the warehouse for toxic substances and review the ventilation requirements. These organizations were not able to determine a cause for the outbreak, but did recommend that changes be made in the ventilation system to increase the air movement within the warehouse. Initial efforts were made by management to remedy the stale air conditions in the warehouse. However, in June another illness episode occurred. Several employees reported to the nurse's station complaining of headache, nausea, faintness, puffy and irritated eyes, shortness of breath and sore throat. Affected employees were sent to the hospital for tests, and the remaining work force was sent him. Consequently, the company requested a NIOSH Health Hazard Evaluation; this was in mid-August.

Since the possibility of mass psychogenic illness had been mentioned in the Health Hazard Evaluation request, NIOSH psychologists accompanied the industrial hygiene and medical personnel on the investigation. The psychologists and I interviewed some affected employees in private to obtain detailed information regarding the illness outbreaks. Other employees were informally interviewed at their work stations.

Common symptoms included eye and upper respiratory tract irritation, unusual fatigue, lightheadedness, dizziness, headache, dry mouth and nausea. We concluded that many individual cases seemed to have been due to formaldehyde exposure, but other cases, and particularly the two episodes of mass illness, were not readily explained by this.

On that basis, we decided to administer the psychological questionnaire, and the next presentation will give a detailed explanation of this.

Because of past experience with this questionnaire, where there was what at least the medical people considered too low a response rate, NIOSH authorized a $10 reward for each person completing and returning the questionnaire. This may have been a factor in the resulting 67% response rate. However, the biasing effect of such a monetary motivator on the

questionnaire summary results is not known.

Since some of the symptoms were compatible with formaldehyde exposure, environmental sampling, both long-term, general area and 30-minute personal sampling for formaldehyde was begun. The areas and employees to be sampled were determined primarily according to the areas where most employee complaints originated.

In order to determine if any unsuspected organic compounds were present, long-term general area samples with charcoal sampling media were also taken in each of the affected areas.

The ventilation system requirements were reviewed. Since it was a simple general supply/exhaust system, no ventilation measurements were made. Conslusions and recommendations on the ventilation system would then be based on qualitative observation rather than quantitative data.

The environmental sampling did not document the presence of any organic compounds other than formaldehyde. General area and personal samples for formaldehyde ranged from 0.2 to 0.7 parts per million, all below the NIOSH exposure criteria of 1 part per million. However, irritative symptoms had been documented at exposure levels below 1 part per million, so this confirms that symptoms reported by some employees could have been due to formaldehyde exposure.

The questionnaire results(the methodologic problems of the questionnaire are acknowledged) indicated that affected employees showed a pattern of elevated job and life stress and more frequent health complaints than did non-affected employees; in this case, affected was defined as one or more symptoms. Specific factors in the pattern--and all of these specific factors are significant at least at the .05 level and many of them at the .01 level--included more frequent feelings of physical discomfort, heavy production pressure and less autonomy at the work place. Higher absenteeism, poorer over-all health and more frequent headaches, feeling of fatigue and medication use were also seen. Affected employees reported noticing odors or stale air conditions in the warehouse more frequently than did non-affected employees.

In summary, while affected employees scored higher on the hysteria and hypochondriasis scales than did non-affected employees, work environment and job stress factors were more strongly associated with reported symptoms.

One other finding particularly suggestive of mass psychogenic illness was that affected employees reported seeing other workers become ill more frequently than did non-affected workers, and this was significant at the .00001 level.

Our conclusion in this case is that while some individual employees may have been exposed to potentially irritating concentrations of formaldehyde, the two episodes of mass illness in the plant were primarily mass psychogenic illness. These were apparently triggered by the perception of odor or irritation of formaldehyde in the air and augmented by such environmental conditions as poor air circulation and elevated temperatures.

These conclusions were presented to the requester and union representative along with recommendations to improve air circulation and to prevent further employee exposure to formaldehyde. The company was subsequently contacted and reported that there were no further problems of this kind of symptoms.

Our next speaker is Clifford Moseley.

MR. MOSELEY: It's probably in the best interests of the audience that I keep this brief.

This example of MPI occurred in a firm which manufactured parts for two-stroke internal combustion engines. The areas which concerned the NIOSH investigation were the Engine Assembly Area and the Carburetor Assembly Area, two areas which were separate from each other. (See Figure 1)

It's important to note the Engine Assembly Area is surrounded by various things that I'll mention in the talk, Engine Testing, Paint Booth, Paint Storage, a Degreaser, a Deburrer and Washer, Synthetic Gas Fired Dryer, and the Carburetor Assembly Area, which is essentially off by itself.

Engine Assembly Area

The work process consists of the assembly of two-cycle engines. This process takes place on two table-high, moving conveyor lines. The room is ventilated at a rate of 2700 CFM which is 100% make-up air. The room is also heated by gas fired furnaces. There is also a gas fired oven used in the assembly process to heat-expand certain parts.

About five different chemicals (xylene, glues, varnishes, thinners and lubricants) are used on a limited quantity basis. There are approximately 40 employees in each of two shifts; almost all are women. The work is manual, repetitive and paced due to the moving conveyor line. No other department within the facility operates on two shifts. Engine Assembly operates two shifts because it is the slow step in the production of the engines. Consequently, there is production pressure in this area.

Carburetor Assembly Area

The Carburetor Assembly Area, off by itself, contains 13 assembly tables on which the carburetor is assembled. The room is ventilated with 100% make-up air and heated with gas fired heaters. Since it is important that the carburetor not be contaminated by dust and dirt, there is a slight positive pressure in the room to keep contaminants from other areas of the plant from being brought into the room by air movement.

There are approximately 80 employees in the first and only shift; almost all are women. Again, the work is manual and repetitive, but seems to require a bit more concentration than the work in the Engine Assembly.

A Brief chronology prior to the NIOSH on-site visit: for approximately one year prior to NIOSH involvement, employees of the Engine Assembly Area had been experiencing symptoms of dry throat and mouth, headache, weakness, nausea, dizziness and lightheadedness. The first episode involved one, then later the same day, several more employees. They all reported to the nurse's station complaining of fumes in their work area.

Examination by the company nurse revealed mild tachycardia; subsequently, all affected employees were sent home. The next day several more employees, some repeaters from the first day, reported similar symptoms. They attributed the symptoms to a "gassy" odor. Again, affected employees were sent home.

On both occasions, plant officials inspected the Engine Assembly Area and could find no apparent source of odors. However, environmental measurements for carbon monoxide revealed concentrations up to 25 parts per million. The prime suspected source of CO was the deburring and washing machine with a gas fired dryer which had recently been installed in an adjacent work area. There were also other sources of CO nearby: the gas fires expansion oven, gas fired room heaters, and propane powered fork-lift trucks.

One week later there were further employee complaints of gassy odors. One employee became ill and was taken to the hospital, complaining of dizziness, nausea, weakness and dry mouth. Pulmonary and neurological examinations, complete blood count, arterial blood gasses, urinalysis and electrolytes were within acceptable limits.

A state industrial hygiene consultant visited the plant in response to this episode and suggested height changes in the roof stacks for the deburring and washing machine and vapor degreaser under the theory that exhausted contaminants were being reintroduced into the work place by adjacent intake vents on the roof. These recommendations were followed.

Prior to stack height changes, solvent vapors and CO levels were measured but were below harmful levels. The Federal Occupational Safety and Health Administration also visited the plant, measured CO levels of approximately 25 parts per million, and concluded "no apparent health hazard existed".

Two weeks later, odors were again detected in the Engine Assembly Area and eventually 36 employees reported to the nurse's station complaining of dizziness, nausea and headaches. This resulted in the work area being shut down for the remainder of the day and the employees were sent home.

A private consultant hired by management visited the plant soon after this major illness episode and obtained several ambient air and breathing zone samples for CO and methyl chloroform. All values were within current standards. However, leaks in the degreaser were discovered that resulted in high concentrations of solvent in those local areas. These leaks were subsequently repaired. Other recommendations-- installing exhaust hoods, tuning potential CO sources so that CO output was as low as possible-- were implemented. Evidently these remedial actions had some affect for no unusual numbers of employees reported to the nurse's station for almost a year.

About ten months later, another illness episode occurred involving six employees. One month after that eight employees complained of fumes and reported to the nurse's station. Up to now, all affected employees had been on the first shift. However, after the episode involving eight first shift employees, 13 second shift employees reported ill on the same day. Consequently,management had the expansion oven in the Engine Assembly Area turned off and the wall separating the Engine Assembly Area from the main plant thoroughfare torn down to improve air circulation. At this time, management also contacted NIOSH and requested an immidiate on-site visit.

Three and two days, respectively, prior to the NIOSH on-site visit, and unknown to NIOSH at the time, the mysterious illness had migrated from Engine Assembly to the Carburetor Assembly Area, where two employees on each day had reported ill, complaining of burning eyes, dry throat, lightheadedness, weakness,etc.

The NIOSH on-site visit began with an opening conference involving management, the union representative and the industrial hygiene, medical and behavioral representatives of NIOSH. During the opening conference, word was received that employees in the Carburetor Assembly Area were complaining of fumes or odors in their work place.

In planning the study, we have been aware only of illnesses in the Engine Assembly Area; therefore, our investigation

strategy was designed for that area only. During the first day of environmental sampling by the hygienists, review of medical records and interview of Engine Assembly employees by medical personnel, Carburetor Assembly employees began reporting ill to the nurse's station. Since we were only set up to monitor employee exposure in Engine Assembly but based on previous factors felt there would be a repeat performance in the Carburetor Assembly the following day, it was decided to just visually review the Carburetor Assembly situation and sample this environment later. However, the medical team did interview and do limited physical examinations on those reporting to the nurse's station.

The Engine Assembly employees experienced no problems that day, nor would they the next.

The next day, personal environmental sampling for toxic substances was conducted with Carburetory Assembly employees. Later that morning, the employees began getting sick. In all, 22 Carburetor Assembly employees, over the two days, became ill and reported to the nurse's station. Detector tube measurements for phosgene, ozone, and ambient CO measurements, were all with current standards. Ambient air samples from Carburetor Assembly, Engine Assembly and a control, or non-affected area, were analyzed by a portable infrared spectrophotometer. The results in all three of these areas were identical; the presence of substances uncommon to all three areas was not indicated. Personal sampling would also indicate that levels of organic contaminants were not present in excess of current standards.

Medical interviews of the 22 Carburetor Assembly employees were performed. All but one employee was female. The predominant symptoms were dry throat, headache, dizziness, lightheadedness, weakness and nausea. Fourteen of the 22 noted smelling an "exhaust-like" odor prior to becoming ill. Physical examination of ten employees revealed no pulmonary, cardiac or neurological abnormalities. All recovered within 40 to 45 minutes. High carboxyhemoglobin levels were determined on three employees; however, this data did not correlate with environmental sampling. Breath analysis on the other employees indicated acceptable breath levels of CO less than ten parts per million.

The source of the "exhaust" odor was not identified, nor could the odor itself be detected by the investigative team.

On the second day of the investigation, the Carburetor Assembly work force demanded that the plant be closed until a cause for the illness was found. In order to discourage the work force from walking off the job and thereby adding to the confusion and anxiety, a NIOSH industrial hygienist and a physician spoke to the group. The Carburetory Assembly employees were

told that management would permit them to go home if they wished; however, the investigative team had not identified any contaminant in an amount that would exceed current NIOSH exposure criteria or that would cause a health threatening situation.

Following that, the 80 employees went back to work. However, later that day, employees noticed a strange tar-like odor and became concerned. Investigation by the NIOSH industrial hygienist determined that the source of the odor was from a highway work crew tarring the road outside and the odor was being introduced into the work place via the ventilation system. When employees were told this and were assured that the odor was not from an in-house work process, their concern decreased. No further illnesses were reported.

The confusion at the plant arising from the illness episode impeded both the distribution and return of the questionnaires. The response rate was only 43%. The major findings of the questionnaire were that affected employees were less satisfied with their jobs (i.e. quality and quantity of supervision, production pressure, job scheduling, concern regarding job layoffs), felt their jobs interfered with their home life or vice versa, and showed more frequent health complaints than nonaffected employees.

This questionnaire was distributed to both the Engine Assembly and Carburetor Assembly employees. Unfortunately, the data did not allow a comparison between these two areas.

Our conclusion in this case was that at one time there may have been sufficient concentrations of carbon monoxide and/or methyl chloroform in the Engine Assembly Area to cause headaches, nausea and lightheadedness in susceptible individuals. Subsequent illness episodes in this area were possibly a result of heightened anxiety over potential exposure to an unknown toxic substance. There had been so many prior physical changes in the Engine Assembly environment, that we could only speculate what conditions were like in the past. The contagion of symptoms to the Carburetor Assembly Area could have been a psychological response to an undiagnosed illness in the Engine Assembly Area, aggravated by the presence of outside investigators studying a group of fellow workers engaged in essentially the same work process.

This conclusion was based on the questionnaire data and the lack of any abnormal medical or environmental findings.

The next two presentations will be by Mr. Paul Pryor.

Mr. PRYOR: The remaining two case studies are somewhat unique.

The first concerns a group of airline reservation agents who received not only good pay but very excellent fringe benefits, which is unique in comparison to most of the case studies that we have been involved in. The second case involves a group of electronics workers and this is unique in the sense that this case lasted over two years, with six to eight employees becoming ill, normally the same group, and occasionally large numbers of employees being affected on and off over the two-year period.

Again, the first case concerns a group of airline reservations employees. During a three-day period in August of 1978, a large number of these employees became ill with symptoms of lightheadedness, headache, burning and swelling eyes, and fainting.

Before I describe the events which occurred during the three-day period, I will first describe the task and the environment in which the people worked.

There were approximately 300 employees working at the reservations office, of which 99% were female. The work schedule consisted of nine overlapping work shifts for a given 24-hour period. Seventy-five percent of the employees worked their eight-hour shift between 8 a.m. and 12 midnight. Each employee is allowed two 15-minute breaks and a half hour break for lunch.

The reservationists worked at a keyboard video display terminal and used a telephone operator headset to communicate with the customer. The reservation agents normally attended to 17 calls per hour, equalling approximately 124 calls in a given eight-hour shift.

The work environment, or the room in which the reservationists worked, was approximately 5,200 square feet. On the outside of the facility was the air intake system located at ground level. There were nine work rows within the room and eleven work stations on each side of the row.

The next few points I will be discussing will pinpoint some of the problems which we felt were considered to be the chronic concerns in this episode. There were a lot of women working relatively close together due to the placement of the display units. There was approximately a foot and a half to two feet of space between each of the reservationists.

Each reservation agent normally had numerous reference materials lying around the work station to assist in scheduling flights. This was also considered to be a portion of the problem. There were a number of different materials which they use to communicate with the customer and so make reservations.

When we evaluated the lighting levels, we found only 10-18% of the recommended illumination. The primary reason for this was that the women asked to have the lights turned down because of glare. Finally, the work area was illuminated by fluorescent lights and there was a central heating and cooling system which controls the temperature and humidity in the facility.

The following is a chronology of events which occurred at the Central Reservations Center during the August episode.

The episode began in August of 1978 at 10:00 a.m. when a single employee became ill. This employee was transferred to the hospital by ambulance. Over the next 45 minutes, seven other persons became ill and were taken to the hospital. At 11:00 a.m., the Airport Authority was contacted by the reservationists' manager because of a problem with the ventilation system. The Airport Authority evaluated the ventilation system and determined the controls were not operating properly. Therefore, they manually opened the cold air ducts to the work area in order to restore the temperature to normal.

By this time, five other employees reported ill. Other illnesses continued to be reported throughout the early afternoon. Union officials decided to contact OSHA and they arrived at approximately 2:00 p.m. that afternoon. The facility was evaluated at 2:25 p.m. at OSHA's request. Later that day, OSHA officials advised that the facility could be used, but additional work breaks should be granted.

Later that day, OSHA was advised that there was a jet fuel spill which had occurred earlier in the day. This occurred upwind of the outdoor air intake system to the reservations office that I pointed out earlier.

Based on these findings, it was OSHA's preliminary opinion that the direction of the wind and the location of the jet fuel spill, combined with the temperatures and humidity problems in the facility, could have accounted for the problems which developed in the reservations office.

OSHA conducted multiple tests in the facility during that first day. All of these tests were negative with the exception of traces of formaldehyde, which were less than a tenth of the standard.

Between 4:45 p.m. and 6:45 p.m., seven additional people were reported ill and taken to the hospital. In all, 15 reservation agents required hospitalization during this first episode of the first day.

The following day at 10:40, employees again began experiencing illness. OSHA officials again returned. By that time, eight additional persons were reported ill. Management requested

representatives of the Environmental Protection Agency as well as the County Health Department to come in to evaluate the facility. Their findings were basically inconclusive.

Throughout the afternoon, additional work breaks were given to employees; however, 16 additional employees reported ill during this time and were also sent to the hospital. During this first two-day period there were literally ambulances lining up to take these people away.

At 9:15 that evening, a meeting was held with union, management, OSHA and a company consultant physician. The physician's opinion was that an irritant appeared to be present in the facility; also, it was brought up at the meeting that on numerous occasions prior to the illness episode, employees were using an insect spray for what was considered to be machine mites. This was never substantiated. The active agent in this insecticide was pyrethrin compounds. It was determined that these compounds pose only a low acute oral or moderate inhalation toxicity.

During the second day, a total of 17 reservationists were affected, of which only three were repeaters. The third day, three employees became ill at 11:00 a.m. and five others later that evening.

The airline's insurance carrier sent an industrial hygienist to evaluate the illness. He arrived the next day and also conducted a thorough survey. A clinical psychologist was also retained by the company on the recommendation of the company's consultant physician. The psychologist and physician concluded that the illness outbreak placed all the employees in a stressful situation and therefore the psychologist recommended the employees participate in relaxation therapy. A number of the employees did,in fact,participate in this and this was considered a rather positive portion of resolving the immediate stress.

During September of 1978, approximately one month after the episode, NIOSH received a request for a Health Hazard Evaluation from the airline reservations union. Two investigations were performed by NIOSH over the next few months.

The first visit conducted by NIOSH was a combined environmental and medical survey; the follow-up was an environmental; medical and behavior or human factors investigation. Our concern on the second follow-up survey was primarily the lighting as well as the noise and other problems which we felt might have existed.

We also became aware that a number of different tests as well as general information was gathered by OSHA, the consultant

industrial hygienist, the EPA and the State Health Department during this episode. The following is a brief description of the facts collected by these investigators.

OSHA monitored all the weather conditions over the week, such as temperature, humidity, wind direction, etc. They also sampled the oxygen and carbon monoxide levels during the first and second day. There were no abnormal results that came out of this testing.

The company industrial hygienist consultant literally sampled everything imaginable. This included toluance, benzene, xylene, methyl ethyl ketone, methyl isobutyl ketone, trichloroethylene, pyrethrum, carbon monoxide, just to name a few, and there are many more. Again, all of these were insignificant except for the trace of formaldehyde that I mentioned earlier.

NIOSH reviewed all this information and attempted to recreate the events which occurred during this episode. Our medical survey consisted of a questionnaire which elicited general information concerning the alleged illness, types of medical attention sought, and personal habits during the August episode.

Three hundred and twenty-three questionnaires were completed and returned. Of the 323, 59% indicated some symptoms during the three-day episode. It was determined that 61 reservation agents required transportation to the hospital; all of the symptoms except for faintness were noted by the majority of these agents. Lightheadedness, headache and eye irritation were the most frequent complaints mentioned.

The questionnaire determined that a large number of persons developed at least one symptom; however, it was determined that these symptoms were not indicative of any specific disease process but they may be consistent with a number of diseases including an acute toxic reaction to hydrocarbons, carbon monoxide, organophosphate insecticides, or hyperventilation syndrome.

These illnesses can be differentiated by physical examination and a laboratory test, but only if the tests are performed during or relatively soon after the acute illness. Unfortunately, NIOSH was not called into the case until a month after the episode. We were, however, successful in obtaining medical records on 13 reservation agents for whom laboratory tests were performed. A multitude of tests were performed by the various physicians on these people, such as chemistry screens, blood counts, chest X-rays, and thyroid function tests. There were no noteworthy laboratory abnormalities along these tests.

Two persons had carboxyhemoglobin tests performed the first

day. Both were smokers and their COH_6 levels were considered normal for smokers.

NIOSH investigators attempted to determine any chronological relationship between events which might have occurred at the airport during the episode as well as those prior to the episode. The information we collected was impressive in terms of potential cause-effect relationships between a number of different factors and the distribution of illness. The following is a summary of our findings.

First, we concur with OSHA that the primary environmental concern in this episode was the high temperatures and humidity conditions both outside, as well as inside the facility during this period. This would, in fact, produce a stress-full work environment. Also, the Air Pollution Index during this period exceeded a hundred, which is also a very unfavorable situation to work in, especially if the air conditioning unit is not operating.

Other factors which NIOSH considered very relevant were the physical work conditions as noted earlier, such as poor lighting. We measured the lighting in this area and we found that over 75 percent of the room had only 5 to 20 footcandles. This is equivalent to the lighting found in a hallway.

It was also determined that there was a lack of adequate work space for the reservationists and high noise levels, that is, the general background averaged between 70 and 85 DBA, which is very loud for concentrating purposes. Thus, we felt all of these contributed to the stress experienced by the reservation-ists.

In summary, it was our feeling that the over-all synergistic effect of all of these variables coming into play in a short period of time were, in our opinion, responsible for creating the stressful work environment at the airlines reservations. This concludes the first talk.

Electronics Plant

The last case of MPI I will be discussing is one which I'll refer to as being chronic or of long-term duration in regards to its impact on both management and the employees.[24] This change occurred in an industry in which episodes of MPI are becoming more and more pronounced, i.e., the Electronics Industry. This company has a work population of approximately 500 employees and the company produces primarily small electrical parts. Among the different activities performed at the company are electroplating, soldering, brazing, degreasing, and paint spraying.

The problem first began in October of 1976 when a 5 gallon drum of Toluene Diisocyanate (TDI) spilled in the electronic assembly department. Attempts to absorb it with newspapers and absorbent compounds began immediately. After ten minutes, the vapors became intolerable to the employees in the electronic assembly area, and within another ten minutes this area, as well as the entire plant, was evacuated. Two local fire departments were called in to clean up the spill and industrial fans were used to expel the fumes from the plant. An ambulance had been summoned to the plant, but no one required hospitalization.

From October 20 to December 28, 1976, a total of forty employees complained to the occupational health nurse at the company with complaints of dry mouth, lips, tongue, and general irritation to the face and upper respiratory tract.

During January 1977, the Electronics Company retained an industrial hygiene and environmental control engineering group to assist in determining the unknown source of odors and fumes in the plant. It was determined during their initial review that a practice of air drying photo plates in one of the adjacent departments was also a cause of odor and therefore must be stopped. These plates were treated with Freon as a final stage in the developing process and once this stage was complete,the operator would place these plates in front of a make-up ventilation duct to dry. Numerous complaints were registered with the company nurse regarding odors and fumes present in this department and the adjacent department prior to this finding.

During the later part of January, the environmental consulting group, retained by the company, began their environmental health investigations in the plant. Also, State OSHA, operating under an approval state plan, i.e., a compliance inspection under the federal audit program, conducted a safety inspection at the plant. No health complaints were registered on any of the days that the Federal, State, or consulting group was in the plant.

From February 1977 through July 1978, a series of investigations by the Federal, State and Area OSHA offices, EPA, State Health Department, and the Consulting Industrial Hygiene Group was performed at the Plant.

On March 5, 1978, 6 employees complained to the company nurse regarding various health problems from unknown sources. These symptoms included sensory abnormalities (e.g., numbness, tingling feeling, and swelling which affected the lips, shakiness, nervousness, "light-headedness" or"dizziness", headache, dry mouth or throat, nausea, "passing out", blurred vision, and "hot" or "flushed" face). It was interesting that not one of these complaints were given while any of the

investigative groups were in the plant.

One morning in July 1978, numerous relay-floor employees complained of irritating odors which apparently came from a burning transformer. As some employees began experiencing symptoms, all the persons in this area moved outside. Also, during this time, a boiler had malfunctioned, thus producing abnormal stack emissions. During this episode it was decided by OSHA, the State Health Department, local police, and management to close the plant for the remainder of the day. Thirty-one employees sought medical attention for symptoms associated with this episode. The most common symptoms, based on data from 28 persons, were nausea, headache, "light-headedness", "burning" throat, and weakness. A variety of other symptoms, including diarrhea, vomiting, abdominal cramps, loss of appetite, drowsiness, numbness or tingling of the mouth or tongue, throat "tightness", shakiness, "cold", "tetany", "hysteria", nosebleed, "burning" eyes, and "acute bronchospasm", were also reported, but no more than 1 complaint was reported by more than two persons. Of the 25 seen at one of two emergency rooms, all were discharged without a specific common diagnosis. Among the six employees who sought medical attention elsewhere, one had a chest X-ray, and one pregnant woman had two laboratory "tests"; all were reportedly normal. For those employees who required medical attention, recovery time ranged from a few hours to a few days.

During the first part of August 1978, the local Area OSHA office began sampling for what was suspected as organotin and lead exposure from the soldering operations. Their sampling and analytical technique was unsuccessful, therefore, they had to abate this pursuit. A few days after the OSHA's investigation, another episode of mass illness occurred which involved 13 employees in the electronics assembly area. Their symptoms included headache, bitter taste, "burning" nose, "light-headedness", shakiness, and nausea.

On September 1, 1978 - thirty employees were sent home suffering from alleged nausea, headache, as well as burning eyes, nose and throat irritation from an unknown fume and odor problem. Again, OSHA and the EPA were brought in to investigate this episode and they hypothesized that the origin of fumes may be from a plant located next to the Electronics Company.

NIOSH - HETAB REQUESTED

On September 27, 1978, the Hazard Evaluation Technical Assistance Branch received a request for a Health Hazard Evaluation from an authorized representative of the Electronics Company. The request stated that since 1976, the company has experienced repeated incidents where employees became ill.

During October 1978 NIOSH personnel conducted a combined environmental/medical evaluation at the company. Background information was obtained from the Area OSHA office prior to meeting with union and management officials from the company. Upon completion of this meeting, NIOSH, union, and management officials toured the plant. It was determined that the following were the chemicals and environmental tests that were performed by OSHA and the consulting groups that had been in the plant: Methyl chloroform, Tuluene, Xylene, Butyl acetate, total Hydrocarbons, Freon-113, Organotins, leads, general and local ventilation measurement, stack emission tests, and evaluation of work habits.

Based on the information collected by OSHA, EPA, and the Evnironmental Consultants,as well as our own findings, it appeared to us that the variables to be considered as the primary contributing factors involved in the problems at the Electronics Company were:

1. Isolated incidents - triggering events

    a) Oct. 1, 1976 (TDI exposure)
    b) July 14, 1978 (transformer failure)
    c) Other mass illness episodes (causes - unknown odors, etc)

2. "Possible chemical contamination" from soldering or brazing operation.

    a) Tin
    b) Lead

3. Improper local exhaust ventilation, flow rates and/or hood designs.

    a) Spray paint operations were not being effectively exhausted.
    b) Electronic assembly, e.g., soldering and brazing operations which had exhaust systems that were not operating properly.

4. Improper exhaust stack height on the roof.

5. Wind direction, i.e., potential odors being brought into the plant from outside sources when the wind direction was right.

6. Poor work practices

    a) Use of fans at worksite.
    b) Failure to cover flux and solvents after use.
    c) Lack of protective gloves when cleaning parts.
    d) Freon TF (55 gallon drum) not capped.

e) Mixing of chemicals or cleaning of parts not being performed under an exhaust hood.
f) Improper use of existing exhaust hoods and/or maintenance, e.g., brazing, soldering, and paint spray booth.

Based on the evaluation of each of the factors just mentioned, the following recommendations were made:

1) Each of the improper work practices described earlier should be resolved, i.e., with the various department supervisors and employees. Emphasis should be placed on educating these individuals as to the problems which can occur if improper work practices are allowed to continue;

2) The proper duct size, hood design and flow rates for each of the soldering and brazing exhaust systems, as well as those systems proposed are essential for the workers' health and safety;

3) The maintenance performed on the paint spray booth, e.g., filters, flow rates, etc., should be increased. Also, the length of time between the completion of the painting operation and turning off the exhaust system there should be extended. This procedure should reduce the likelihood of odors emanating into the Electronics Assembly Area;

4) A partition closest to the paint spray booth, e.g., that partition which separates the Paint Spray Department from the Electronic Assembly Area should be extended to the ceiling. Therefore, the possibility of drawing odors from the spray booth operation and into the assembly area should be eliminated. Also, a door should be installed at the entrance way leading into the Paint Spray Area. This should also reduce the potential of odors emanating from this area and into the Assembly Department;

5) Develop strong communication between management and union officials, as well as management/union and the employees at the plant.

CONCLUSIONS AND RECOMMENDATIONS

In the genesis of MPI there is almost invariably a "trigger", such as the introduction into the work environment of substances, or processes with which the worker in unfamiliar, and/or the accumulation of odors or fume in concentrations to which the worker is not accustomed, albeit at non-toxic levels. Thus, stress and anxiety will only be allayed when fear, which

is based on ignorance of the nature of the chemicals and/or agents to which they are exposed, is removed from the minds of the workers. This entails the cooperation of management and labor in worker education.

In conclusion, due to the lack of adequate information in the area of Mass Psychogenic Illness, it seems impossible to successfully foresee and prevent an outbreak of psychogenic illness at this time. However, we have made several non-quantitative observations concerning the general environment surrounding cases where, in our opinion, MPI was considered as a primary factor in (1) either extending the length of the episode, or (2) increasing the sum of the reaction to the affected employees. The following are a few of the more common problems noted from these investigations.

1. When governmental or private environmental and occupational health groups become involved in these investigations, they are normally unaware or do not consider the possibility that MPI is a factor in the problem. Thus, when these groups are faced with outbreaks where employees are affected by unknown contaminants, they will usually pursue their investigation in the same manner that they would any occupational health problem, i.e., searching for chemical, physical, or biological exposures. In MPI cases, however, this procedure is only partially productive in the sense that one must not limit the investigation to only these concerns, but must also go beyond these and consider the psychological, sociological, and/or ergonomic factors which are common to MPI episodes. Another situation which occasionally develops when these groups become involved is what can be referred to as the "Sterile Dress Approach". This is when the investigators arrive at the company wearing jump suits, hard hats, and respirators. Employees see these investigators dressed in this attire and their fears naturally are heightened. In all those cases of MPI that we have evaluated, there has been no cause to suspect that there was a need to wear any unnecessary personal protective equipment other than that normally required in the work process. However, this does not mean that personal protective equipment should never be used, but rather it should be fully understood whether this equipment is actually necessary prior to arriving at the company.

NIOSH, not being immune to these human frailties, has in the past reacted in similar ways; however, over the last two years, we at HETAB have made an effort to resolve these problems when conducting investigations where MPI is suspected.

2. Another common situation which we often find is mishandling by managment in regard to the attention given to the problem. This commonly manifests itself as either overconcern or underconcern. Also, frequently there are apparent labor-managment or intra-union difficulties, and thus labor or management

representatives take a position from which they are unable to withdraw. There the issue of industrial health literally becomes of topical interest with specific emphasis on a "Them Versus Us Attitude".

3. In many of these episodes, emergency vehicles are requested. These vehicles normally arrive with lights flashing and sirens blaring, which more often than not intensifies the apprehension of the employees.

4. Another major problem is trying to determine whether an individual is or has been exposed to chemicals in the work place. In some instances, we have attempted to do personal sampling on employees only to be confronted with statements like "I do not wish to wear the sampler because it will draw the chemical into my breathing area". An additional problem, in respect to sampling employees, is occasionally this will precipitate adverse reactions in these employees or employees around them.

5. Most episodes of MPI are very dramatic and often attract the attention of local media. The following headlines are common: "Factory Ills Still A Mystery", "Mystery Unsolved in Poison Factory", and "Mystery Fumes Investigated by Feds at Plant". Employees often read these articles and express concern about the lack of specific information regarding their health and safety while working at their job. Thus, the news media often attempts, and succeeds, in sensationalizing even ordinary comments, and hypotheses or general comments often turn out to be alarming statements in the morning's newspaper.

6. In past episodes where environmental sampling has been performed, there have been occasions where instruments or sampling techniques have failed or backfired. These types of problems bias the affected work force and therefore, adversely affect the investigator's credibility.

7. Finally, a common confusion in MPI cases is the misunderstanding between exposures to toxic gases or fumes versus odors which are only offensive i.e., when employees are interviewed they often refer to toxic odors, gases, and fumes.

RECOMMENDATIONS

The following are those steps and recommendations which we feel should be considered in order to reduce and/or eliminate problems when MPI is suspected.

Note: These steps and recommendations may vary according to the role the professional is in, e.g., governmental versus private industry.

I. PREPARATION PRIOR TO INVESTIGATION

A. As appropriate request or assign;

1. Medical
2. Industrial Hygiene
3. Behavioral Scientist

B. Contact appropriate personnel at company (to ascertain)

1. Onset and duration of exposure
2. Symptoms and complaints
3. Size of affected versus total work force
4. Type of work being performed (in all areas)
5. Type of ventilation in use (exhaust and make-up)
6. Plant layout (e.g., floor plans, ventilation systems, sewer systems, etc.)
7. Weather conditions (e.g., wind directions, temperatures, and humidity)
8. Lighting - task versus background
9. Noise - task versus background
10. Type of personal protection required

C. Other concerns

1. Time of year
2. Unusual changes or events

D. Advise management to

1. Remove potential contaminants if known.
2. Assign a laison officer to interact with all the parties involved.
3. Maintain calm deliberation.
4. Isolate affected employees away from nonaffecteds
5. If emergency personnel are required (ambulances, police, etc.) ask them to refrain from using sirens and lights.
6. If media becomes involved, release only prepared statements. If you don't want to be quoted, don't say it!
7. Request a closing meeting to review the situation with appropriate personnel (company, union and outside investigators).
8. Have worker representatives notify employees concerning your proposed arrival and intended investigation.

E. If applicable, contact other investigators - Federal, State, Local, or private concerns (Request)

1. Their findings
2. Observations

3. Impressions
4. Results
5. A meeting prior to your survey

F. Select equipment to evaluate environment and be familiar with

1. Use of equipment
2. Sampling protocol
3. Workability of equipment

II. FIELD INVESTIGATION

A. Meet with other investigators involved in episodes away from company

B. On-site investigation

1. Meet with appropriate company personnel (management, union, etc.).
   a) Reiterate information gathered to date.
   b) Review any information requested (e.g., floor plans, ventilation, etc).
   c) Review medical and environmental data.
   d) Describe medical concerns - symptoms, toxicology, distribution of exposed employees, etc.
   e) Arrange any anticipated meetings with:
      1. Worker representatives
      2. Management
      3. Other, e.g., maintenance, plumbing and ventilation experts, etc.
2. Perform a walk-through survey to familiarize yourself with the plant and areas of concern (with management and union personnel in attendance).

   NOTE: Introduce yourself to other managers, supervisors, and concerned employees; if necessary, during your investigation. Let them feel like they're part of resolving the problem.
3. Begin medical and environmental survey.
4. Request closing conference with management, union and any other concerned groups.
5. Emphasize open communications between employees and management, especially concerning findings and any proposed changes which may improve the situation.

III. INTERACTION WITH MEDIA

A. Conduct interviews outside the company if possible.

B. Prepare statements
C. Notify concerned parties regarding statements

IV. NOTIFY ALL THOSE CONCERNED GROUPS WHEN RESULTS ARE AVAILABLE

This concludes our presentation,and on behalf of myself and the other members on the panel,I would like to thank you for listening. At this time,we will entertain any questions which you might have regarding this subject.

Dr. Elesh is going to summarize some of our findings and our recommendations.

Based on our review of previous mass psychogenic illness cases, two dilemmas seem to prevail during many of these episodes as well as in others,reported in the literature. Therefore, the way in which these cases are responded to and handled is very important.

First is the type of response that takes place during these episodes by many of the public and private environmental and occupational health groups who have been brought in to investigate these cases. Often where the problem may be obvious and easily handled by one or two people, it's not uncommon to see entire teams of environmental, medical and psychological personnel being sent to manage an investigation worthy of a major environmental disaster.

Over the last two years, HETAB, (Hazard Evaluation and Technical Assistance Branch), has attempted to reduce these problems by being very selective as to what expertise is given to these cases and during what phase of the investigation the different professionals become involved. Usually one member from the following groups will be considered; 1) physician or industrial hygienist: 2) physician, industrial hygienist and behavioral scientist; 3) physician, industrial hygienist, with a behavioral scientist being brought in later.

The second dilemma which seemed to develop in many of these cases is that despite the thoroughness that has been given to many of these studies, it is not uncommon to find either management or labor or even both being dissatisfied when NIOSH attributes a majority of the problem to mass psychogenic illness. Employees may feel offended or embarrassed and think the investigators are, at worst, "bought off", or at best, are incompetent or unsympathetic. Thus, it appears that the employees would prefer that an environmental contaminant be found so that a correctable environmental explanation can be provided for their complaints.

Management, again, may be unduly reassured and believe that in the absence of a causative environmental contaminant, they have no contributory role in the occurrence of the outbreak.

In summary, it appears that a Catch-22 type of situation can be expected when one becomes involved in these cases. Therefore, it is our feeling that it is not only important to be selective in regards to the number and type of experts who respond to and handle these cases, but also the way in which these cases are handled. That is, the sensitivity that investigators give when interacting with management and union personnel, and how they present data.

In conslusion, then, in the genesis of mass psychogenic illness, there is almost invariably a trigger,such as the introduction into the work environment of substances or processes with which the worker is unfamiliar and/or the accumulation of odors or fumes in concentrations to which the worker is not accustomed, albeit at non-toxic levels.

Based on a review of all the outbreaks of MPI with which NIOSH has been concerned, as well as published and unpublished data on similar incidents in which other investigators have been involved, the only common factor appears to be the diversity of the trigger, the work environments, the work process, and the work forces; hence, prediction is almost impossible.

Stress, with accompanying anxiety, is a fact of life to which we all react according to our nature and upbringing. That anxiety is contagious cannot be doubted. Almost invariably as has been illustrated in our description of the four outbreaks, the trigger has been a change in work processes, possibly superimposed on some previous change, ventilation modifications, the introduction of some new chemical into the environment, or the development of management-worker disaffection, etc.

If any of the above are contemplated or anticipated, management would be well advised to thoroughly and simply explain to the workers and their representatives the reason for any changes, and how this may affect the quality of the working environment and tasks of the work force.

Therefore, in conclusion, due to the lack of adequate information in the area of mass psychogenic illness, it seems impossible to successfully foresee and prevent an outbreak of psychogenic illness at this time. However, we've made several non-quantitative observations concerning the general environment surrounding these cases where in our opinion psychological factors were considered to have extended the length of the episode or increased the number of affected employees.

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BEHAVIORAL AND MOTIVATIONAL FACTORS

PSYCHOLOGICAL, PSYCHOSOCIAL INFORMATION

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I would like to describe the protocol followed by NIOSH behavioral scientists in investigating cases of apparent psychogenic illness and briefly summarize our results to date.

The behavioral Factors Evaluation consists of two aspects; First, semi-structured interviews with some of the affected employees; and 2, the distribution of a lengthy questionnaire to workers at the facility.

The questionnaire was designed to be broad in scope and surveys sociodemographic and epidemiological factors in addition to the following specific factors. First, job stressors, and here we looked at both the physical stressors such as physical discomfort at the work place, temperature variations, as well as things like work load and work pace. Psychological stressors that we measured included role conflicts, boredom, and relations with supervisors, to name several. These scales have been used in previous NIOSH studies of job stress. We also used the work environment scale which examines the social structure at the work place and is predictive of worker dissatisfaction; some of the dimensions measured with these include worker commitment to the job, peer cohesion, work pressure, management support, and physical comfort.

The second major area covered was medical health status, and we used a checklist for previous symptoms and illnesses, for medication use, and for a global self-rating of overall health.

The third area that we looked at was personality factors. For this we used three standardized tests: The Eysenck Personality Inventory, and Internal-External Scale, and three-subscales of the Minnesota Multiphasic Personality Inventory.

The last area surveyed was sociometric factors and here we were looking at communication patterns in the work place and how well the individuals knew those who became ill during the outbreaks.

The results which I will discuss today were derived from five work establishments and represented responses from over 200 affected workers and 200 non-affected workers. The work establishments were each engaged in a different work process,such as assembly of electric switches, shoe manufacturing,while others involved simple assembly-type tasks.

Despite the diverse nature of the work groups, the symptomology reported by the affected workers in each case was remarkably similar. The most common symptoms were: headache, lightheadedness, dry mouth, dizziness and sleepiness. The number of symptoms reported by employees varied considerably between one and 19 symptoms, but the average number reported was about three symptoms.

The number of symptoms reported was correlated with scores on the psychodiagnostic tests to isolate those factors which related to affected status. Those employees who became affected during the outbreaks did not differ significantly from unaffected employees in terms of their age,race, marital status, level of education, nor the seniority status.

In each investigation, we found higher levels of job stress in affected employees relative to the non-affected employees. In one case, stress emanated from repetitiousness on the job, a fast work pace, high production pressure and feelings of job insecurity. In another case, sharp increases in work load, unwanted overtime and financial pressure to retain the job generated undue stress. In still another case, feelings of boredom, high work pressure and little recognition from supervisors loomed as significant stressors.

There were several indicators of stress and strain which did appear with some regularity in our investigations. These included, first, physical discomfort at the work place such as being bothered by temperature variations, noise levels, and unpleasant odors; second, feelings of increased work pressure which often resulted in much overtime work; third, poor relations with supervisors and feelings of decreased support from management; number four, feelings of financial pressure to continue to work in order to make ends meet; and five, generally, poor health status or poor self-reported health status in the affected employees relative to the non-affected employees.

Thus, the type and level of job stress varied somewhat between cases, but in each case higher stress was found in the affected group. Levels of perceived job stress and strain were the best predictors of affected status of all of our response measures.

In order to detect differences in specific pre-illness health complaints, respondents were asked to indicate how often during an average month they experienced symptoms or illnesses and how often they used prescription and non-prescription medication during an average week. The findings for the health status factors were not consistent across cases. In one case, the affected workers reported more frequent feelings of fatigue and exhaustion, nervousness, and tension, faintness, and more frequent colds than non-affected employees. In other cases, the frequency of specific symptoms did not differentiate groups. Medication use revealed similar inconsistent results.

Thus, aside from the affecteds rating their over-all physical health as poor, there did not seem to be a relationship between pre-illness health status and the probability of experiencing symptoms in these five cases.

We were very much interested in examining the relationship between specific personality traits and affected status. A major question of interest was, is there a certain personality type which is particularly susceptible to mass psychogenic illness?

To answer this question, we selected personality tests which measure such traits as depression, hypochondriases,hysteria, neuroticism, extroversion and stability. For all tests save one sub-scale of one test, there was no consistent relationship between personality traits and affected status. There is no personality type which predisposes employees to mass psychogenic illness.

The one exception was the hysteria subscale of the Minnesota Multiphasic Personality Inventory. Here, affected workers scored higher than non-affected workers in most of our investigations. However, in light of the fear, confusion and uncertainty which most employees experience during the illness outbreaks, this result is suspect. Such attitudes would be expected to bias the results on this particular sub-scale. One can safely conclude that personality factors, at least those measured here, were not good predictors of affected status.

Our final measure was communication patterns among workers within each facility. In two cases reported here, and in two more recent investigations, we found that the affected workers more frequently witnessed others becoming ill during the outbreaks than did non-affected workers. Interviews with affected workers confirmed the fact that knowledge of or the witnessing of others becoming ill served to increase the probability of more workers becoming ill and acted to promote the spread of symptoms throughout the work place.

Sociometric patterns need to be investigated in much more detail in such cases in order to better documents the epidemic-like characteristics of these illnesses. These finds, coupled with previous reports, indicate that mass psychogenic illness affects women in a predominantly female work force who are experiencing physical and psychological stress above normal levels. The actual outbreak of illness is usually triggered by a physical stimulus, such as a strange odor which is perceived by one or more workers and is believed to be the cause of their discomfort. The specific symptoms may vary somewhat between cases, but typically consist of subjective complaints such as headache, lightheadedness, dry mouth and dizziness.

Two issues need to be addressed at this time. First, the sex difference in affected status. Why would females become affected much more so than males in these investigations? As mentioned earlier, females were over-represented in the target population

and this may explain in part the observed differences.

However, another hypothesis is equally reasonable. The working woman, saddled with an outdated domestic role, may frequently find herself trapped between the conflicting demands at home and on the job. To the extent that work-related conditions such as unwanted overtime or fatigue interfere with perceived domestic responsibilities and vice versa, the working women may experience considerable guilt and frustration. The conflicting demands of holding down essentially two jobs can generate a significant amount of distress, resulting in altered health status.

A second issue concerns the interpretation of these results. To suggest that the illnesses had a strong psychosomatic component in no way implies that the workers are abnormal or that the symptoms are not serious. In contrast, the results of five investigations and more recent ones have indicated that the work force is essentially normal and the affected workers are seriously physically ill.

The illness may be compared with a more familiar condition, a stomach ulcer. These are very serious disorders and are largely caused by stress, but it is not implied that the victims are crazy.

It is important to remember that describing an illness as stress-induced does not settle the issue. In cases of true toxic exposure, isolating the cause is only the first step.

Unfortunately, no single remedy to alleviate mass psychogenic illness outbreaks has been tested sufficiently to warrant recommendation. However, steps which reduce stress levels, such as making the work place more comfortable or improving management/labor relationships, would seem to be meaningful alternatives. Since the specific stressors vary between incidents, remedial actions may also have to be incident-specific to be maximally effective.

ISSUES IN THE IMPLEMENTATION OF REMEDIAL INTERVENTIONS IN PSYCHOGENIC ILLNESS

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Issues concerning the implementation and evaluation of remedial interventions for mass industrial psychogenic illness are discussed. The basic argument is that any remedial action focused on the outbreak itself will be difficult to implement because of both the organizational characteristics of industrial firms, and the inevitable complexity and uncertainty arousing nature of a remedial intervention. As a result, it would be difficult, if not impossible, to evaluate a remedial program.

Additional analysis addressed the larger issue of whether industrial mass psychogenic illness was a meaningful focus of inquiry, and whether the individual within an organization was the appropriate unit of analysis. An argument was made that further research in this area needs to be integrated with research on employee satisfaction and job design.

The instance, or rather reporting, of mass psychogenic illness, has waxed and waned for many years in the psychiatric and psychological literatures. Sirois (1974) attempted a detailed typological breakdown of seventy reports dating to the 1870's. More recent and pertinent to this analysis, Colligan and Murphy (1979) reviewed 16 reports from instances in organizational settings, including schools, hospitals, and industry in an attempt to identify key explanatory variables.

Notably missing from these and other references has been a cogent approach of how one might intervene, either preventively or remedially, relative to these outbreaks. Virtually all cases of mass psychogenic illness in organizational settings have subsided without an explicit "treatment" being applied.

This paper will not present an immediate remedy to the problem of industrial psychogenic illness. However, it will discuss problems relating to the possible deployment and evaluation of remedial action. In this analysis, two cross-cutting concepts will guide the discussion: (1) that any remedy will have to be implemented in an organizational/bureaucratic setting, and the nature of such social systems is to resist the imposition of new practices in the absence of clear incentives for their use; and, (2) that depending upon how one views the etiology, and unit-of analysis involved in mass psychogenic illness, the focus of both scholarly inquiry and remedial interventions could conceivably undergo a radical shift. The crucial issue here is whether the problem of mass psychogenic illness is to be construed as individual aberration, albeit facilitated by informal social networks, or as a rather bizarre manifestation of more persistent and pervasive organizational dysfunction.

As an emerging concern of both private and public stakeholders in the industrial setting, the treatment of mass psychogenic illness presents interesting intellectual and logistical

problems. However, at the outset one crucial issue needs to be addressed. Any remedial intervention will have to be adopted, and implemented, by industrial organizations, perhaps in collaboration with government. That adopting-implementing process will be an exercise in social change in the industrial setting. It will mean that something euphemistically known as an "innovation" will need to become incorporated into ongoing organizational practice. One ought to get a realistic awareness of the general difficulty of that task. Consider the following classic quote of forewarning:

> "Although every significant social change begins as an innovation, the appearance of innovators and innovations does not directly and automatically result in changes in the social system. No innovation is ever more than the cornerstone upon which, given proper social acceptance, a new element may be erected" (La Piere, 1965).

It should also be noted that government has been in the past--and is currently--engaged in efforts to promote technological innovation in different sectors of the economy. The results of these efforts have been, at best, mixed. That this pessimism is bounded in empirical reality can be seen from the early NASA experience in attempting to transfer space program technology to the private sector:

> Of the 21,000 companies which NASA thought could use the inventions, 30 companies or 0.15% had adopted or seemed to have a good prognosis for adoption. Of the 3,100 companies from the preceding 21,000 which had agreed with NASA that the invention did sound relevant, 1% were in this adopted or likely to adopt group of 30, and of those 550 companies which on further investigation with NASA did not drop out of contact or say "No," only 5.5% ended in the final favorable group of 30 who had adopted or seemed to have a good prognosis for adoption. (Wright, 1966)

While the NASA experience involved "hard" technology, data perhaps more relevant to the issues at hand can be found in the innovation process literature involving public sector organizations. That this is a legitimately analogous situation can be argued if one realized that any remedial intervention is likely to be a "software" innovation, and there are likely to be few, if any, market incentives for adoption. Therefore, it is instructive to report that in a recent national dissemination of innovation research (Tornatzky, Fergus, Avellar, and Fairweather, 1979), a rather dismal experience was encountered with a group of mental hospitals. Beginning with a sample of 108 hospitals, 81 institutions agreed to a no-cost workshop presentation about a demonstrably effective treatment innovation. Of these, 31 made an ostensible commit-

ment to implement the program. However, of that group only 13% (3.7% of the sample of the initial sample of 108) actually implemented a replicate of the innovation, after over two years of consultation, training, and technical assistance. Without elaborating this litany of woe, the point is very simple--to implement an innovation in a complex organization is an extremely difficult undertaking. To blithely assume that any remedial intervention will be easily deployed is to ignore certain realities of organizational functioning.

Most industrial organizations can be described in most particulars as bureaucratic systems. The normative and theoretical rationale for their structure and internal process borrows heavily from Max Weber (1947), classical scientific-management theory (Massie, 1965), and business school ideology. Such organizations can be described as social systems designed to rationalize the accomplishment of tasks. As such, they have various features and operate under certain premises, in accordance with that metagoal.

Classical bureacratic epistemology, such as exists, rests on the belief that the world is a knowable, rationalizable place, and that knowledge requisite for accomplishing tasks is available and reducible. The intent of organizational systems is, correspondingly, to focus knowledge and expertise on task accomplishment. One way to accomplish this is to specialize, and to assign each worker only a small component of the total task. The assumption is that the average worker can gain mastery of repetitive skill easily and therefore, considered in the aggregate, there will be a net increase in the organization's small case knowledge or skill level. Given specialization, the need for coordination becomes more crucial. Explicit rules and standardized procedures help accomplish this, such as in the traditional production line setting. Additionally, a supervisory hierarchy is employed to clarify rule application and to maximize cooperation between specialists. In order to prevent subcultural values, or personal idiosyncracies, from fostering potential conflict, interpersonal interaction is minimized and formal communication employed whenever possible.

This classical "machine model" (Katz & Kahn, 1966) is assumed by many to be the most efficient way to organize work in the industrial setting. A secondary assumption is that its efficiency will reward its members in material terms, which is ostensibly the principal motivating force behind employees' behavior. Although there are many obvious deviations in a real-life operational sense from the classical bureaucratic-rational model, its influence is pervasive--but not to the exclusion of other views.

In the past several years, some of the more interesting rethinking of organizational concepts has come under the rubric

of contingency theory. The central premise is straightforward: the best way to structure an organization depends on the nature of the task being accomplished.

Most contingency theorists attempt to enumerate task features, or contextual vaiables, into two categories--those compatible with highly structured, bureaucratic organizations, and those more congruous with open, participative, "organic" (Burns & Stalker, 1961) organizations. The guiding assumption is that it is equally inappropriate to impose a structured organization form on certain tasks, as it is to employ a participative, non-bureaucratic organization, on other types of tasks.

For example, Litwak (1961) has identified those tasks which are repetitive, have a knowledge base, are not value-laden, and are uniform, as being appropriate for highly structured organizations. Lawrence and Lorsch (1967) mention stability of market and technology as being congruous with a bureaucratic solution. Perrow (1967) mentions task situations and technologies, in which there are few exceptions to standard procedures, and where an orderly search process is operative when exceptions occur. Other writers (Thompson, 1967; Galbraith, 1973) have made similar analyses--if a task if in fact rationalizable and repetitive, then a structured, "bureaucratic", rationalized organization can handle it best.

In contrast, if a task of "non-uniform" (Litwak, 1961) or nonrepetitive, idiosyncratic,and lacking in a knowledge base, a more open, participative organization form is seen as appropriate. If the environment, market, or technology is "turbulent", and changing rapidly (Lawrence & Lorsch, 1967) a similar conclusion is drawn. Perrow (1967) discusses task situations in which there are many exceptions, and an unanalyzable search process prevails. The assumption underlying all of these conceptual frameworks is that some task situations call for maximum organizational flexibility and fluidity; to impose inappropriate structure, rules, procedures, and formality is dysfunctional.

To capture the thrust of these concepts it might be useful to employ the notion of uncertainty. If the amount of uncertainty introduced into a system is increased, more flexible, open, participative organizational forms are demanded. Why this might be so from a social-psychological perspective becomes clearer if we ponder the implications of social comparison theory (Festinger, 1954), or the relative "communication-richness" of various forms of social interaction (e.g., Chapanis, 1971) and their comparative utility in transmitting complex information.

Uncertainty and Innovation: Is a Remedy Possible? The implication of this statement for the problem of industrial mass psychogenic illness will shortly become apparent. If

any intervention is to be introduced to either prevent, or heal the problem of psychogenic illness, it will constitute an innovation in practices and procedures for that organization. The fact is that most organizations are not structured to cope with the process of innovations, but rather to operate in a relatively stable state. In turn, the acceptability of an organizational innovation will be related to the extent to which its features intrude, or arouse uncertainties.

In fact there is a rich literature on the difficulty with which innovations are introduced into organizations, some of which has been alluded to. An important focus of this literature is on the aspects of innovations that are predictive of the ease in which adoption and implementation might occur.

Rogers & Shoemaker (1971), and Zaltman, Duncan, and Holbek (1973), list a number of such factors, some of which seem to have an obvious relevance to the dissemination-adoption-implementation of a remedial program for mass psychogenic illness. For example, Zaltman, Duncan and Holbek (1973) list twenty "attributes of innovations" that are related to innovation processes. Some of these predictors that seem especially relevant to this analysis include the following: the clarity of results of an innovation; its compatibility with local norms and practices; complexity; its impact on interpersonal relationships; its communicability; and, or particular relevance, the relationships between financial and social cost, and perceived returns to investment. What is a firm to gain, economically-speaking, by deploying a remedial intervention? Another important attribute identified by Zaltman et al (1973) is the demonstrability of an innovation. Can the potential benefits be easily demonstrated to potential users? This would seem to be quite problemmatic in the case of mass psychogenic illness, an extremely rare, non-repetitive event. This is also related to Rogers and Shoemaker's (1971) concept of trialability. Can an innovation be experimented with, or adopted on a trial basis, prior to complete incorporation? Once again, in the case of a remedy for mass psychogenic illness, what a "trial" would constitute remains obscure.

In view of the above discussion, the question needs to be asked as to whether anything approximating a "remedy" can be deployed to deal with the outbreak of mass psychogenic illness. If the focus of intervention is on the epidemic outbreak itself, a remedial program is likely to be a complex, unwieldy affair. It is likely to incorporate virtually all of the negative predictors of successful adoption and implementation described above. The organizational uncertainties aroused in deployment would be considerable and the requisite firm level capacity for implementation would be slim. One can visualize the training and outfitting of firm, or NIOSH, psychological assault teams, interminably at the ready, for an idiosyncratic,

likely non-reportable event. The cost of the cure will far outweigh the cost of the epidemics.

Can an Epidemic-Focused Remedy be Evaluated? The previous discussion has centered on the general difficulty of disseminating and implementing a remedy for mass psychogenic illness. However, that general problem has serious implications for the research and development process, that would need to accompany the creation of any viable remedy. If we assume that a remedy will be focused on the outbreak per se - either preventively or as treatment--then the difficulties of implementation will virtually assure the impossibility of adequately evaluating effectiveness. If we assume that the desired outcomes, or dependent variable, of a remedial intervention is the rapid damping of an epidemic underway, then there are profound problems with N size and the possibility of even evaluating alternative treatment interventions.

For example, let us suppose that members of the research group are able to conjure up a hypothetically viable intervention program, based on their reading of the available literature. The parameters of that program are indeterminate,but a "package" intervention might hypothetically include the separation of the "initiator" from the rest of the work force early in the history of the illness outbreak, an attempt at debriefing non-affecteds in the work force, the disruption of social network channels of contagion, and perhaps a judicious application of psychological legerdemain. This would seem to be reasonable assembly of program features,but can it be tested in an evaluative sense?

If effect, each deployment of an innovative remedial intervention in a psychogenic illness outbreak would give us one data point for program evaluation purposes. Since most psychogenic illness outbreaks are non-repeatable events in a given firm, our experimental intervention would have to be at the ready for years to accumulate sufficient data to begin to make inferences about its effectiveness.

This also assumes that each data point replicate will be roughly equivalent. From the previous discussion, it should be clear that the implementation of an innovation in an organizational context is frought with difficulties, and the probability of achieving an accurate replication of a conceptual model is considerably less than unity. As has been pointed out by Hall and Loucks(1978), we can probably assume that a hypothetical innovative intervention would yield many different types of implementation "configurations." In some settings the remedial program would be implemented as designed and one-trial data on effect could be gathered; in other settings,more perverse implementation patterns would occur (including outright rejection by members of the organization) and the effects on data quality would be difficult to interpret.

In the evaluation of social programs, the program--or in this case the remedial intervention-- is the independent variable. If the program becomes adulterated in implementation, the possibility of a meaningful evaluation is nil. For an enlightening discussion of these problems the reader is referred to Boruch and Gomez (1977).

Another organizational issue relating to an evaluation of alternative interventions is what will constitute the control group. An outbreak of mass psychogenic illness is only defined as psychogenic after other toxic agents have been ruled out. The process of gathering air and water samples from the industrial site, whether testing turns out negative or not, constitutes a treatment in itself. In reading the case studies of industrial mass psychogenic illness, one gets the impression that some rather powerful psychological effects are being produced by toxic substance testing. Where does the investigative effort stop, and the remedial intervention begin?

The above discussion has attempted to highlight various problems relating to implementing, and evaluating, remedial intervention in an industrial organizational context. Most of this discussion has been congruent with the existing literature's implicit assumption that the epidemic outbreak, and post-outbreak events, are the most fruitful focus for study, and by extension, for possible remedial intervention. However, in the next section, we will consider a set of alternative premises: that the incidence of "psychogenic illness" may be the wrong dependent variable; that the unit analysis in these studies needs to be altered; that remedial action needs to be proactive and system-oriented, rather than reactive and individual-oriented.

If one reviews the history of social problems and responses to social problems, what will often emerge is a strong relationship between the conceptual-epistemological frameworks of reformers, and their choices of ameliorative action. If one is of a medical/biological bent, one will look for biological causality, and remedies; if one is oriented toward social systems, one will attempt to explain phenomena through that lens, and to seek social solutions. For example, the field of mental health involves a never-ending debate between advocates of biochemical explanations of behavior and chemotherapy treatment, versus champions of social determinants and social support interventions, A similar problem exists when discussing the problem of industrial psychogenic epidemic.

If one peruses the literature on psychogenic illness outbreak, there are several common themes apparent: (1) a focus on individual variables, and a considerable amount of implicit "victim blaming"; (2) a dearth of comparative organizational studies, or cross-cultural studies, although several social system variables seem implicated; (3) an implicit adherence

to a cryptomedical model of remedial intervention, that emphasizes dispensing psychological balm to "affecteds"; (4) an absence of consideration of vocational, familial, and social roles as explanatory concepts, and a disinclination to look at management culpability in the problem.

Let us consider each of these in turn. Individuals vs. systems: The unit of analysis. Perhaps the most pervasive tendency of the existing literature has been the repeated attempts to incorporate the phenomenon of mass industrial psychogenic illness into trait-state personality psychology. Despite the long history of "personality variables" being only minimally correlated with anything (Mischel, 1968), there have been continued attempts to relate the incidence of psychogenic symptoms to various individual descriptors, traits, and states. There have been assessments of hysteric personality through the Eysenck Personality Inventory, of coping style, of hypersensitivity to health, and so on. The usual methodology used in analyzing these variables is to gather comparative post hoc data from "affecteds" and "non-affecteds" in a population that has recently undergone a psychogenic illness outbreak.

The implicit assumption behind this research strategy is that if one could only understand how these individuals were affected in this bizarre behavioral contagion, then one could correspondingly prescribe "treatment". This approach begs several questions. First, it assumes that an exercise in postdiction will be equivalent to a predictive data-gathering approach. One could make the alternative hypothesis that any post-incident self-report data is likely to be colored by the experience itself. Second, there is little, if any, data indicating that differences found between affecteds and non-affecteds have any longitudinal or cross-situational generalizability. Are we to assume that the affecteds will henceforth, and in other settings, be inclined (and conversely non-affecteds not inclined) to exhibit "hysterical" illness behavior? Unfortunately, there are no data to suggest that this might be the case. In fact, as has been pointed out by Colligan and Murphy (1979), their general pre-incident general health status is well within the rage of normality.

The thrust of the above argument is that further research comparing affecteds and non-affecteds may be self-limiting in both the inferences that may be drawn, and in providing a rationale for a remedial intervention. There are other research strategies.

Toward Comparative Organizational Studies. One of the classical exploratory concepts in the literature on hysterical contagion has been the notion of "system strain" (Smelser, 1963). The assumption behind this concept is that social systems, or organizations, may evolve into a state of functioning in which

all members are under chronic undue stress and conflict. In other words, while not denying the reality of individual differences in coping with job and live-situation stress (anyone who believes in a normal distribution of human characteristics would concur) or in the role of small informal social networks in the transmittal of symptomatology, an argument can also be made that different social systems have different baseline levels of stress-inducing stimuli. If this ambient level of stress-inducing stimuli is above the thresholds of a significant number of organizational members,then a large number of stress-related behaviors will be observed; if that baseline level is above only a small percentage of organizational members' coping threshold, then only a few stress-related behaviors will be observed.

This tentative hypothesis has implications for considering the psychogenic illness problem. The bizarre behaviors reported in the psychogenic illness literature--hyperventilation, fainting, nausea--should be considered as low probability members of a whole class of behaviors that can be considered as negative manifestations of poor organizational climate. Included in this class of behaviors--particularly in the industrial setting--are such more normative phenomena as absenteeism, employee turnover, malingering, lowered productivity, poor supervisor-employee relations, and the like. A second related hypotheses is that if members of firms beset by an epidemic of mass psychogenic illness were magically transported to an organization in which the milieu were dramatically and positively different, the aberrant behaviors would disappear (nor would they likely have ever occurred) and related stress-produced behaviors would be markedly reduced in frequency.

To test these hypotheses would demand a somewhat different research strategy. The shift would be away from comparisons of groups of employees within an organization, and toward companions of normal organizations with those beset by epidemic psychogenic illness. This would approximate a matched control group design and could be relatively easily implemented. For example, one would need to first identify a sample of 10 to 15 industrial organizations in which mass psychogenic illness has occurred in the last five years. Then, a matched control for each such organization would also be identified--perhaps equivalent on firm size, employee demographics, profitability, and the like. Then a comparative data-gathering approach could be employed. Particular focus would be on two domains of measurement: (1) other stress related indices such as absenteeism, turnover, and employee satisfaction; (2) organizational structure and process variables such as decision making patterns, control profiles (Tannenbaum, 1968) role clarity, incentive structures, and communication processes. Special efforts might be employed to obtain historical, archival data not contaminated by the psychogenic illness outbreak

itself. The assumption, and guiding hypothesis, is that if such a comparative study were performed, significant differences would be observed in both domains of measurement, and that pre-existing social system attributes would be implicated not only in mass psychogenic illness, but in more normative measures of employee attachment to the work setting. If this were the case, what would be the implication of such findings for research into remedial interventions?

The problem of mass psychogenic industrial illness is nominally within the purview of industrial hygiene as a mode of inquiry, and under the bailiwick of NIOSH, in particular among the federal agencies. Unfortunately, that assignation has probably perpetuated the search for a medical-model solution, or technological "fix," for the problem. The focus of concern and activity may have to be altered significantly if mass psychogenic industrial illness is construed as merely another minor- albeit bizarre- indicator of work discontent with lousy work settings.

In our earlier discussion of classical organizational theory, it was mentioned that traditional views tended to assume that material rewards were primary motivators of worker behavior. However, in recent years, a more detailed consideration of the role of work in people's lives has developed. Although a consistently applied monetory reward system can motivate employee performance (Lawler, 1971), sometimes there are negative side effects. Moreover, there are considerable data to suggest that a host of non-monetary attributes of the work setting can have significant impacts on employee satisfaction, and sometimes, productivity. For example, productivity issues aside, there are data to negatively relate worker satisfaction with turnover and absenteeism (Srivastva et al 1979), with coronary heart disease (Work in America, 1979 P. 79), and with mental ill health (Kornhauser, 1963). Moreover, these relationships can become exacerbated by dead-end, low-paying, boring jobs--those mostly held by women, and those most descriptive of the type of jobs in the psychogenic illness literature (Work in America, 1971 pp.58-64).

If the conceptual--and empirical--case can be made that mass psychogenic illness is merely one more manifestation of general worker dissatisfaction, it will have interesting implications for what type of remedial intervention should be mounted. Rather than considered as a socio-medical problem, mass industrial psychogenic illness would become one more bit of evidence for the utility of social system change in American industry. Seen in this light mass industrial psychogenic illness <u>per se</u> would be a minor footnote to a more pressing public policy issue.

Towards Quality of Work Life. The thrust of the above argument is that remedial intervention in industrial organizations, and

related research, ought to have a considerably wider focus than that of the epidemic psychogenic illness literature. For example, an interesting recent development in American, and Western European, industrial organizational development has been loosely described as the Quality of Work Life movement. It encompases such activities as job redesign, the use of group performance incentives, enhanced participative decision making, and various permutations on all these themes.

By illustration, one relatively well-known set of naturalistic experiments has involved breaking up classical production lines, into batch processing teams of workers (Steers & Porter, 1975). The Swedish automobile companies, Volvo and Saab-Scania, have organized major portions of their operations around these principles. A team of workers--with stable membership--is assigned responsibility to assemble so many engines, or other major components. How they organize themselves, set their work pace, or interact among each other, are left to the discretion of the group. The effects seem to be reduced absenteeism and turnover, and positive impact on productivity.

In addition to these job design programs other interventions have attempted to alter both organizational processes, and organizational incentive structures. As alluded to above, one of the negative side effects of strictly monetary piece-rate incentive plans is the enhancement of interworker competition and potential conflict. There is evidence that some types of a collective contingency-reward systems can avert these effects. One of the most notable of such programs in the Scanlon Plan (Frost, Wakeley, and Ruh, 1974) developed over twenty years ago by Joseph Scanlon at M.I.T. Since then, the program has been implemented in a variety of industries including machine tools, automotive parts suppliers, furniture manufacturing, and office supplies.

The key features of the plan are the use of factory-wide committees (including representation by both management and workers) to evaluate changes in production procedures, and a bonus plan to collectively reward productivity gains. Unlike "suggestion box" formats, any suggestions for internal innovation are approved by representatives of stakeholders, and the monetary benefits that result from productivity gains accrue to everyone. The effect is to reinforce a common fate situation, and to give substance to the participation decision-making. Generally, the implementation of the Scanlon plan has resulted in remarkable gains in both worker satisfaction and productivity.

The most wide-ranging, and potentially impactful, Quality of Work Life effort has been undertaken by General Motors since the mid-seventies (General Motors, 1978). It should be noted that this effort has been undertaken in collaboration with

the United Auto Workers. The operationalization of the general concept has been translated into different schemes in various settings. For example, at one Fisher Body plant, the structure of the organization has been broken into several semi-autonomous, and decentralized, business teams. Each has its own policy-making committee, involving lower level participation. The result has been that several indices of worker satisfaction have taken a positive turn. At another GM assembly plant participation by workers was incorporated into plans for plant rearrangement, and a set of training programs in team problem-solving and communication were begun. Grievances have been sharply reduced and productivity enhanced.

Another category of work-life change has been labeled as "job enrichment," and a case study at AT&T is instructive (Ford, 1973). Here a typical rationally organized office structure proved to be dysfunctional in terms of both performance and satisfaction indices, such as turnover and absenteeism. A job redesign effort including physically grouping workers together who performed a related set of functions for a common geographically-bounded group of customers. In addition, individuals had the scope of their job responsibilities expanded into a more personally meaningful array. Then, job-enriched positions were "nested" organizationally so that an atmosphere of group fate control, and teamwork could be enchanced. In one comparative experiment the absentee rate for an enriched group was 0.6%, and 2.5% for the control group. Task error rate 2.9% for the experimental group and 4.6% for the comparison group. Indices of both performance and satisfaction were altered.

These examples are not comprehensive but illustrative. The point is that industrial organizational interventions ought to be focused on larger issues than those represented by psychogenic illness epidemic. It should be noted that many of the dependent variables in these studies overlap with variables under scrutiny in the mass psychogenic illness literature.

Putting Mass Psychogenic Illness into Perspective. Several years ago Daniel Moynihan suggested that a policy of "benign neglect" be pursued regarding a well-known social issue. I would like to make a similar suggestion for the issue of mass psychogenic industrial illness. The question is whether this issue should remain an intellectual curiosity-piece, or a matter of public policy, and ultimately, publicly-funded intervention efforts. That question turns on whether the focus of scrutiny, or unit-of analysis, is the psychogenic epidemic outbreak per se of those behaviors set in the context of the organizational pathology of some segments of American industry.

It should be noted that no one, based on the existing literature,

has died from epidemic psychogenic illness, nor suffered long-term symptomatology. However, there are data to strongly suggest that the quality of work life can be contributory to alcholism, heart disease, divorce, and longevity. Considered in the aggregate these problems seem more ominous than the brief, bizarre, symptamatology reported in the literature on epidemic psychogenic illness. I would suggest that research in this area be refocused, to become part of a larger--and more socially significant--inquiry into the nature of industrial social organizations, and their impact on workers. I have seen enough of social scientists amusing themselves with intellectually fascinating but socially meaningless research problems to suggest otherwise.

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PSYCHOGENIC ILLNESS: INDIVIDUAL AND AGGREGATE DATA

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Both individual and aggregated company data relating the number of physical symptoms reported by people in companies in which large numbers of people were ill with no apparent physical cause to various individual, job satisfaction, and organizational climate variables are presented. There are a greater average number of reported symptoms in companies in which the total family income is low, there is dissatisfaction with the supervisor and personnel practices and a perceived lack of "Staff Support" and "Clarity" with respect to work assignments. It is argued that these data are more appropriately analyzed at the company level. Problems associated with aggregation of data and guidelines as to how to proceed are discussed. Research involving multiple companies and assessing organization-level variables as well as individual variables is encouraged.

The title of this conference, "The Diagnosis and Amelioration of Mass Psychogenic Illness" suggests that the phenomenon we are discussing is a group rather than an individual characteristic. In fact, in his review of published literature on this topic, Colligan (1978) has defined contagious psychogenic illness as "the collective occurrence of a set of physical symptoms and related beliefs among two or more individuals in the absence of an identifiable pathogen." While the psychogenic nature of the illness might be, and has been, debated, we would like to concentrate on the group aspects of mass psychogenic illness and what this means in terms of research strategy and the interpretation of data collected to date.

Until recently, all research on mass psychogenic illness or industrial hysteria has been on a case study nature (see Colligan and Murphy, 1979, for a review). While data were typically collected from many or all affected individuals, in all cases there was only one affected company or group of individuals. Case studies are certainly a rich and valuable method of inquiry,especially in the early descriptive phases of a study. However, in experimental or quasi-experimental work in which one desires to make generalizable statements, larger sample sizes are essential. Further, the point could be made that researchers engaged in studying mass psychogenic illness do not typically report their data in a way that indicates recognition of this fact. Treating instances of mass psychogenic illness as single events means that individual data should be aggregated to allow computation of group scores. Hence, a series of methodological problems concerning aggregation and the appropriate unit of analysis arise. In this paper, we will use data collected by NIOSH in eight different companies to illustrate some of these problems but also the reasonableness of treating companies (or work groups) as the unit of analysis in studying mass psychogenic illness. It should also be noted at the outset that the NIOSH data collection effort is the only one we have knowledge of which involved

the systematic collection of similar data in more than one affected unit.

The most commonly used research design has involved the comparison of affected and non-affected individuals on a variety of demographic/psychological variables collected by interview or questionnaire in a company which has experienced a mass outbreak of illness for which there is no discernible physical cause. In this design, the sample size is the total number of respondents and the implication is that those variables which correlate with the affected status of the respondents are, at least associated with, if not determinants of the illness. Data of this type collected by NIOSH on selected variables thought to be associated with the symptoms of mass psychogenic illness are presented in the first nine columns of Table 1. These data are all self-report questionnaire data,the dependent variable being the number of symptoms out of a possible 25 reported by the subjects.

The first four variables are purely descriptive measures of employee tenure, hours, and income. The next set of fifteen items were single item scales measuring the degree of satisfaction the respondents reported concerning various aspects of their job situation. The last 10 variables were measured by the Work Environment Inventory (Insel and Moos, 1974). Each of the 10 variables is measured by four dichotomously scored items. These 10 scales are defined as follows:

Involvement is the extent to which workers are concerned, committed to, and enthusiastic about their jobs.

Peer Cohension reflects the degree to which workers are friendly and supportive to each other.

Staff Support reflects the extent to which management supports workers and encourages supportive relationships among workers.

Autonomy is the degree to which workers are encouraged to be self-sufficient, make their decisions, and develop their own potential.

Task Orientation measures the degree to which the work climate emphasized good planning and efficiency and encourages people to get work done.

Work Pressure reflects the degree to which the press of deadlines and work dominates the climate.

Clarity measures the extent to which workers know what to expect in their daily jobs and how explicitly rules and policies are communicated.

Control is the degree to which management uses rules and pressure to keep workers under control.

Innovation is the amount of variety and change which are emphasized in the work environment.

Physical Comfort measures the extent to which the physical surroundings contribute to a pleasant work environment.

If one examines the correlations for the total sample (N=867) there are a number of significant, but relatively small correlations with the number of reported physical symptoms. The highest of these correlations,that of Work Pressure and Symptoms is only .182. Further, correlations across individual companies, are inconsistent; in many cases, the signs of the correlations are dissimilar. Some of this lack of consistency, or lack of significant correlations can be attributed to sampling error. Another possible reason lies in the possibility that there are really not any nonaffected people in these companies or that within a company everyone is similarly affected. If so, then there should be a restriction of range in the number of symptom variables and all correlations would be near zero. An examination of the average number of symptoms reported by the respondents in the eight companies indicated a range from 1.49 to 5.44. A one-way analysis of variance indicated the companies were significantly different, $F(7,818) = 5.64$, $p < .0001$, in terms of the average number of symptoms reported by their employees.

Our next step was to take these averages and correlate them with averages on the hypothesized stressors. This, then shifts the unit of analysis to the company level and leaves a sample size of 8. These correlations are presented in the last column of Table 1 and one can see at a glance that more are substantially higher than the non-aggregate data and 7 of the 29 correlations are statistically significant. The direction and size of the significant correlations indicate that the companies whose employees reported higher personal incomes had a lower number of average reported symptoms. Similarly, companies whose employees reported a greater number of symptoms had employees who expressed greater dissatisfaction with overtime pay, salary, the company disciplinary system and the determination of days off. Further, there was a lower perceived degree of Staff Support and Clarity of roles in these companies. Finally, there are a number of sizable, but non-significant correlations.

The thesis of this paper is that more studies of "mass" psychogenic illness or stress in general should treat the company or the work group as the unit of analysis, but before generating undue enthusiasm about the size of the aggregate correlations in Table 1, a number of cautionary statements are in order. First, it should be pointed out that all eight companies were affected and consequently, there should still be a restriction of range in the dependent variable and a resultant attenuation of the aggregate correlations reported in Table 1. This

situation involves analysis problems generated by the aggregation of data when indeed such aggregation in inappropriate or alternately drawing inferences about individual relationships based on aggregate data.

The comparison of data at different levels (company, work group and individual) produces aggregation problems. Robinson (1950) brought the "ecological fallacy" issue to the attention of social researchers forcefully when he concluded that one cannot use aggregated data to make inferences about individual correlations. He showed why the ecological correlation based on aggregated group data was likely to be higher than a similar correlation based on data collected from individuals except in the case in which individuals are randomly assigned to groups. That this is reasonable is clearly seen in Figure 1 where the scatterplot for all individuals indicates a correlation of approximately 0.5. However, if one computes the correlation between x and y using group means, the correlation would be 1.00 in the case depicted, or at least higher than the correlation based on individuals. This explains some of the difference between the total sample correlations and the aggregated correlations in Table 1. Hammond (1973) has identified this situation as one of "homogenious grouping" and rightly indicated that it is not a serious problem since the regression coefficients for individual and grouped data are identical in this case. That this is true is also evident in Figure 1, since the same straight line describes the relationship between x and y for individual and group data. Consequently, these regression coefficients are useful in drawing inferences about individuals from aggregate data. It should also be noted that Hammond, and sociologists in general, are usually concerned about inferences from the aggregate level to the individual when the individual level data are nonexistent or hard to retrieve, so the nature of these relationships is extremely important.

What Hammond labels aggregation bias, however, does present more serious problems when one hopes to make inferences about individuals based on aggregated data. Aggregation bias occurs when the relationship between the variables of interest is systematically different in the different aggregate units. These specification errors occur when deviations from equality of within group regressions are associated with other variables. For example,if one is interested in the relationship between religion and voting patterns, and the density of people of one religion or another affects the proportion of that religion voting one way or the other, one has aggregation bias. In a precinct where 95% of the voters are Catholic, custom and group pressure may be more effective in producing a solid Democratic vote.

Or, using an example from Table 1, if the questionnaire respondents in some companies are primarily married females with

large numbers of children there may be an artificially high positive correlation between total family income and the number of symptoms reported. This could happen because the phenomenon is more frequently associated with women and there is some indication that it is also associated with the strain arising from the financial and emotional responsibilities associated with a large family and the necessity to hold a job. This would mean that one should expect a positive correlation between the aggregate number of married women with children and the aggregate number of symptoms, when in fact this relationship has to do with response rates. Alternately, if there is a differing proportion of women respondents across companies and if there is the usual sex difference in income, a spuriously high negative correlation between aggregate personal income and aggregate symptoms would be observed. This could be produced through differential pressure to respond to questionnaires. In one data collection effort by the senior author the proportion of women respondents was twice as large as the male respondents. If nothing is done in some companies to encourage participation while participation in other companies is mandatory, the lower average income of females and their seemingly greater susceptibility to mass psychogenic illness would produce the observed high negative correlation between average personal income and average number of symptoms.

A similar spurious elevation of the aggregate correlation between one of the job satisfaction variables and the number of symptoms reported in Table 1 is possible. If the determination of days off is more important to women employees whose family obligations are greater, then differences in proportion of women respondents across companies would serve to raise the aggregate correlation between dissatisfaction with determination of days off and symptoms. It should be noted that within company correlations both the examples cited from Table 1 are quite different - with some negative and some positive. This, of course, is the difference among within group regressions which Hammond (1973) identified as a possible aggregation bias problem. Whether there is a problem or not depends on whether the within company differences are systematic rather than random. Obviously, then one needs to know the phenomenon he/she is interested in before data collection in order to collect data on the appropriate variables. It is equally obvious that this is not always possible.

If one has data on the appropriate variables, both at the individual level and the group level, there are a number of guides that indicate the likelihood of problems associated with cross-unit inferences and/or the appropriateness of aggregation. Recently, Burstein (1978) has evaluated the usefulness of three approaches to testing the appropriateness of grouping methods. Two of these methods - the structural equations approach (Hannan & Burstein, 1974) and the X - rule

(Firebaugh, 1978) - proved adequate in estimating differences in individual and group coefficients when various types of aggregation bias were present.

The question of concern has to do with the degree to which the regression coefficient relating x and y at the individual level (B_{yx}) and the aggregate level ($B_{\bar{x}\bar{y}}$) diverge. Hannan and Burstein (1974) dealt with the problem by expanding the linear model to include its relation to both X and Y. These latter relations are then used to forecast the consequences of grouping by any specific characteristic. Formally, the difference between these two coefficients is shown to be equal to:

$$D_{HB} = B_{yz.z} B_{xz} \frac{6_z^2}{6_x^2} \frac{6x - 6\bar{x}}{6\bar{x}}$$

where z represents an ordered grouping variable. This equation is instructive because it indicates the conditions under which there is no difference between grouped and ungrouped data namely,

(1) Z has no effect on Y when X is partialled out or $B_{yz.x}=0$.

(2) Z has no effect on X or $B_{xz}= 0$ or

(3) $6\bar{x} = 6x$.

When differences between grouped and ungrouped co-efficients are observed, then some subset of the three conditions listed above does not hold.

The divergence of $B_{\bar{x}\bar{y}}$ and B_{yx} is estimated by Firebaugh (1978) in the following fashion:

$$D_F = B_{yx.x} (1 - n_x^2)$$

where n_x^2 equals the correlation ratio of the between group to total sums of squares. This implies, as Firebaugh (1978) indicated, that downward cross-level inferences avoid bias when the group mean is unrelated to Y when X is partialled from Y.

Both of these approaches are useful in the single predictor case, but they become cumbersome and impossible as the number of regressors increases and/or the grouping variable is a nominal one. Most importantly, these techniques are useful only when one has both grouped and ungrouped data; when this is true, however, one can compare the coefficients directly.

Such prodedures might be useful when it is too expensive to analyze all the data, though a random sample of the cases would also be appropriate. In some instances, disclosure of indiviudal cases is unethical. When this is true, one could use the procedures above to assess the seriousness of the distortion resulting from aggregation; if there are no differences one could report aggregate data and draw inferences at the individual level.

One can also test for the significance of the difference between grouped and individual coefficients using Burstein's modification (1978) of the Fiege-Watts statistic (1972):

$$F = \frac{(b_{\bar{y}\bar{x}} - b_{yx})^2 - \frac{1}{SS(\bar{x})} - \frac{1}{SS(x)}}{SS(res)/N-1}$$

$SS(\bar{x})$ and $SS(x)$ are the sums of squares for X for aggregate and individual data respectively;
$SS(res)$ equals the sum of squared residuals from the individual-level regression model; and
N is the number of individuals.

Like other significance tests, this test is dependent on the sample size and the investigator is still forced to make decisions about what difference between coefficients is practically important.

Where does this discussion lead in terms of practical guidelines? First, one should heed Burstein's guides for collecting and maintaining our data; namely, 1) measure all variables at their lowest possible level; 2) data from individuals should be matched with data from the groups to which they belong; 3) all information should be identifiable at its lowest possible level with retrieval capabilities at multiple levels; 4) use whenever possible,measures with established reliability and validity. Further, any decision to aggregate individual data whould be based on sound conceptual grounds. This means that data on grouped variables which might affect within group relationships must be collected. We have no better guide concerning what data to collect than is suggested by our theory(ies) and understanding of the phenomena of interest.

Thus far the discussion of aggregation bias and/or the ecological fallacy has focused on the distortion which may occur when one attempts to infer something about the relationships among variables for individuals when one has only group data. Another question involves the aggregation of individual level data (for example, the attitude and demographic variables in Table 1) to get group scores which are then related to inherently group data (for example, company size or technology). Again, it must make theoretical or conceptual sense to say

that these attitudes/participation variables are characteristics of the company. For example, differences in the clarity of articulated personnel policies would result in company differences in the job satisfaction and climate variables hence they might justifiably be aggregated. Beyer (1978) has suggested that a minimal test of whether it makes sense to treat individual data as a group property is whether the ratio of the variance of responses between groups is significantly larger than the variance of responses within organizations. If this F-ratio is significant, we have support for the existence of a particular construct as a property of the groups in question. For all the variables reported in Table, 1, this F-ration was statistically significant ($p < .05$).

If the F-ratio is not significant, the construct may apply only to the individual level of analysis or the investigator may have erred in its operationalization. A non-significant F-ratio indicates lack of within group consensus concerning the variable in question, or in a psychometric sense, a lack of interrater consistency. When aggregation seems reasonable, conceptually but there is no unit consensus, a careful rewording of items may indicate to the respondent what level of analysis one is interested in. In this context, it may be better to ask questions which one is interested in aggregating in a descriptive sense rather than an evaluative one. For example, it would seem better to ask people how often they offer suggestions to management about work procedures than how they feel about participating in their company's activities.

In summary, two variables that are to be correlated must be at conceptually similar levels of analysis. The decision to deal with aggregate of individual data makes sense only when it is relevant to the question of interest. A theoretical base for one's investigation or at least an educated guess as to the relevant variables affecting the phenomenon of interest will dictate the level of abstractness. In the case of psychogenic illness, studies of organizational-level stressors would seem particularly appropriate.

In first suggesting the study of psychogenic illness at the organization level, we noted that the data presented in Table 1 are all data from "sick" companies; consequently, company scores on the symptoms variables as well as its correlates will be elevated and there is likely some restriction of range, though there were still significant company differences. How does one investigate the problem since instances of mass psychogenic illness are quite rare and we certainly don't know when to predict an occurrence of the phenomenon? If mass psychogenic illness is viewed as the final stage in an organization's reaction to stress, then we should be able to study random samples of non-affected organizations to achieve insight into organizational stress. Companies which experienced outbreaks were likely under stress for a considerable period of time and are probably not much different from many other

companies in which the reaction to stress is present but less dramatic. While such generalizations are obviously dangerous, those familiar with Selye's work (1974) will note that this idea is similar to his description of the general adaptation syndrome in various species. This is, individuals can carry on relatively normal lives under extreme stress for a long period of time before the final exhaustion stage occurs. We are proposing that an outbreak of mass psychogenic illness is similar to an individual's exhaustion stage.

What variables should one study as possible indicants of a stress-prone organization? Two recent conceptual reviews (Beehr and Newman, 1978; McGrath, 1976) of the stress literature as well as the data in Table 1 suggest some possible correlates of mass psychogenic illness. McGrath suggests three major sources of stress: the physical technical environment, the social interpersonal environment and the person. Each of these components also interacts to produce additional sets of variables. For example, a person entering a given social context gradually develops a role. Role ambiguity has been identified as a significant source of stress in several previous studies (for example, see House, Rizzo, & Lirtzman, 1970, and Table 1 of the present paper). Similarly, Beehr and Newman indicate a set of possible environmental stressors and a number of person characteristics thought to be associated with stress and suggest that there are also possible person-environment interactions.

Specifically, Beehr and Newman (1978) list such organizational characteristics and conditions as company size, job security, union constraints, required relocation, organizational structure, work hours, staffing policies, management style, evaluation, control and reward systems, socio-technical changes, training programs, advancement opportunities, and subsystem relations. Of this list, Beehr and Newman (1978) indicate that only company size has been related to stress. The data in Table 1 should encourage further study of the role of management and staff in creating stress. Role conflict and role ambiguity as determinants of stress have been frequently studied, but not to our knowledge as organization-level variables. We have no data on the degree to which the organization's external demands or conditions produce stress. We have no data relating stress to location of the work place and the availability of decent transportation, number and nature of clients and customers, union strength, government regulation, technological and scientific developments, suppliers of materials and services, and consumer movements. There is some information on the effect of job demands and task characteristics; for example, the effects of shift work have received some attention (Rentos and Shepard, 1976; Tasto, Colligan, Skiei, & Polly, 1978). Beehr and Newman indicate there is no available information on the effects of responsibility levels or the job characteristics thought to increase intrinsic

interest level of the work. Further, the economic problems or instability of the company or industry may serve to produce stress on company executives which in turn produces stress for others in the organization.

All of these variables could be conceptualized as organization-level constructs and all could reasonably be expected to contribute to the stress experienced by a group of people. Relative to the number of potentially important elements listed by both McGrath (1976) and Beehr and Newman (1978), the number of elements that have been studied is few. We think that a study comparing stress levels in various organizations to the organizational variables suggested above would be extremely valuable in increasing our understanding of stress in general and mass psychogenic illness in particular. Such studies should not rely solely on questionnaire measures; in fact, many of the variables suggested above could not be measured in this way. Finally, while this is not the focus of our paper, these studies should include the collection of longitudinal data and the use of "natural changes" to allow for stronger causal statements about the factors involved in stress.

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MASS PSYCHOGENIC ILLNESS: THE CASE FOR SOCIAL COMPARISON

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The occurrence of mass psychogenic illness (MPI) has been traditionally explained in a variety of ways, ranging from contagion and group influence analyses to consideration of social isolation (e.g., Kerckhoff et al., 1965). The present paper considers outbreaks of MPI from a different perspective, using research and theory on social comparison processes, affiliation, and evaluation of emotional states to derive hypotheses about the etiology of MPI. A general framework for this view will be developed, an analysis of the fit between this framework and the existing data will be made, and implications for experimental explorations of MPI will be discussed.

The literature on MPI is sometimes confusing and unsystematic. This occurs, in part, because of the nature of MPI; it is not only a spontaneous and relatively short-lived phenomenon (making prospective studies costly and difficult) but it is also seen at the time of its occurrence as caused by an external pathogen. Thus, it is only after medical or hygiene personnel have determined that the outbreak of illness has not been caused by a toxin that psychogenic considerations are explored (e.g., Colligan and Murphy, 1979).

While the occurrence and symptomatology of MPI have varied, certain consistencies across most outbreaks have been identified. In their review of MPI in organizations, Colligan and Murphy (in press) have listed five factors that seem to be characteristic of work environments in which outbreaks have been observed: (1) the presence of physical stressors, (2) boredom, (3) perceived pressures toward increased production, (4) strained labor-management relations, and (5)lack of interpersonal communication between workers. Clearly these factors can be considered contributors to stress and may thereby be linked to physiological reactions such as secretion of cortisol and catecholamines, and their consequences--increased blood pressure, rapid heart rate, and the like. More importantly, however, stress can also induce greater perception of bothersome or uncomfortable symptoms (e.g., Pennebaker, 1979; Baum, Aiello, and Davis, 1979). As Engel (1962) has noted, "What is felt includes not only such vague to precise mental content (the cognitive part), but also the awareness of physiological changes, such as palpitation, sweating, flushing, muscle tension or 'butterflies' in the stomach." These phenomena will beg explanation and the process by which workers go about labeling these internal sensations may have implications for MPI.

Physical stressors such as noise (Colligan, 1978: Shepard and Kroes, 1975; Stahl and Lebedun, 1974), air contamination (Shepard and Kroes, 1975) and noxious odors (Colligan, 1978; Colligan, Urtes et al., 1979; Folland, 1975; Kerckoff and Back, 1978; Philips, 1974; Shepard and Kroes, 1975) have been observed as antecedents of outbreaks of MPI. Glass and Singer

(1972) and others have shown that unpredictable and uncontrollable environmental stressors can have behavioral costs and physiological consequences for people even after the cessation of the physical stressor. Stress has been linked to discomfort, illness, and a range of other problems (e.g., Frankenhaeuser, 1975). A factor which compounds the effects of physical stressors in the workplace is that most of the labor performed by workers involved in MPI outbreaks is assembly line work, consisting of monotonous repetitive tasks. In their study of sawmill workers, Frankenhaeuser and Gardel (1976) found that people who did machine-paced, short-cycle tasks exhibited stress reactions as measured by increased ratings of strain and ill health as well as by heightened catacholamine production. As Frankenhaeuser and Gardell note:

> The quest for increased productivity in industrial countries has been most intense in industrial manufacturing. This has enhanced the pressure on industrial workers as expressed in ever-increasing demands on pace and effective use of working time, and in decreasing options for variety, relaxation, and social interaction at work. To some extent, the growing demands on the workers emanating from this development tend to be reflected in more frequent dispensary visits, sick leaves, and early retirements.

Thus, stressful work environments can have health outcomes and those characterized by low levels of responsibility, variation, and satisfaction have been associated with heart disease and mortality (e.g., Caplan et al. 1975).

It is interesting to note that in commenting on stressful environments, Frankenhaeuser and Gardell list three of the five common characteristics of settings in which MPI occurs. Boredom, increased pressure towards production, and limited contact with coworkers are indicated as stressors with health-related consequences. It should not be surprising, then, that environments in which these problems are confounded by environmental stressors and strained relations with management should be characterized by increased perception of illness symptoms.

This allows us to conclude that workers in settings in which MPI has occurred have probably experienced physiological symptoms of stress. Exposure to environmental stressors, boring repetitive tasks, and increased pressure all can predispose an individual to experience headaches, dizziness, nausea, fatique, and the like, many of which are the same as symptoms found in most cases of MPI (e.g., Kerckhogg and Back, 1968). It is our intention to demonstrate that the ways in which workers attribute these symptoms will be of great importance in predisposing them to psychogenic illness and hysteria.

Social comparison theory as originally formulated (Festinger, 1954) suggests that people tend to evaluate themselves, that is, their beliefs and their abilities, through comparison with both social and nonsocial standards. In the absence of objective, i.e., nonsocial, standards, individuals will seek to compare themselves with others who are similar to them. In some areas, it is relatively easy for individuals to evaluate themselves by comparing their performance with some objective criterion. In other areas, such as attitudes, there is a significant absence of objective standards for comparison, and social forms of comparison become more important to the individual for both evaluation and validation of his or her beliefs. In such cases, we would expect a greater amount of comparison with other individuals. Although the general case for social comparison is usually made in terms of attitudes, the process seems equally applicable to emotions (cf. Schachter & Singer, 1962). Reactions to stress or anxiety may be especially difficult for an individual to evaluate objectively. More recent work extending the theory of social comparison still hinges on this basic mechanism (cf. Latane', et al 1966, and Suls and Miller, 1977, passim).

Studies of affiliation indicate that some individuals do seek the company of others when they are in an anxious state. Schachter (1959) brought subjects into a laboratory where they were told the experiment was investigating the effects of electric shock. In the high fear condition, the shock was described in ominous terms: it would be intense and painful but would cause no permanent damage. Subjects in the low fear condition were told that they would receive very mild shocks which would not be the least painful. Subjects were then told that there would be a delay while the experimenter set up the equipment and were given a choice of waiting in a comfortable room with magazines and easy chairs or waiting in an empty classroom with other subjects. They were also asked to indicate how strong their preference was.

The results of this study indicated that the degree of anxiety was an important determinant of whether or not subjects chose to affiliate. In the high fear condition, 62.5% of the subjects chose to wait with others as opposed to only 33% of the subjects in the low fear condition. The preference to wait with others was also much stronger among highly fearful subjects.

Social comparison theory hypothesizes that individuals prefer to compare themselves with similar others. This notion is supported by another affiliation study (Schachter, 1959). Using a similar experimental paradigm, all subjects were given high fear instructions, but only half were given the choice of waiting alone or with other people waiting to be in the same experiment, that is, with people whose plight was similar to their own. The remaining subjects were asked to choose between waiting alone or with students who were waiting

to see their advisors, that is, with people who presumably were in a different set of circumstances. Significantly, more of the subjects who could wait with similar others chose to affiliate. Fearful individuals did not affiliate indiscriminately but tended to affiliate with others in a similar situation. Additional studies established that this selective affiliation was used by the subjects to evaluate the relative degree of their own arousal and its appropriateness for their situation.

Schachter also found large individual differences in the desire to affiliate that were related to birth order. First born individuals were more likely to seek out the company of others when fearful or anxious than were second borns, second borns were more likely to affiliate than third borns and so on. It might prove interesting to look at the birth order of individuals affected in a MPI outbreak, both as initiators or as followers.

In these studies, subjects were aware of the reason for their anxiety: the instructions were quite explicit about the nature of the shocks. The source of arousal outside of the laboratory may not always be so immediately recognizable. In such cases, individuals would search their environments for an explanation of their arousal.

To investigate the role of social comparison processes in evaluating physiological arousal, Schachter and Singer (1962) injected subjects with epinephrine, a drug which has sympathomimetic qualities. Some of these subjects (the Informed group) were given an objective referent for their arousal: the effects of the drug were fully explained to them. The remaining subjects (some uninformed as to the drug's effects, others misinformed as to its true effects) were told that the injection was a simple vitamin and were not led to expect the side effects actually produced, such as palpitation and tremor. All subjects were then placed in a room with an experimental confederate who played one of two roles in this situation: within the experimental context, he became either increasingly euphoric or he became angrier and angrier as the experimental session progressed.

The results of this study indicate that social comparison has a strong influence on the interpretation of physiological arousal. Informed subjects were relatively unaffected by the confederate's behavior. In contrast, the other subjects--uninformed or misinformed-interpreted their arousal in terms of situational factors, i.e., the behavior of the other person. Uninformed subjects placed with a euphoric confederate rated themselves as being in a suphoric state while those with an angry confederate reported feeling a significant amount of anger. Behavioral observations also paralleled these findings. In addition, all comparisons were made to placebo injected

subjects as well.

These results seem to indicate that two factors are involved in the identification of emotion: (1) physiological arousal and (2) cognitive labeling of that arousal. Labeling occurs as a function of establishing a cause and effect relationship between environmental stimuli and perceived arousal. New or ambiguous physical states, such as those associated with stress, will create pressure for people experiencing them to determine what it is they feel and what is responsible for their discomfort. It is possible, then, that MPI involves the perception of physical symptoms (induced by the stress characteristic of affected settings), and the way in which workers explain them. As Kerckhoff and Back (1968) point out, "To the extent that the new label "makes sense and is thus easily accepted, it will be adopted readily by all those who have the experience but have been unable to conceptualize it satisfactorily."

What we have proposed so far is that (1) work settings in which MPI has occurred are characterized by multiple stressors, (2) these stressors are responsible for changes in workers' physiological states, and (3) that many of these symptoms are the same as those reported in MPI outbreaks. We have also argued that workers will seek to understand their feelings, and that a primary source of information about them is other people. Thus, stressed workers will engage in social comparison in order to label their feelings. Under normal circumstances, workers would discuss these conditions and their reactions to them. By comparing their symptoms and their interpretations of them, these workers could be expected to come up with a reasonably accurate appraisal of stressful aspects of their jobs and their reactions to this.

In comparing epidemic hysteria with panics and riots, Sirois (1974) has noted that the conflictual object in the former is much less obvious. The "conflict object" is often unknown or not dealt with directly and it is frequently difficult or impossible for workers to openly acknowledge and deal with the conflict. Colligan and Murphy (in press) have noted little informational support prior to the outbreak of MPI and Kerckhoff and Back (1968) observed a greater reluctance among affected individuals to discuss job-related problems and work dissatisfaction. Smith, Colligan, and Hurrell (1978) report that affected workers seem to be more financially dependent on their jobs than nonaffected workers (e.g., were more dependent on their jobs for total family income, had more children at home, would be more likely to quit if they didn't need the income, and so on).

In industrial settings that have experienced an outbreak of MPI, opportunities for communication between workers are generally limited. This limitation may be due to such factors

as scheduling of lunch and break times (Stahl and Lebedun, 1974) or noisy environments (Colligan, 1978; Folland, 1975; Kerckhoff and Back, 1968; Philips, 1974; Shepard and Kroes, 1975). Such a curtailment of communication would make it difficult for workers to engage in the open communication and discussion which might enable them to attribute their somatic complaints to the appropriate job-related stress stimuli. And in addition to limited communication among workers, communication between workers and management is frequently inadequate or misleading (e.g., Colligan and Urtes, 1979).

In an unpleasant work situation containing stressed individuals who have limited opportunities for either objective evaluation or interaction with one another, there are two additional factors which are important in determining the outbreak of MPI: a triggering event and an index case or cases. Most of the triggers in industrial settings appear to be strange odors while triggers in nonindustrial settings range from rumors to spirits. The lack of a specified triggering event is more common in nonindustrial settings.

While a precipitating event is not always necessary, it is generally present in industrial settings. An index case is obviously essential and provides a potential explanation for the worker's physiological reaction to the stressful environment. As stated by Colligan & Urtes, (1979) "the detection of a strange odor...or witnessing others become ill, may have provided the affected workers with an explanation of their discomfort and triggered a contagion reaction."

If social comparison processes do play an important role in the spread of MPI, some relationship between social groupings and contagion would be expected. Evidence along this dimension is presented by Kerckhoff, Back, and Miller (1965) and Stahl and Lebedun (1974).

In their analysis of the "June Bug" incident, Kerckhoff et al noted a number of similarities among the affected workers: almost all were women, white, worked the first shift, and were located in one area of the plant. Two months after the incident, affected and nonaffected women were asked to name their three best friends; the experimenters also had a record of the date on which each of the affected women had been "bitten". The results indicated more links between affected women and other affected women than between affected women and controls (unaffected women):

> Affecteds were twice as likely to be chosen solely by other affecteds, and controls are twice as likely to be chosen solely by other controls. Finally, of the mutual choices participated in affecteds, three-fifths are with other affecteds. Similarly, of the mutual choices participated in by controls, two-thirds

are with other controls. (pp. 9-10).

The investigators divided the incidents of MPI into three major periods: "before," "Day 1," and "Day 2." Most of the cases (76%) occurred on Days 1 and 2, while 17% occurred beforehand. Of those who initially exhibitied symptoms, 50% were social isolates. Although isolates appeared during other periods (at least 25% of the affecteds), they were most heavily concentrated in the initial phase before the mass outbreak.

In a study of a different outbreak which replicated and extended the June Bug study, Stahl and Lebedun (1974) interviewed 20 employees at a university data-processing center following an outbreak of MPI among 35 of the 60 workers employed there. An index of job satisfaction, a four point scale of symptom severity, and a sociogram of friendship patterns were constructed. Respondents indicated that the first person to become sick was a central figure in the sociogram analysis. Stahl and Lebedun noted that "many of the workers appeared quite aware of the temporal sequence of who got sick first and what the specific symptomatology was. This was especially true among workers who were engaged in friendship networks with other workers. Social isolates, on the other hand, reported a few symptoms". Social networks also seemed to influence symptom severity. The friends of the workers with the highest severity scores (2 or 3) reported a mean severity score of 2.06, while the friends of those workers with lower severity scores (0 or 1) reported a mean score of 1.6. This would indicate that friends tend to exhibit similar symptomatology.

Interestingly, those individuals with the lowest level of job satisfaction tended to select as friends those workers who had the highest level of job satisfaction and vice versa. Although the Data Center was particularly limited in opportunities for communication between coworkers due to excessive noise and scheduling of lunch and break times, employees were able to form social networks (as evidenced by one respondent's complaint about the "set group of ten girls who don't let anyone in the group"). Given the limited amount of communication, workers may simply not have discussed job satisfaction with one another. Alternatively, if they did discuss this, the observed discrepancies in job satisfaction might have served to intensify the need to evaluate their reactions to their job rather than to validate them.

Both the previous studies indicate that MPI spreads throughout a social network and provides support for a social comparison model. The Kerckhoff, Back, and Miller study shows that affected women tend to be friends with other affected women, while controls are friendlier with other controls, while the Stahl and Lebedun study demonstrates a relationship between friendship patterns and symptom severity.

These two studies provide an interesting contrast between an incident of MPI in which the initial cases were social isolates and one in which the index case was a socially central individual. Since individuals are most likely to compare themselves with their immediate reference groups, we would expect the spread of MPI to be much more rapid in those situations in which the initial case is centrally located in a social network. This appears to be the case: while the June Bug epidemic lasted about one week and affected 62 of 965 workers, the Data Center incident took place on two consecutive mornings and affected 35 of 60 workers.

The existence of social networks or the establishment of social groups within the work environment is not always a necessary antecedent to an outbreak of MPI; in fact, work environments in which MPI occurs tend to inhibit the development of social ties, by making it difficult for workers to communicate with each other. While verbal communication may be difficult or impossible, the work setting generally is such that employees have considerable visual access to one another. When employees work together in a large open room, the "attacks" that an affected worker suffers can be visible to her coworkers. In the case of the more extreme symptomatology such as screaming and struggling violently with others (e.g., Chew, Phoon, and Me-lim, 1976), these attacks can scarcely be ignored. Colligan, Urtes et al. (1979) found that nonaffected workers reported hearing of more people becoming ill than affecteds, while affecteds reported witnessing more people becoming ill (author's emphasis). Observing coworkers having attacks which they attribute to some environmental stimuli can certainly be a potent standard for comparison with one's own somatic complaints.

In any incident of MPI, the total working population is not affected. While affected workers do not differ markedly from nonaffected workers, several factors seem to be related to susceptibility to MPI. Of these, absenteeism, age and educational level, and ethnic background are ones which are relevant to a social comparison analysis.

Prior work histories indicate a higher absenteeism rate among affecteds prior to the actual outbreak of MPI (Colligan and Murphy, in press 1979). Higher absenteeism means that these individuals will have even less communication with their co-workers, further decreasing already limited opportunities for social comparison. This, in turn, may inhibit labelling of job-related stressors and somatic complaints.

One possible explanation for the higher absenteeism among affecteds is that these workers are experiencing a stronger physical reaction to stressors in the work situation. Smith, Colligan, and Hurrell (1978) have noted that affected workers are more likely to report being bothered by environmental

factors such as noise, poor lighting, irritating odors, and temperature variations in the workplace and indicate higher levels of job-related stress as a result of such factors as work pace, task repetition,job insecurity, and lack of supervisory support. While the affected workers surveyed by Smith et al. reported poorer general health than unaffecteds, some studies using more objective measures of general health (e.g., previous health complications, reported medicine usage) have found no differences (Colligan, Urtes et al., 1979; Colligan, 1978). Kerckhoff and Back (1968) found that affecteds were more likely to have been taking pills or other medication prior to the outbreak but were not more likely to have "had to stay home because of organic sickness only during the past year".

Alternatively, it is possible that these individuals are simply more prone to symptom reporting. Research has indicated that affecteds have a greater tendency towards somatiziation (Knight et al., 1965) score higher on the Hysteria scale of the MPI (Colligan, Urtes et al., 1979; Colligan, 1978), and are more likely to seek medical attention (Kerckhoff and Back, 1978). In a follow-up study conducted 25 years after an outbreak of epidemic hysteria in Tristan de Cunha, Rawnsley and Loudon (1964) found that affected women were later characterized by frequent medical consultations.

If the differential absenteeism rate is a function of greater symptom reporting or help-seeking, this could reflect a stronger need to evaluate physical reactions among affected individuals. This notion is supported by Kerckhoff and Back (1968) who found that affecteds tended to focus on emotive or physiological responses rather than potential external sources. If this is the case, such individuals should engage more actively in social comparison in an attempt to evaluate their physical distress and would thus be more susceptible to MPI.

Affected workers tend to be young individuals with lower educational attainment (Colligan and Murphy, in press). While this is to some degree true of the entire work populations in the situations where MPI has occurred, this limited experience and educational background should make these workers more susceptible to MPI. Older and better-educated people may have more knowledge of, or access to, objective referents which would allow them to accurately evaluate their somatic complaints and reduce pressures for social comparison.

Ethnic background is also an interesting variable, although susceptibility to MPI does not seem to be a function of race, per se. Since individuals are most likely to compare themselves to similar others, social comparison theory would predict that an outbreak of MPI in an ethnically diverse setting would tend to be concentrated in one ethnic group. Support for this hypothesis is provided by the work of Chew

and his associates (Chew, 1978; Chew, Phoon, and Mae-Lim, 1976). Chew discusses an incident that occurred in a large television assembly factory which employed 65% Malays, 23% Chinese, and 9% Indians. Of the 84 affected workers, 83 were Malays; no Chinese workers were affected and one Indian Muslim was only "frightened". In discussing an incident that occurred in a southern manufacturing plant, Kerckhoff, Back, and Miller (1965) note that almost all of the victims...were white. Although data on the racial composition of the work force were not presented, Kerckhoff and Back (1968) did use black employees as a comparison group for affected workers.

Although information on birth order of affected workers is not available, such data could provide interesting insight into the role of social comparison processes in MPI. The finding that first borns are more likely to affiliate in a stressful situation (Schachter, 1959) was based on the presumption that first borns rely more heavily on social sources of evaluation and suggests that these individuals should be overrepresented among affected workers. Since communication between coworkers is minimal prior to the outbreak,first borns would be more likely to compare their symptoms with those of the initial cases and label them accordingly. In those cases where communication exists, or where first borns, under heavy evaluative pressure, initiate communication, the discussion of the circumstances which produced the stress-related symptoms may provide a nonpsychogenic explanation for the internal state and serve to circumvent or prevent the MPI outbreak.

The most common characteristic of affected workers is their sex. In their survey of 16 incidents of MPI, Colligan and Murphy (in press) note that 93% of the workers reporting symptoms were women. Although the populations observed were often predominantly female, women were still disproportionately represented among the affecteds. This is apparently not just a modern-day phenomenon. Sirois (1974) notes that demonopathic manifestations from the 15th-19th centuries were almost exclusively female. In his historical survey, Markush (1973) reports many predominantly female incidents of epidemic hysteria (e.g., biting nuns in the fifteenth century, nuns mewing like cats in the 18th) In 1883, Hirsch noted that women were affected either "exclusively" or "especially" by epidemic hysteria.

While purely sociocultural explanations for the preponderance of women among affected workers are certainly possible (i.e., the female sex role making displays of illness more acceptable or leading to greater attentiveness and responsiveness to physiological states), we would like to take an approach that explores the interaction between biological and social factors.

One of the indisputable differences between males and females

is that women have menstrual cycles. It is not our intent to debate the issue of whether menstrual symptoms (premenstrual syndrome, dysmennorhea) are genuinely physiological reactions or the result of culturally transmitted beliefs and attitudes. Whether physiological or psychogenic in origin, physical and emotional discomfort immediately preceding and/or during menstruation are reported by significant numbers of women. Consideration of the menstrual effects suggests several alternative explanations for their role in MPI. a) It could be argued that the combination of a hormonal system already out of balance from the adrenal response to stress and the usual menstrual changes produces, in general, the sort of erratic behavior which folklore often ascribes to women during the menstrual cycle. The tenability of this explanation, however, is minimal: Major reviews of the literature on menstruation fail to show any systematic erratic behaviors or psychopathologies associated with that condition (Ruble, 1977, and Sommer, 1978).

b) Recent psychological theory has emphasized attributional processes in shaping and determining behavior (Jones, et al., 1972, and Shaver, 1975). This view would suggest that women, during menstruation, would attribute any unusual or untoward symptoms occurring at that time to the most salient cause--in this case, their menstrual period. Stress produced symptoms stemming from job-related conditions would be explained as but another manifestation of menstrual distress. Unfortunately for this view, the implication is that a definite fraction of the women workers would, therefore, be less susceptible to MPI (instigation) which would result in an overrepresentation of men among the MPI affecteds. This is patently at odds with the data.

c) Menstruation may affect MPI by serving as an ancillary physiological factor; social comparison may be heightened if there are internal states other than the stress induced ones discussed above, which also call for explanation. For example, if there were cases of incipient flu present, the general loginess and aches preceding the major outbreak might represent an internal state needing evaluation and could trigger off social comparison and its products--homogenization of self-description and sick-like behaviors. The medical exams often given to MPI victims make it unlikely that flu and its symptoms are a feasible progenitor of the phenomenon, but other alternatives are worth considering.

As detailed above, MPI is more frequently observed in women than in men. It is possible that a unique feminine event, the menstrual cycle, is involved in MPI and contributes internal symptoms as well as providing a change in internal state, not classified as illness, which increases evaluative pressures. Of course, this possibility would only apply if a sizable proportion of the female workers were in synchrony with respect to their menstrual cycles. While this is unlikely on a purely chance basis, there exists some evidence that

such synchrony occurs as a consequence of female participation in the same work group.

McClintock(1971) has briefly reviewed the animal literature on synchrony of oestrous and concluded that conjoint living produces such synchrony in mice and other species. More cogenly, she conducted a study of college students in a dormitory which demonstrated that over the course of the school year, both close friends and roommates, although asynchronous at the start of the year, had closely correlated cycles just a few months later. Appropriately analyzed control groups rule out the possibility that the school pressures, exams and the like produce synchrony. Equally germane to this discussion is the report by McClintock that seven female lifeguards, whose menstrual onsets were scattered at the beginning of the summer, after three months of working together,had cycles with onsets within a four day span. This suggests that synchronous work cycles and physical proximity may be sufficient conditions to trigger menstrual synchrony.

There are indications, but weaker ones, that similar coordination of endocrine functions exists for a variety of other cyclically produced hormones as well (e.g., Orth and Island, 1969; Aschoff, 1976: Zucker, 1976; Fulkerson, 1978; Kaul and Berdanier, 1975). These cycles may also provide pressures for evaluation. While we are not attributing MPI to menstrual distress, we are suggesting that the evaluative activities triggered off by work related stressors might well be augmented by a variety of synchronous endocrine changes, of which menstruation is the best known. These changes simultaneously affect a large part of the work cohort with yet another internal state to evaluate. The menstrual history of women involved in previous MPI outbreaks have not been reported, but might be worth monitoring in future occurrences.

There are two major problems in understanding mass psychogenic illness in industrial settings. The first is explaining why it occurs; the second is explaining why it does not always occur. Logically, there is no reason why MPI could not be a similar product of several different causes, but the consistencies in the characteristics of workers, settings, and events noted both by previous reviews and in this paper suggest that a similar process is at work. Of the many possible processes which could be utilized as an after-the-fact explanation, we have made a case for <u>social comparison</u> and we have done so for several reasons;

a) The theory of social comparison processes provides a rather neat conceptual fit to the data. It can explain a way in which MPI comes about.

b) The theory can explain the circumstances under which MPI does not occur, that is, it can point to conditions likely, other things being equal, to prevent MPI.

c) The theory can help specify some of the characteristics of

those people likely to be susceptible or resistant in a particular case of MPI.
d) The theory can point to particular measurements, e.g., birth order, menstrual data, which can provide inferential support for intermediate processes of the proposed mechanism. It has the virtue of being falsifiable both in whole or part, and hence, modifiable.
e) The explanation provided by the theory is concrete enough to provide a guide for experimental tests of MPI either by analogy in the laboratory or directly in the field.

One final word. MPI, as with many other psychosomatic illnesses, is a real illness. That there is economic loss is clear, but it should also be noted that the victims of it are ill--the lack of a specific pathogen to be held accountable does not mitigate their real distress. It would only compound their illness to imply that because the instigating factors are psychological their illness is "only in their heads". They would then be justified in voicing remonstration similar to the one contained in the old textile song, Hard Times In the Mill, "Ain't it enough to break your heart? You work all day and at night it's dark".

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COMPLEXITIES, CAUTIONS AND CONCEPTS IN RESEARCH ON MASS PSYCHOGENIC ILLNESS

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This paper will try to bring some conceptual and methodological ideas from certain other research areas to bear upon research on "Mass Psychogenic Illness". The latter, a content area with which I have only recent and limited familiarity, has a surprisingly rich and extensive literature. I found three reviews especially useful, both as secondary sources and as sources of useful concepts and hypotheses. An historical review by Markush (1973), titled Mental Epidemics, deals with 100 "epidemics" from the tenth century until 1967. Sirois (1974) covers 70 cases of what he titles Epidemic Hysteria, from 1876 to 1972. His coverage is broadly international. Colligan and Murphy (1979) provide a detailed analysis of 16 case studies in work organizations and schools, titled Mass Psychogenic Illness. Much of the substantive material of this paper is drawn from those three reports.

The first section of this paper will discuss some of the complexities of the problem, Mass Psychogenic Illness in organizational settings (MPI). That section begins with a brief description of how the problem varies as it is viewed from different perspectives. Then some special features of the MPI problem that makes it especially difficult to deal with as a research problem are described. The third part of the first section considers the underlying logical structure of the research questions involved in this problem area, and tries to specify just what comparisons are needed in a study in order to draw useful conclusions from it.

The second section of this paper steps back from the immediate problem and considers five very general conceptual and methodological cautions that describe potential traps for unwary researchers in a number of problem areas within the behavioral sciences. Three of these derive from the sad fact that we behavioral scientists rely very heavily on the use of verbal labels to define our key concepts. The other two are methodological problems which reflect more or less inevitable dilemmas within the research paradigms of the behavioral sciences. One has to do with measurement of key variables; the other has to do with research design in general.

The third section of the paper is an attempt to lay out some concepts that may help build a model of the focal problem. The section begins with a very brief overview of major findings and of major theoretical approaches in this problem area. These overviews rely very heavily upon the three sources noted earlier, especially Colligan and Murphy (1979). Then, two

especially central constructs, exposure and susceptibility, are examined as a basis for development of a model of the problem, and are related to the main substantive findings already reviewed. The section, and the paper, ends with an attempt to integrate all of this into a single coherent formulation.

II. Some Complexities in the Problem

The Problem as Viewed from Different Perspectives

We are dealing here with situations of the following general class: There is a particular setting--an industrial plant, a school, or the like. During a particular, bounded period of time, but not at earlier or later times, some but not all inhabitants of that setting exhibit more or less the same set of symptoms. The set may involve both physical and psychological symptoms; for example, it may include nausea, headaches, vomiting and anxiety. Both the individuals who exhibit the symptoms, and at least some of those who do not, believe that the symptoms have been caused by certain triggering events or conditions (e.g., bug bites; a noxious odor; a viral desease). Certain other relevant people, such as setting managers, physicians, researchers, on the other hand, do not believe the symptoms to be causally related to the alleged triggering events or conditions, or at least not to be related to them in terms of our usual instrumental-technical scientific-medical model or paradigm of cause-effect relations.

Such patterns of behavior are referred to, in this symposium, as "mass psychogenic illness" (MPI). Elsewhere, they have been called mass hysteria, mental epidemics, fads, panics, and the like. They represent a "problem" because:

(a) The symptoms produce discomfort for the persons affected by them.
(b) The existance of the symptoms for a number of people provides negative publicity for the setting, at least, and may indeed "generate" other sufferers of the same pattern.
(c) Since the symptoms are not considered to be "real" consequences of the alleged "triggering" conditions, by persons who are either powerful in the setting or experts brought into the setting, the symptoms are also not considered amenable to effective treatment by treating the alleged causal agent. Indeed, the symptoms might not be treatable in any case.

The situation poses a problem for all parties involved. But the nature of the problem, and certainly the conditions for solution of the problem, differ for different parties: the victims, the non-victims, the setting managers, and the researchers.

For the affected individuals, the problem is one that could be eliminated if the symptoms were to cease, and if those symptoms proved to have limited negative after-effects. For the unaffected inhabitants in the setting, the problem is a problem because they might yet be affected. From their point of view, it could be eliminated if the symptoms were to stop for the "affecteds", and if "affection" were to stop spreading. For setting managers, the problem is a problem because it is costly, in terms of lost manpower and effort, and because it may have long-run negative effects on the setting (such as recruitment difficulties). The setting managers' problem could be eliminated if the symptoms were to cease, the "spreading" were to stop, and no causal agent for which they are responsible (such as toxic substances or noise) were identified as the "actual" cause.

The problem is also a problem for physicians/researchers/epidemiologists who come to the setting to study it. For them, the problem does not cease until they, collectively, can develop and verify an "explanation" for the occurrence.

Thus, the problem is not altogether the same when viewed from different perspectives: and the conditions that represent a solution to the problem for different parties in the matter are sometimes in conflict with one another. So any clear analysis of "the problem", and potential solutions to"it", must begin by specifying whose problem is to be dealt with--whose axe is to be ground!

Note for example, that from the point of view of either the researcher or the setting managers,it would not be a problem, or at least it would be a quite different problem, if any one of the following things were true:

(1) If only a very few setting inhabitants had "it".
(2) If "it" (that is, the symptom pattern) differed from one victim to another.
(3) If the explanation of the symptoms--even the same symptoms--differed from one victim to another.
(4) If it was clear that baserates inside the setting were comparable to baserates outside the setting.
(5) If "experts" believed the alleged causal link.

Note also, that, in all of these instances, while the problem would vanish or be much altered for both researchers and setting managers, the problem would still be the same, and equally intense, for the victims.

The problem would also vanish, or be radically altered if any of the following things were true:

(6) If everyone in the setting got it (or, of course, if no one did).

(7) If the same thing had happened in this setting, with regularity, in the past.
(8) If the same thing was happening simultaneously in all comparable settings.

Here, again, the problem would be eliminated or altered for the setting managers and researchers; but it would remain as serious for the affected individuals.

It is probably worth asking whether or not it would be a problem if the symptoms were defined as positive--euphoria, pleasurable, spiritual--even if those symptoms led to an equal loss of personnel time on the job.

Some Special Features of the Phenomenon as a Research Problem

The phenomenon of mass psychogenic illness, viewed as a research problem, has several special features that make it especially hard to study in a systematic, scientific sense. A case of mass psychogenic illness can only be identified post facto. It has to already have happened--a number of persons have to have already "caught" it--before setting managers and/or researchers can know it exists. Moreover, it can only be identified residually. That is, it is only after setting managers have explored and eliminated a number of physical-medical alternatives--germs, toxic substances, and the like--that they reach the conclusion that "it" is a case of mass psychogenic illness.

The problem of deciding when something is and is not a case of MPI is quite complex, and also quite crucial both politically and scientifically. As long as MPI must be defined residually, it is, strictly speaking, logically impossible. One cannot prove the negative case. Furthermore, as various branches of basic and applied research relevant to industrial hygiene have advanced, it has become clear that humans are susceptible to ill effects from an enormous range of substances and that in some instances such susceptibility involves long time accumulations of exposures to tiny amounts, and may even involve interactions of a complex of conditions. Such complexities make it even more difficult to decide, at a high level of probability, that a certain shared set of symptoms is not due, at least in part, to some biological or chemical agents.

There has been some discussion of the need for ways to establish "positive", rather than residual, diagnoses of MPI. That would certainly be desireable, for both scientific and political reasons. But like many other "needs" in the methodology of the behavioral sciences, the strength of the need is uncorrelated with the probability of the attainment. I do not think it likely, on either logical or practical grounds, that we will develop broadly acceptable "positive" bases for

defining an MPI in the near future. So, MPI researchers will probably have to continue to work with a phenomenon that is defined residually--with all of the undesirable political and scientific consequences that that entails.

A third constraining feature of MPI is also definitional. The setting managers, and the MPI researchers who have been brought into the situation by them, are committed to a definition of the "cause", that is, in direct conflict with what the affected persons (and other potentially affected persons in the setting) believe to be the case. The affected persons, and others in the setting, believe that the common symptoms are caused by a particular agent or event--a bug, a toxic substance, a specific disease pathogen. The setting managers are specifically and explicitly disputing this belief when they bring in a group of "mass psychogenic illness" researchers. And those researchers,by definition, are siding with the setting managers in that definitional dispute.

Because of these special features of the problem, the mass psychogenic illness (MPI) researcher has several strikes against him or her before the inquiry starts. Much of the information must be gathered by retrospective methods from various sets of people, many of whom have some stake in maintaining a certain definition of the problem and its cause. The affected have a stake in maintaining their definition of the causal agent; otherwise, they are to some degree responsible for their own problem: they are deluded, gullible or malingering. Researchers will need to get some of their information from those affected individuals. They will have to do so under less than ideal conditions of subject-researcher rapport.

Other inhabitants of the setting, who have not yet become affected (but might), may also have a stake in maintaining, or at least considering, the "triggering event" theory of the outbreak. Setting managers, on the other hand, have a stake in redefining the problem as a case of "mass psychogenic illness", for several reasons. First, they are likely to have some degree of responsibility for the occurrence of the triggering event (e.g., a toxic substance). Second, they certainly have a stake in getting the problem resolved so that they do not have continuing costs to bear. The researchers--I mean those investigating this "mass psychogenic illness"--certainly have a stake in maintaining that MPI definition rather than the one held by the affected individuals. Otherwise, there would be nothing for them to investigate!

Retrospective data is difficult at best. In the MPI case, it is even more difficult to get accurate information from the people in the setting. All of this makes it even more crucial that the researcher search for convergence among many different sources of information. (There will be more discussion of convergence of measures, and of multiple operationalism, in

the second section of this paper). The post facto and residual nature of the MPI problem also impose some serious constraints on potential research designs. For one thing, it is not possible to conduct an experiment, in the laboratory experiment sense of a deliberately-imposed manipulation of levels of a key independent variable. It might be possible, though, to design what is often called a "natural experiment". It certainly seems possible, at least some of the time, to take advantage of what Campbell and his associates (e.g., Campbell & Stanley, 1965; Cook & Campbell, 1976) have called "quasi-experimental designs). Cook and Campbell (1976) have identified nearly two dozen designs which can offer some degree of inferential power, even when full experimental control is not possible. Of these, some of the time-series quasi-experimental designs may not be possible for most MPI cases; but others might be. Furthermore, a number of quasi-experimental designs, of correlational, regression discontinuity, and non-equivalent control group forms, potentially can be used in many MPI research studies. In any event, it is important that the researcher not use the unfeasibility of doing a "true experiment" as an excuse for not being concerned with having appropriate controls and comparisons in the study. (This, too, is discussed in the second section of the paper.)

A Formulation of the Problem in a Comparative Context

One of the striking features of research on MPI is that it does not really come to grips with the baserate question. To paraphrase the old vaudevillian's one liner: "Compared to what?" In an MPI, some people (but not others), in a particular work or school setting (but not in other settings of apparently similar kinds), during a particular period of time (but not at earlier times), exhibit certain symptoms, which either mirror those of an index case or mirror those that would be expected to follow a certain triggering event that is believed to have occurred. It is not sufficient to consider characteristics of those victims in comparison to characteristics of others in the situation who did not "break out" with similar symptoms. Furthermore, we must ask why MPI occurred. One easy answer to that question is that no one got the MPI symptoms in other settings because the triggering event (or index case) of the MPI setting did not happen there. But how many studies even consider that question, much less actually collect the data necessary to answer it?

The problem is still more complex. Why didn't all of this occur in this setting before now? Again, the argument that no triggering event occurred earlier is implicit and undocumented. Moreover, we not only need to ask why these people and not others; we also ought to ask why these symptoms and not others. In other words, why is it that this triggering event or index case was modeled, rather than some other event?

So, we can translate the "compared to what" question into a set of questions: Why these people? Why this setting? Why now? Why these symptoms? These questions imply that MPI studies must be done not with a single control group but with a whole spectrum of control comparisons. (This is, of course, the case for many problems in the behavioral sciences, even though it is seldom noted and even less often practiced.)

Let us consider two sets of persons, one set that falls victim (V) and the other set that does not fall victim (v') to the MPI symptoms. We can assume that there may be some differences between those two sets--whether these be personality traits, or biographical characteristics like sex and age, or past behavioral traits like absenteeism. We ought to be able to identify a comparable two sets of people in other settings in which the outbreak did not occur. We can also identify these sets of people at an earlier time. We can identify, then, eight separate "cases":

Case A	Victims(V) of MPI in setting S at time T: VST
Case B	Non-victims (V') in S at T: V'ST
Case C	Those same victims (V) at an earlier time (T') in S: VST'
Case D	Those non-victims (V') at T' in S: V'ST'
Case E	People like the victims (V) who are in situations like S but in which MPI did not occur (S') at T: VS'T
Case F	People like the non-victims (V') in S' at T: V'S'T
Case G	The victim-like people (V) in S' at T': VS'T'
Case H	The non-victim like people (V') in S' at T': V'S'T'

The problem for the MPI researcher is not just to describe differences between V and V' in S, T. (That is, differences between case A and case B). The MPI researcher must also ask how V and V' differed in ST', in S'T, and in S'T', even though neither set showed the MPI symptoms in any of those cases.

The MPI researcher also needs to account for why V in S, T (Case A) showed MPI symptoms while V in S, T' (Case C), in S'T (Case E), and in S'T' (Case G) did not.

The main point here is that the MPI problem implies a very complex set of comparisons. These comparisons involve individual differences, situation differences, and temporal differences. Furthermore, it is unlikely that we will solve the puzzle in terms of any simple personality difference between those who are and those who aren't affected by an MPI (that is, $V \neq V'$ on property X_1), or even in terms of differences on a whole set of individual properties (that is, $V \neq V'$ on properties X_1, X_2, X_3, ...X_n.). It is much

more likely that we will come to understand MPI in terms of complex interactions of personal differences, differences in time, and differences in setting. We will explore some of the implications of these complex comparisons in the third section of this paper, after first considering some more general conceptual and methodological difficulties.

II. Some Conceptual and Methodological Cautions

While the MPI problem has certain special features, some of which have been noted and discussed in the preceding section, it also has many aspects in common with a number of other areas of social and psychological study. These include areas such as the study of factors in human stress, the study of the spread of innovations, and the study of the occurrence of accidents. Furthermore, the MPI area shares certain conceptual and methodological problems with all of the social and behavioral sciences. Some of these represent serious and subtle pitfalls that are particularly likely to ensnare the MPI researcher. This section is an attempt to offer certain cautions which, if heeded, could help the MPI researcher keep out of at least some of these pitfalls.

The Tyranny of Words

Several of the pitfalls, and the associated cautions, arise because much of social and behavioral science is heavily dependent on concepts formulated in words. When we use words to label constructs in social science we can mislead (ourselves and others) in essentially three ways, and those three ways are the bases of the three cautions to be listed here.

Caution #1: Try not to let the terms you use prejudge the answer to the question you are studying.

Sometimes researchers label a phenomenon that they intend to study with words that are heavily value-laden. This can have the effect of shaping the researchers results before the inquiry starts. The study of stress provides a classic example of this problem. Researchers who study "stress" find that high levels of "it" lead to "deterioration" of performance. They seek ways to cope with "it". They sometimes try to find factors that give people stress resistance. Some other researchers, who study what seem like the same or related phenomena but do so under labels such as "activation","arousal", or "motivation", find that increases of "it" lead to improved performance. They seek ways to help people not lose "it." Coping, a good thing, becomes habituation, a bad thing. They certainly don't try to find ways to make people resistant to such motivation.

Examples abound in other areas as well. Those familiar with

the literature on social influence will recognize that when "conformity" vs "deviation", as studied by Sherif in the thirties, became "yielding" vs "independence"as studied by Asch in the late forties, essentially the same responses to social pressure changed places in terms of which was "good" and which was "bad". Social psychologists aren't the only sinners. The same reversal of good and bad meanings for a construct occurred when the religious term,resistence to temptation, was replaced by the psychoanalytic term,repression.

The MPI researcher should consider this idea very carefully. Calling these things "hysteria" has already been recognized as perjorative--and perhaps sexist. Calling such things panics, or mental epidemics or even mass psychogenic illness, also connotes some answers to the very questions being studied: What are the causes of the symptoms? How do they spread? Who is responsible?

Even though there is already a plethora of names for the phenomenon--mass hysteria, mental epidemics, mass psychologic illness, and the like--I want to add one more. Let me suggest that we label the phenomenon Multiple Occurrences of Unexplained Symptoms (or MOUS). I am trying to choose a label that is descriptive, but that avoids prejudging the nature and causes of the phenomenon. I avoid "mass" because some of the cases covered in the reviews involved only 3 or 4 people,many involved less than 10. I avoid "contagious" and "epidemic" because they imply the answer to the question: "How do the symptoms spread?" I avoid "psychogenic" because I don't know how one decides which vomiting, which headaches, and which nausea are "physical", and therefore "real", and which are in some sense "unreal". I also avoid the term "illness", because some of the incidents covered in past reviews could scarcely be called that, except in metaphorical terms. The "dancing mania" of centuries past and the "rock concert" and "disco mania" of current times may differ more in our cultural interpretations of them than in manifest behaviors of the "affected". Why are the former "illnesses" and the latter "fads" or "recreation"? I even avoided the term "outbreak", which seems to me to imply that the phenomenon is entirely non-volitional. I don't think even that should be decided in advance.

So, I offer the label: Multiple Occurrences of Unexplained Symptoms. (MOUS!) I'd welcome a good"E" word at the end, to round out the acronym!

In the present context, we are more interested in MOUS that (a) occur in industrial/organizational settings; (b) are interpreted by the inhabitants as involving negative symptoms; and (c) are interpreted by setting managers, and/or outside experts, as not having a specific pathogen and more precisely, as not having been caused by what the affected (and perhaps

the unaffected in the setting) believe to have been the cause. But even within these limits, what we call the phenomenon is important because that label guides our search for causes, consequences and understanding.

Caution #2: Try not to let phenotypic similarities, especially those based solely on verbal labels, mask genotypic differences.
In the social and behavioral sciences, constructs are defined in verbal terms. As the Humpty Dumpty character in Alice in Wonderland has pointed out, we can make words mean whatever we want them to mean. So, when a researcher applies a given verbal term to a given phenomenon, operation, variable or event, that researcher and others are likely to proceed on the assumption that "it" is somehow the same as everything else that has received that same verbal label.

An example from the stress area is again appropriate. The array of events and conditions that have been called stressors or stress conditions is remarkably broad and diverse. It includes:

(a) night infantry combat;

(b) wintering over in the arctic;

(c) failing a test;

(d) losing a loved one;

(e) being told that other college sophomores in an ad hoc group don't like you;

(f) being told that other college sophomores in an ad hoc group disagree with you;

(g) undergoing potentially fatal surgery;

(h) being divorced;

(i) being married;

(j) being out of the company of other people (in one case, an infant being out of sight of mother for a whole minute).

It is powerful magic, indeed, that enables the researcher to make all of these things somehow the same merely by giving all of them the same verbal label (and waving a PhD at them as a magic wand). This problem, too, is not limited to the stress area. Consider, for example, the array of things that have been taken to be "group productivity," or "aggression", or "liking", or "attitudes", in the literature of experimental

social psychology.

The MPI researcher should keep this caution in mind, too, when he or she sets out to identify, classify, or catalogue events that will be considered members of the class of phenomena being studied. Is the reaction to the Orsen Wells version of H. G. Wells War of the Worlds the same kind of thing as the early Children's Crusades? Are the activities of the Flagllants of European "dark ages" to be assigned to the same class of phenomena as the various polio scares of the past half century? Are the various "dancing" manias" of the tenth through sixteenth centuries in middle Europe the same kind of thing as the modern industrial MPI? A review of the literature of this area might lead one to conclude that the answer is"yes"to all of these questions. At some point, a verbal label that is used to cover such a wide array of events loses its usefulness as a scientific construct. Internal differentiations may be needed. Sirois (1974), for example, describes five different types of MPI, with differentiations being made primarily in terms of the time course of the "epidemic". Perhaps there is a need for more analytic effort toward developing taxonomies of types of MPI.

Caution #3: Try not to let phenotypic differences, especially those based on verbal labels, mask genotypic similarities. This is the obverse of Caution #2. Just as merely attaching a particular verbal label to two sets of events does not make them the same thing, so merely associating different verbal labels with two sets of events does not guarantee that they are dissimilar. Social scientists have the bad habit of inventing new labels for what persons with less at stake might see as the "same old" concepts. I have always been surprised, for example, that the concept "obedience" was treated as if it were a totally new idea, by researchers in a field that had been studying conformity, deviation, authoritarianism, and other aspects of social influence for several decades. I have also been amazed at how many different terms there are for positive interpersonal regard (terms such as liking, positive affect, positive interpersonal perceptions, positive sociometric choices, cohesiveness, and the like). A colleague and I once labeled this kind of labeling as a search by the social scientist for "marginal differentiation of product" (McGrath & Altman, 1966).

Sometimes it can serve a very useful purpose for a researcher to consider the similarities between two or more sets of phenomena that have been given different names and treated as different "things". One example of such gain, from the stress literature, is in the very fruitful sets of ideas that have come about because some stress researchers decided to consider certain stimulus "under loads" (e.g., social isolation) as potentially related to task or stimulus "overload"--related, that is, in their stressing effects on the "loadee".

In this context, then, it probably is worth asking the MPI researcher whether the dancing manias of the earlier times ought to be considered as related to the current disco dancing popularity, or the recent popularity of rock concerts. Similarly, it ought to be worth asking about the similarities, if any, between cigarette smoking and heroin addictions on the one hand (both of which are considered by Markush as mental epidemics); and on the other hand, such phenomena as: (a) the relatively rapid rise in use of sugar in certain western countries; (b) the vast increase in caffeine addiction: (c) the rapid spread in television watching; and also (d) even our "addiction" to various forms of higher education? I am not proposing that any or all of these matters ought to be labeled as "problems", much less as MPI problems. I am suggesting that analysis of the features that these matters do and do not have in common with MPI may be well worth the effort in terms of new insights gained.

In summary, then, MPI researchers, like researchers in other social science areas, must understand that they cannot make things good or bad, similar or different, by merely labeling them. But they also must understand that, by labeling concepts in particular ways, they may mislead others, and even themselves, regarding these matters.

The Tyranny of Logic

There are several pitfalls--indeed, dilemmas--that arise from the central fact that social science is committed to the always-fallable empirical measurement of constructs, and to the always-limited and always-uncertain inductive logic of inference. While the consequences of these pitfalls are legion, I will concentrate here on two cautions. One deals with empirical measurement and its meaning. The other deals with research design and its consequences.

Caution #4: Because all measures are fallible, try not to let a concept get tied to one and only one method of measurement.

There is not enough space in this paper to lay out in detail the ways in which measures of concepts within the social and behavioral sciences are error-laden, in both reliability and validity senses. Nor is there enough space to argue the case for multiple operationalism, which is a limited,but as yet best available, strategy for gaining confidence in both the precision and the meaning of measures. The reader is referred to the body of literature on reliability and validity of measures, and on internal,external and construct validity of empirical relationships. (See, for example: Campbell & Fiske, 1959; Campbell & Stanley, 1965; Cook and Campbell, 1976; Runkel & McGrath, 1972; and Webb, Campbell, Schwartz and Sechrest, 1966.)

Let me summarize one major thrust of much of that literature. Both reliability and construct validity hinge on the twin ideas of: (a) the convergence of data obtained from different methods of measurement (that is, different operations), that are intended to be measuring the same thing; plus (b) the divergence or differentiation of data from two measures that are intended to be measuring different things. This, in turn, implies that it is necessary to find or develop more than one method for measuring every key construct; and that it is highly desireable that such multiple methods be as different as possible from one another in form, so that they will not share the same weaknesses or fallibilities but rather will offset each other's weaknesses.

Examples of the problem of single-method concepts are ubiquitous in the literature of social science. For example, certain personality characteristics are defined in terms of a score on a certain inventory or scale, and no other operation is considered to be a measure of the same thing. Androgeny is the score on the scale developed by Bem. Authoritarianism is the score on the California F-scale. Machivellianism is the score on the Mach scale. The multiple-operationalism advocated here is certainly honored more often in the breach than in the observance. Moreover, there is at least some reasons to suspect that a very large number of our favorite concepts in the social sciences would probably not survive the test of convergence of multiple measures if they were so tested. But no matter how infrequently the multiple-operation strategy is followed in practice, and no matter how infrequently it yields positive research outcomes, it remains the case that convergence of data from different sources is necessary within the empirical/inductive strategy of the behavioral sciences.

For the MPI area, measurement of the dependent variable--expression of the MPI symptoms--possesses special problems. Nearly always, that measurement must be done retrospectively. Use of "subjective" methods (self-report questionnaires, ratings or interview responses) pose all sorts of problems in any event, especially problems of reactivity. (See Webb et al., 1966, for a sophisiticated treatment of this problem). These problems are aggravated a lot when self report measures are used under conditions where the respondant is likely to be hostile to the research. (See the first section of this paper for a discussion of the poor rapport conditions facing the MPI researcher.) These problems apply whether the researcher then uses the self-report of 1,3, or 37 "symptoms" as the definition of "having" the MPI.

On the other hand, to simply substitute some "objective" indices of MPI, such as records of who received medical treatment for the symptoms on specific days, does not altogether solve the problems. Medical records are far from error-free,

in a clerical-error sense. Moreover, the recording of symptoms is very likely to be influenced, from one case to the next, by the pattern of symptoms that seems to be "going around", so to speak. On top of that, just who does and does not actually get medical attention, under the confused, problematic, ambiguous, and even angry circumstances that often characterize MPI in situ, may be more or less random. Solving the reactive problems of self-reports by substituting an array of data-quality problems is not likely to be an effective tactic. Instead, the MPI researcher must learn to use all the measures he or she can get, and to use them together, to transcend the limits of any one of them.

The use of multiple operations to show convergence of and discrimination between constructs is one approach that can help deal with the problems mentioned in Cautions #2 and #3. One answer to the question of whether things given the same label are in fact the same is whether or not measures of them converge, or covary. One way of finding out whether two things that are known by different labels are in fact the same is by exploring whether measures of them converge or discriminate between them.

Caution #5: Do not let the unfeasibility of doing well-controlled laboratory experiments in the MPI area serve as an excuse for ignoring the logic of experimental design in the broader sense, or as a license to do sloppy research. I'm afraid that this may be one of the most prevalent pitfalls in the social sciences. Researchers who are working in what are thought of as relevant or applied problem areas often construct a strong case for why they cannot do "tight" experiments, in field or laboratory, in these problem areas. Far too often those researchers go on to conclude--or at least to act as if they had concluded--that therefore they and their studies are somehow exempt from concerns about internal validity, external validity, and the logic of inference. They seem to argue as follows: "One can't do really good research in this problem area, since experimentation is ruled out. On the other hand, this is a truly important problem area. Therefore, since poor research is better than none at all, we will just do the best we can, and not worry too much about such exotica of research as the logic of inference, internal validity, statistical significance and all that jazz."

That line of argument is fraught with flaws. Let me point out just a few of them. One is that poor research is not better than none at all. Indeed, it is infinitely worse! Someone might believe it! That is bad enough condition for basic research areas, because it often takes many years to expunge false positives from the literature. But publishing results of poor research is much more serious, even dangerous, in applied areas, because someone might haul off and act on those results!

Second, such matters as construct validity, internal validity, precision and the like, are anything but exotic luxuries for the researcher. They are the guts of the research enterprise. Third, this line of argument seems to postulate two classes of studies: one class of more or less perfect research--experimentation--vs all other, equally imperfect, research. Nonsense!! Experimentation is, by and large, filled with problems; certainly even the best of it is far from perfect research. On the other hand, there are many other forms of research, many shades and variations. Each of them offers some advantages, though all of them have weaknesses. The trick is not to try to plan and carry out the perfect study--it is not possible. Rather, the trick is to do the best we can in each study--while worrying a great deal about validity, precision, and the like; and then to use multiple studies in the same way that we can use multiple measures--to seek convergence across studies on our key constructs and relationships.

Within that broad prescription, and in the context of non-laboratory research on problems such as MPI, it is vital that the researcher understand that much of the strength of any particular study hinges on just what comparisons can be made within it. All knowledge is knowledge of differences. When we proport to have established that a certain phenomenon or relationship holds for a certain body of data, we must make that assertion against the implicit comparative question: Compared to what? This idea includes the idea of baserates and baseline comparisons. It also includes the idea of control groups. Within a research problem of any degree of scope or complexity the idea of "a" control group must be greatly expanded to the more complicated ideas of a range of control and comparison conditions. These ideas, in turn, can be expanded to connect to the much more general ideas of quasi-experimental designs as put forth by Cook and Campbell (1976).

So even if the MPI researcher is not able to run a true experiment, laboratory or otherwise, he or she is still beholden to develop a study plan which takes into account the need for comparison, usually a series of comparisons rather than a single comparison or control group. If the researcher does not do so, then results of the study will really be of very little use. Results will not be able to deal with the question: Compared to what?

The ideas on quasi-experimental designs put forth by Cook and Campbell offer nearly two dozen potential designs. None of them has the logical power of a true experiment, but each of them offers sufficient logical power to allow the researcher to gain some knowledge with some confidence. Not all of these will work for any given problem, especially a problem like MIP which has such special, constraining features. But some of these quasi-experimental designs are likely to be useful for any given problem, even the difficult and constrained

MPI problems. It behooves the MPI researcher to pursue these possibilities. In a highly difficult area such as MPI research, we need all the help we can get!

III. Some Concepts for a Model of MPI

The preceding section dealt with relatively broad and abstract conceptual and methodological issues that seem critical to research on MPI. This section returns to more concrete matters. It starts with a brief summary of some of the main consistent research finding about MPI. That summary is followed by a brief discussion of some of the major theoretical approaches used in this area. Both of these rely very heavily upon material from the three references noted at the outset, especially the Colligan and Murphy (1979) review. The discussion of theoretical approaches makes it clear that there are at least two underlying constructs that are implicated in almost all of them: differential exposure and differential susceptibility. The third part of this section is an analysis of these two concepts. The fourth part of this section is an attempt to integrate these two concepts with the main substantive findings. This section and this paper ends with an attempt to integrate all of this into a coherent formulation.

Sex, Stress and Somatization: A Summary of Some Consistent Findings

Sex differences. The most consistent single finding in the literature of MPI is that females "catch" such "diseases" far more frequently than males. On the other hand, it is clear that the kinds of settings in which these outbreaks occur tend to have almost all females at the "assembly line" level of workers. It is not entirely clear whether the very high percentages of affecteds who are female (perhaps 80% to 90% or more) is really higher than the baserate of females in those or comparable settings. So the overall frequency of female victims of MPI can be interpreted as meaning either: (a) that more females than males are victims of MPI's; or (b) that MPI outbreaks tend to occur in those settings where most of the workers (or students) are females. Both formulations leave some unanswered baserate questions.

For the most part, researchers who have noticed and discussed this high incidence of females in MPI have tended to assume the first of the two statements above, and have tried to account for why females are victims of MPI more often than males. In fact, the very term "hysteria" is a label for the disorder that predicts, and at the same time "explains", the disorder as one that only females will suffer. It is said that Hippocrates used the word hysteria because he thought it was caused by the uterus, literally, wandering in search of semen. Most current researchers in this area would reject

such an explanation, and indeed would probably reject the use of such a sexist term as hysteria. Perhaps part of the reason why the finding that most victims are female is given remarkably little discussion in the MPI literature is because many researchers, in this still male-dominated area of behavioral science, want to avoid seeming to be sexist.

The attempts to "account" for the high female frequency are most often built upon notions of learned differences in sex roles. At least two such explanations are offered by Colligan and Murphy (1979). One starts with the idea that females in the work setting are in conflict. They have major role responsibilities in the home in relation to family and household. They are uncertain of their role in the work place. Thus, they are under role stress, and the MPI is an expression of that stress. But if this were so, then we would expect to find that single women, and/or women with more feminist views, would be less susceptible to MPI. We should also expect to find that men who have such dual role responsibilities (male heads of single parent families) would therefore be more susceptible to MPI than other men. Such comparisons have apparently not been made; and Colligan and Murphy (1979), appropriately, call for research on this question.

An alternative explanation is based on differences in the content of traditional sex roles. The traditional female sex role, into which most adult females have been socialized, is one that emphasized expressive (as opposed to instrumental) behavior. In this explanation, the MPI is to be viewed as an expression of (that is, a somatization of) job stress. Males, because of their sex role training, are more apt to externalize (that is, blame the work-conditions, the boss, etc.). This explanation has several problems. First, while there is considerable evidence that the victims of MPI are more likely to express stress in the form of bodily symptoms (that is, somatization; see below), there is no convincing evidence that women somaticize more than men in a general sense. Second, in most usages of the dichotomy "expressive" vs "instrumental", the term expressive <u>does not</u> particularly refer to expressing things in the form of bodily symptoms. Nor does the term instrumental connote <u>projection</u> of stress or symptoms onto environmental targets. Moreover, if this differential sex-role explanation were sound, then it ought to be possible to find a subset of females and males who are not socialized into those alleged traditional roles (e.g., females or males who have high scores on Bem's androgyny scale.). Such a test does not seem to have been made.

The second interpretation--that MPIs tend to happen in situations where most of the workers (or students) are female--deserves a comment. Some other factors related to incidence are pertinant here. MPI seem to happen to the relatively

young, to persons in relatively menial jobs, to persons in relatively unpleasant settings (see later material on job and environmental stress), and in settings where communication to peers and to superordinates is limited or difficult. Perhaps the high incidence of females, as both affected and non-affected members of the settings in which MPI occur, simply reflects our tendency to put females in low status and otherwise unrewarding jobs. If so, then "female," along with "young" and "poor", should be treated as a variable that signals "low social status", not some character flaw like "hysteria" or "hypochondria".

Perhaps the MPI symptoms represent a "last resort" means for low-status persons (female, young, poor, etc.), in high stress situations, to express their distress.

Personality. Considerable effort has been devoted to trying to find "the" personality traits that differentiate victims of MPI from those in the same situations who do not fall victim. As has so often been the case with efforts to find personality traits that predict behavior, in many other problem areas, results here have been weak, contradictory and not very helpful. The two most frequent candidates for such differential personality traits (apparently for reasons of convenience, rather than for strong conceptual reasons) have been neurotocism and extroversion. Colligan and Murphy (1979) summarize results of many of these efforts by noting that some studies show MPI victims to be high on both, some show them to be high on one but not on the other, some studies find no differences on either of them. In other words, the findings are neither consistent nor replicable.

There are some sound logical reasons why we should not expect such "traits" of the individual to be very strong correlates of displaying or not displaying MPI. Those reasons involve the obvious interactions of "who"with "where" and "when" in these MPI outbreaks. So, for both conceptual and empirical reasons, we ought to expect little help in understanding MPI from the personality-trait approach.

Illness related behaviors: Somatization. One relatively consistent set of findings that bear on the question of why particular individuals, and not others, fall victim to MPI, indicates that the "affected" also show higher rates of absenteeism, more sick leave days, and more medical consultations. That constellation of indicators could be signalling either of three things about victims of MPI. First, it could mean that they are more sickly, in some general sense, and hence are more likely to fall victim to the MPI (or any other) symptoms. Second, it could mean that they are more hypochondriacal, in general, and hence are more likely to imagine that they have the MPI symptoms. Third, it could mean that they are not more sickly or hypochondriacal than

others, in general, but that given high stress they are more apt to use somatization--expressing stresses in the form of bodily symptoms--as a preferred coping style. The small amount of evidence available seems to refute the first and support the third(and perhaps the second) of these explanations (Colligan & Murphy 1979). It seems reasonable to hypothesize--though tentatively, to be sure--that the occurrence of MPI is the somatic expression of stresses in the situation, and that those who become MPI victims are those who use somatic expression as a preferred mode for coping with such stresses. To refer back to an earlier section, it is somatization that sorts out victims vs. non-victims of MPI, whether or not that somatization is connected to female sex role socialization or sex role conflict.

Situational factors: Stress. Besides the efforts to find bases for predicting who will and who will not fall victim to an MPI in a situation where it does occur, some effort has been given to trying to find out why MPI occurs, where and when they do (that is, why in this situation, and why at this time). For one thing, there is almost always a triggering event, or an index case. The triggering event could be the company's change in production process, or in work schedules, or in the solvent it uses, or the like. An index case is the occurrence of a case of "actual" illness--that is, one that medical personnel diagnose as caused by a germ, a toxic agent or some other denotable pathogen. The MPI consists of later "cases" that (a) mirror the index case in symptoms, or offer symptoms that are reasonably compatible with effects that the triggering event might cause; and (b) are attributed by victims to the triggering event or to the same pathogen that caused the index case; but for which (c) the medical personnel involved do not accept that diagnostic attribution.

It also turns out that, in most of the MPI occurring in industrial settings in recent years, there is evidence of considerable negative job and environmental conditions. There are often reports of excessive noise, noxious odors, poor lighting, variations in temperature. The settings frequently involve extremely boring, repetitive tasks. In a high proportion of cases, there is evidence of poor labor-management relations: worker-supervisor friction, ambiguous supervision, heavy production pressures, and poor conditions for worker-to-worker communications.

Such results suggest a situational-stress interpretation of the phenomenon. Perhaps we can regard the relatively bad conditions of environment (noise, odors, etc.), of job (boring, repetitive, work production pressures) and of interpersonal relations (supervisor friction, poor worker communication, etc.) as representing a high level of stress in the situation. If we act as if these are substitutable

one for the other, then we don't have to show that any one of them is present in all cases, only that "a bunch" of them are operating in each case. But presumeably such conditions are present and having an effect on all workers in the situation, but only some of them become victims of the MPI. There is also the problem that such conditions are probably operating, at comparably high levels, in lots of other industrial settings, but that most of those settings do not spawn MPI. Finally, there is the problem that this setting has probably had such bad conditions operating for quite a long time, and MPI have not occurred before. So stress appears to be a necessary part of the explanation of MPI, but not by any means the whole explanation.

Summary. These considerations point to some key questions and comparisons, already noted in the first section. We need to be able to specify some of the features that distinguish this situation and time from others like it. We need to understand the role of triggering events or index cases in "unleashing" certain responses, or coping styles. We need to identify characteristics on which the population of victims are different from non-victims--and different from them in ways that are related to the relevant situational conditions and triggering event(s). In other words, the crucial features of MPI may be first or second order interactions, of person, situation, and temporal circumstances.

The major consistent findings seem to support three constructs, as suggested in the title of this part: sex (actually gender differences); stress (in terms of job, environmental and interpersonal conditions); and somatization (that is, the coping style preference for reacting to stress in terms of bodily symptoms). These represent a reasonable beginning, but by no means a complete set of substantive concepts. Nor do they answer some crucial theoretical questions, to which we now turn.

Some Theoretical Approaches to MPI

Some of the "theoretical" discussions of MPI place heavy reliance on psychoanalytic concepts. Others rely more on epidemiological and social psychological ideas. The latter include convergence, contagion, and emergent norms theories. Colligan and Murphy (1979) offer a useful discussion of some of these, and the following material relies upon that discussion. (See, also, Kerckhoff, Bach & Miller, 1965; Milgram and Toch, 1969.)

Psychoanalytic approaches. These interpret the manifestation of MPI symptoms as reflecting some deeper, unconscious psychological problems of those individuals. From such a point of view, the particular symptoms displayed are relatively

unimportant. If they didn't exhibit these symptoms, they would exhibit some others instead. To setting managers, on the other hand, and to the victims, the symptoms are the problem, and getting rid of the symptoms is the goal.

Basically, the psychoanalytic approaches are neither verifiable as theory nor useful as guides to interventions. They hinge on interpretation of all responses as manifestations of inner conflicts. Since such underlying conflicts are only known when manifested as "symptoms", and since all behaviors are symptoms of some underlying problems, the psychoanalytic theorist, in principle, has no conceptual tools to anticipate which persons will use MPI symptoms and which will use other "symptoms" to express their (underlying and unknowable) problems. Nor does the psychoanalytic theorist really care which sets of symptoms the individual uses (i.e., MPI or other). Rather, that theorist is concerned with ferreting out the underlying problems--the genotypic problems, for which MPI and other symptoms are phenotypic expressions. So, the psychoanalytic theorist has neither interest in nor tools to solve the MPI problem.

Convergence. The central idea of convergence is that a bunch of individuals all are exposed to the same index event, and are all affected by it independently of one another. The people who are affected are somehow different than others, who either were not exposed to the same sources, or were not affected by that exposure. Temporal differences in exposure are more or less unimportant variations in the relation between being exposed and being affected.

One of the psychoanalytic approaches can be viewed as a convergence theory. In it, the victims identify with the index case, and thus succumb to the same illness; whereas non-victims do not identify with that particular person. Evidence supporting the idea that the index case is somehow notable, and therefore to be identified with by a number of people, is rather weak.

Contagion. The basic idea of contagion is that A, the index case, somehow transmits to B, who in turn transmits to C(and, of course, perhaps to more than one other person) and that this occurs in sequence. In this approach, the time effects are not trivial, but reflect order-or-exposure. They thus tell us a lot about the process of contagion, and about how the"disease" sweeps through a group. In the contagion approach, who gets the symptoms is not a function of their relation to the index case, but rather a function of their relation to one another in both physical and social senses. We would expect job and social isolates to be least affected, task and social "stars" to be most affected.

The evidence supporting this is again weak, especially if we

insist that any one MPI must be either contagion or convergence. Actually, Colligan and Murphy (1979) make a good case for the idea that convergence may fit the early part, contagion the latter part of an MPI. Kerckhoff et al. (1965) and Milgram and Toch (1966) also support that idea. At first, it is people who "identify with" or "imitate" the index case, but who are not especially related to one another, who "get" the MPI. Then, "it"gets into and spreads through social networks, in a contagion manner.

Both of these raise the question: why does the MPI not sweep through the entire population? Perhaps one answer is that some people are in social networks that never do get "infected". Even though for some people the spread of the disease is facilitated by their ties in social networks, for others a particular network acts as a defense against the MPI.

Emergent norm. The basic idea here is that an emergent norm develops in the group, such that getting and expressing the symptoms becomes an acceptable way of dealing with the considerable stress in the situation. (See Turner, 1964; Turner & Killian, 1957.) This can be seen as a restatement, in social psychological terms, of the more epidemiological convergence and contagion theories. Convergence could occur in an early stage before the norm exists; with contagion occurring as the basis for spread of the norm after the norm gains strength. The epidemic ceases short of a 100% victim rate because some social networks may never accept the emergent norm, thus serving as a defense against contagion.

Summary. The convergence, contagion and emergent norm approaches probably should be taken as complementary rather than as competing theories. Together they reflect a "flow" model, with different conditions affecting rate, direction and cessation of flow. There are at least two concepts that seems to be at the core of all of these approaches: the idea of differential exposure of individuals; and the idea of differential susceptibility given exposure. The next part of this section tries to analyze these two concepts as a step toward a model of the MPI problem.

Two Core Concepts for a Model of MPI

Both the main consistent findings (sex, stress and somatization) and the main contending theoretical views (Convergence, contagion, and emergent norms) all suggest that any viable model of the MPI problem will need to include at least two underlying concepts: differential exposure and differential susceptibility. The concept of exposure is basic. The whole idea of an epidemic, or contagion, implies that something is transmitted to a victim by contact. In the general case, either a toxic substance is transmitted from an environmental source to a victim, or a virus or bacterium is transmitted from one

victim to another.

In the case of an MPI, it is not a pathogenic agent, but rather information, that is transmitted. It is a relatively complex set of information, too. An MPI victim must have been "exposed" to:

(a) the fact of the occurrence of the index case, or the triggering event;
(b) the fact that others have fallen victim to the illness;
(c) the symptoms involved in the illness.

First, the idea that the MPI case involves exposure to information, has several implications for MPI research. First, exposure implies contact, but not necessarily physical contact with either a person or an environmental condition. Furthermore, "contact" and transmission of information can occur one-to-many as readily as one-to-one. Therefore, the meaning of time intervals and sequences in the spread of "contagion" becomes less clear cut. The distinction between convergence and contagion, which partly involves time, becomes blurred. In addition, since the "ingredient" transmitted by exposure is a complex set of information, the exposure in MPI must be regarded as a fairly complex set of conscious cognitive events.

The concept of exposure in a "contagious" situation implies that exposure and its effects are not uniform for all members of the population. If no one was exposed, there would be no "epidemic", If everyone were exposed, then either everyone would become victims (which is usually not the case), or an additional concept--differential susceptibility--would be needed to account for why some but not other persons contracted the illness.

Therefore, we can think of a two stage process. One is the exposure link representing contact between the source and the person. The other is the susceptibility link, representing the relation between the exposed person and the actual exhibiting of MPI symptoms. Each of these links depends on features of the situation and of the person. For any person in the situation, we can consider that he or she has two "vulnerabilities". One is his or her vulnerability of being "exposed" to the "contagious information". The other is his or her vulnerability to contracting the symptoms, given exposure. Each of these links merits some analysis.

Vulnerability to exposure. There are a number of possible models of exposure. One is the idea that contact with a contaminant is random. Some people propose this view of who dies in combat. They say such things as "if the bullet has your name on it...", which presupposes that the probability of being hit with a bullet is random, at least with respect

to the victims actions. But a random model is probably not very viable for MPI. It certainly is not for combat.

If we posit a non-random model of who gets exposed we need to consider not only what substantive features predict differential exposure (sex, age, centrality in a social network, etc.). We also need to consider such things as dosage level, frequency of exposure and the like. Put another way: Does each contact have some probability of contaminating the person, with "contamination" being all or none(as in "catching" a germ) ? Or, instead, does each contact represent an increment of contamination of the person (as in exposure to radiation)? If the latter, then we can pose two more questions:

(a) Is there a "threshold", above which the MPI symptoms occur and below which they don't? Or, instead, is the intensity of the symptoms a monotonic function of accumulated increments of exposure?

(b) Does time mitigate effects of small amounts of exposure--either in a time-decay sense, where dosage rates need to be calculated "per unit time", or in the innoculation sense, where small amounts provide a kind of immunization against later larger exposures?

Keep in mind that the "contaminating ingredient", in MPI, is a complex set of information, not a germ of a gamma particle. So when we talk about contact-contamination, we are talking about:

(a) differential probabilities of different people in a situation being exposed to that critical set of information;
(b) the probabilities that an exposure will be "received" psychologically;
(c) the effects of multiple exposures, including their incremental accumulation beyond some threshold and/or their potential innoculation effects.

When put in these terms, we are clearly talking about matters quite closely related to the whole communication, persuasion, and attitude change areas, for which there is a huge literature and a formidable body of theory. MPI research does not seem to have drawn upon that body of literature nearly as much as one would expect, given this analysis of the problem.

Vulnerability to display of the symptoms. Let us now consider what factors influence who exhibits the MPI symptoms, given exposure to the critical information at above-threshhold levels. This is the question of differential susceptibility. Whereas exposure has to do with acquisition of information, susceptibility has to do with acceptance of the

impact of that information and consequent behavior change.

The notion of susceptibility suggests its converse, resistance. We need to consider vulnerability as a continuum from highly susceptible to totally immune. A person who shows the symptoms is above some threshold on that continuum. What factors affect that person's vulnerability level? and what factors affect the threshold level, beyond which the MPI symptoms are displayed?

We have already considered some ways in which "dosage" levels and rates might serve either to increase cummulative dosages, thus adding to susceptibility, or to provide a kind of innoculation, thus reducing susceptibility. The communication-persuasion and attitude change research on amount of information, and innoculation effects, should provide useful guides here. (See, for example, McGuire, 1969).

There are other potential contingencies between exposure and susceptibility, too, and our MPI model should reflect them. For example, we could posit a model in which more exposure (i.e., more frequent, more intense, more sources of contamination, longer durations of contact) implies greater intensity of "dosage", hence greater susceptibility (i e., greater probability of showing the MPI symptoms). This would be a positive interaction between:

(a) amount of exposure, and
(b) level of susceptibility of the individual.

We could, instead, posit a model that has a positive interaction between:

(a) the number of others already affected (this could be considered intensity of exposure to the contaminating information) on the one hand; and
(b) level of susceptibility, given exposure, on the other hand.

In such a model, the more susceptible persons would become MPI victims first; then as more and more persons did so, individuals with more and more resistance would "contact" the symptoms. Such a model would have trouble accounting for why the MPI didn't eventually produce 100% victims. It would need either some basis for showing that as-yet-unaffected persons did not have sufficient exposure, or else some basis for showing that the unaffected had such high resistance that even the increased intensity, when all others had become affected, did not exceed their threshold.

Substantive Variables in Relation to Vulnerability

What substantive variables relate to exposure and susceptibility,

and how do they tie into the network of variables involved in these potential models of vulnerability? In particular, how might the main consistent findings--about sex, stress, and somatization--fit into these models?

Sex differences. The consistent finding that victims of MPI are mostly females could be viewed in several ways. It is possible that females have higher susceptibilities, either to all illnesses, or to "hysteria" type illnesses, or to "imaginary" illnesses. Such differences could arise from either biological/genetic bases or experiencial/environmental bases. This is possible; but I don't think it is very likely; nor do I think there is any evidence to support it.

Alternatively, females may be more vulnerable to exposure. They may gather and pay attention to rumors and gossip more than males; they may attend to broadcast media more than males. Again, I think this is unlikely on both theoretical and evidential grounds. (Even though there has long been a sterotyped notion that women gossip more than men, that is mainly supported by male definitions of their own conversations as important and therefore "business", and of women's conversations as unimportant and therefore "gossip".)

Colligan and Murphy's (1979) attempts to account for sex differences on the basis of different sex role training implies that females are socialized more than males to express stress in somatic terms. So, given exposure (presumeably not sex dependent), and given high levels of stress (which may or may not vary by sex, as discussed below), females are more susceptible to expressing their stress in physical symptoms (and therefore are more likely to exhibit MPI symptoms) than males, who presumably deal with their stress in other ways(such as ulcers and coronary disease).

Colligan and Murphy's (1979) other explanation for the sex differences posits more sex-role conflict for the woman in the workplace. Here the hypothesis does not propose increased susceptibility of females to MPI symptoms given high stress and exposure. Rather, it hypothesizes that some females will have higher levels of stress because of role conflict. (See further discussion of stress below.)

None of the material I have covered has suggested that females may differ in how they acquire and process information. It would be well worth the MPI researcher's time to search for information on male-female differences both in the communication/persuasion and attitude-change literatures, and in the literature of the currently booming areas of impression formation and cognitive processing. As in so many other areas of social psychology, past research has tended to ignore sex differences. Sometimes there has even been a tendency to sweep them under the rug, on the grounds that they were not

"theoretically interesting". So that search might be long and tedious; but it might pay off well.

Stress. There are several ways in which the stressful features of MPI situations might affect both exposure and susceptibility. For one thing, total intensity of stress on the person might act so as to sensitize the individual to possible "explanations". He or she might seek out information, and accept it more readily. On the other hand, stress may tend to narrow the focus of attention and reduce the individual's ability to take in and process information (See, for example: Appley & Trumbull, 1967; McGrath, 1970). The stressed person might become more sensitive to ways of coping with that stress. This could translate into higher susceptibility to MPI symptoms if that were a preferred coping style. Such a relationship would imply, however, that other people in the same high stress situations ought to show an increased use of other coping styles if they don't use the MPI symptoms. There does not seem to have been any attempts to test such a hypothesis. Such research surely seems worth doing.

Colligan and Murphy (1979) suggest that the stress stemming from poor worker-supervisor relations, and from poor conditions for worker-to-worker communication, deprive individuals of both normative and informational support that might counter the MPI-inducing information. Such poor communications do not reduce the person's exposure to the MPI-inducing batch of information (about the index case, later cases, and the symptoms) because these can flow through many channels including extra-setting mass media. But these poor communication conditions reduce the flow and the credibility of information that might offset the MPI. So the stress of poor communications may prevent reduction of vulnerability but not prevent exposure.

Somatization. One of the most interesting findings regarding MPI is that victims are persons who also show higher rates of absenteeism, sick leave days and medical consultations. Colligan and Murphy (1979) reviewed evidence indicating that this was probably not due to their being in poorer health in general. Their review leads them to suggest that somatization is a preferred coping strategy for MPI victims. Assuming this to be the case, then a preference for expressing stress in terms of physical symptoms can be considered an individual characteristic that increases susceptibility--given exposure and given high stress. Presumably this characteristic does not affect exposure, except in the limited and indirect sense that frequent absences might reduce communication flow to that person within the setting.

An Attempt to Integrate

It seems appropriate to try to integrate all of this--the com-

plexities, the cautions, the concepts--into a coherent formulation. It is probably appropriate to eskew the formal language of "real" science--assumptions, postulates, hypotheses, and all that--and offer a formulation stated as a series of questions and some plausible answers to them.

1. How should we regard an MPI?

The most parsimonious formulation, it seems to me, is to regard an MPI as a reaction to high levels of stress--as one of a number of possible alternative forms by which the individual may cope with stress.

2. Where and when do MPI happen?

MPI seems to occur only in work situations that have some or all of the following negative features:

a. Surrounding conditions of the physical environment (e.g., noise, odors, temperature) are at undesirable levels.

b. The job itself has few positive-motivating factors. It is monotonous; it is low pay; there is much production pressure.

c. Conditions of the social environment are at unfavorable levels. There is poor communication, and often friction, between workers and management. There is poor communication among workers, both because of physical impediments (e.g., noise) and because of other job features (e.g., assigned timing of work breaks).

In other words, MPI occur in situations characterized by high levels of stress from the physical task, and/or social environment of the work place.

3. Who are affected by MPI?

Apparently, MPI occur only for low status persons in the work situation. They never involve foreman or supervisors, much less high level managers. They always involve, exclusively, workers at the "production line" level (but, of course, not all of them). It should be noted that low status persons in a situation are probably under more stress than higher status persons in the same situations. They certainly have far fewer ways to cope with that stress.

4. When is MPI likely to be used, rather than some other means for coping with stress?

One of the ways a person can cope with high stress is by expressing bodily symptoms. It is probably that doing so is

a (relatively) preferred coping style for some individuals in any case. Those individuals, "somaticizers", are likely to be among the MPI victims if one occurs in a given situation.

But as stress increases, the array of possible coping options available to an individual tends to decrease. Similarly, as one's status in the situation is lower, one probably has fewer coping options. As the number of available coping options decreases, for whatever reasons, the probablility of use of any remaining option increases, regardless of whether or not that option was originally a preferred coping style. Many coping options (quit the job; hit the boss; get drunk) tend to be eliminated as practical possibilities for individuals who are low in status and under high stress. But MPI-- that is, the expression of stress in the form of somatic symptoms-- remains always within the individual's response repertoire. Therefore, MPI will remain as a coping option as stress increases and other options slip away. It will increasingly and inevitably become the coping method of choice even if it was not a preferred method at the start.

5. When is a particular triggering event or index case likely to be modelled for an MPI?

A particular triggering event or index case is likely to serve as a "model" for an MPI if it is noticeable, rational, and low-cost. Triggering events are often noticeable because they represent changes in the work situation (e.g., a new work method, a new set of materials). Index cases are often dramatic because they involve dreaded diseases (e.g., polio). By "rational", I mean that the index case, the symptoms set and the causal premise go together, are a "good fit", are a plausible combination of cause and effect, from the point of view of the victims (who believe that some toxic agent or pathogen is involved), regardless of whether they are considered plausible from the point of view of setting managers and medical practitioners (who believe that no toxic agent or pathogen is involved). The symptom set and alleged cause for industrial MPI do, indeed, seem to fit this definition of rational, without exception. Indeed, it is precisely because the symptom-cause package of an MPI is highly plausible that it is so difficult to establish whether a given "outbreak" is an MPI rather than the result of chemical or biological agents, as discussed in the first section of this paper.

It also seems reasonable to suppose that a particular triggering event will be modelled as an MPI to the extent that it offers a(relatively) low cost means for coping with the stress. Consider what an MPI provides as a means for coping with high levels of stress in the work situation:

(a) It can get the person out of the stressful situation, at least temporarily.

(b) It can do so with no "blame to the individual, and perhaps with little or no cost in lost wages, provided the "triggering event theory" of the cause is maintained.

(c) Moreover, it may lay some degree of blame on management, because most MPI in industrial settings involve alleged causal agents for which management bears at least some responsibility (e.g., a new solvent, a malfunctioning ventilating system).

(d) Finally, expression of the MPI can represent evidence of one's empathy and solidarity with one's peers.

Compared with hitting the boss (or the spouse), quitting the job, getting drunk, going over the boss's head, or getting an ulcer, expressing stress via bodily symptoms in relation to a dramatic and rational MPI triggering event certainly seems to offer a reward - cost net advantage! Perhaps we ought to ask, seriously, why MPI do not occur more often, and "infect" a far larger proportion of the work force when they do occur!

6. Why do MPI appear to be almost exclusively a "woman's disease"?

Probably for several reasons, but the most parsimonious formulation seems to me to be as follows: Women are low status in the work place. (So are the poor, the uneducated, the relatively young. So we would expect high MPI rates for them, too, and that seems to be the case.) Not only are women low status in any particular job setting, we would expect a very high proportion of women (and other low status categories, like poor and uneducated) to be in the "production line" jobs of the highly undesireable kinds of work settings in which MPI occur. So, we would expect a very high proportion of women (and other low status persons) both as victims and as non-victims in the settings where MPI occur--and that indeed is what we find.

Since the higher the stress the fewer the coping options, (and the more probable any remaining one such as MPI); and since the lower the status the higher the stress and/or the fewer the coping options, (hence the more probable any remaining one, such as MPI); it follows that persons who are low status in highly stressful situations--such as women in the highly stressful work settings characteristic of MPI--are more likely to use somatic symptoms (MPI) as a means for coping with that stress.

Note that this formulation "accounts for" the high proportion of women who are not affected in MPI settings, as well as

the high proportion of the affected who are women. None of the other interpretations does so. Note, also, that the formulation presented here does not call upon "personality" constructs, nor differential somatization tendencies, nor differential sex role training. It offers a sufficient explanation in terms of stress, status and coping. Other explanations are not ruled out, of course, either as alternatives or, more likely, as complements. For example, it is probable that women have certain coping options denied to them via cultural training; or, rather, it is probable that men are trained in certain coping options not made accessible to women (e.g. hit the boss; break the machine; drive recklessly on the way home from work; swear). Some of the evidence reported by Lazarus and by Folkman, in other papers of this conference, would suggest that the latter may be the case. Furthermore, as was suggested earlier in this paper, men and women may very well differ in how they acquire and process information; and contagion for an MPI is, after all, the acquisition and processing of information, not toxins. There may also be other MPI-relevant differences between men and women, by virtue of differential socialization (e.g. differential somatization preferences), or even by virtue of physiological or hormonal differences (e.g., another participant at this conference suggested synchronization of menstrual cycles). But I think each of these is, in turn, increasingly less likely on both theoretical and evidential grounds; and they certainly seem to be increasingly less parsimonious.

So, as an overall summary and concluding statement let me offer the following: MPI seem to be the response of some relatively low status persons, in high stress work situations, subsequent to triggering events that are dramatic and that provide a rational and low-cost option for coping with that stress. Most MPI victims are women. But most non-victims are also women, and the vast majority of women do not become MPI victims even in situations whose MPI occur. Women are far more often to be found in low status jobs in high stress settings, where MPI occur. So, while MPI victims are inevitably mostly women, MPI non-victims are also inevitably mostly women. What may be remarkable and worth study is that, in MPI type situations, most women cope with the high stress by methods other than MPI.

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THEORIES OF CONTAGION AS THEY RELATE TO MASS PSYCHOGENIC ILLNESS

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ABSTRACT

This paper presents a review of the theories regarding contagion. It discusses the original ideas of Le Bon and Freud that being in a crowd releases primitive impulses and the modern reworking of these ideas in terms of deindividuation. The paper also deals with the theory that contagion is essentially imitation, mediated by restraint release due to observing another perform an action which the individual is in conflict about performing himself. The final major idea is that the amount of space available (density) as well as the number of people who are present affect the likelihood of contagion occurring. In addition, contagion is distinguished from phenomena such as conformity and the emergence of group norms. The various theories are criticized with particular reference to mass psychogenic illness. It is concluded that the theories may provide a framework for considering incidents of mass illness, but that none of them alone nor all of them together offer a full explanation of the phenomenon.

Mass illness due to psychological causes appears to be a classic instance of a phenomenon that has been called contagion. In 1895 Le Bon suggested that in a mob all of the people tend to feel and behave the same way because the emotions and actions of one person spread through the group - a process he termed "contagion". Most of his observations were based on the actions of large groups of people engaged in violent or other anti-social behavior, though it should be noted that he also realized that more positive or prosocial behaviors might also be involved in contagion. He cited the mob's storming of the Bastille during the French Revolution as one instance of contagion which he considered deplorable (though obviously others would view it in a different light).

Whether the actions were positive, negative or neutral, the critical element for Le Bon was that in a mob the people behaved in ways that they would not have if they were alone, and that by themselves each person might act differently, while in the group they all felt and acted the same. This is probably an overstatement of the situation. It is rare that every individual feels and acts identically just because they are in a group. However, the basic idea is sound. There are clearly times when people in a group engage in some activity, or in the present case, exhibit certain symptoms, that few,if any, would by themselves. However, in order for this to be an instance of contagion, it must follow a particular pattern. Namely, one or at most a small number of people in the group must first exhibit the behavior, and then this must spread through the group so that others also exhibit it.

It is important to distinguish this process from others that can also cause the members of a group to behave similarly. First, it may be that there are sufficient reasons for the people to behave in a particular way, and these reasons affect everyone at the same time. For example, if a fire breaks out in a factory and everyone reacts by becoming frightened and running from the building, it would ordinarily not be considered contagion. Rather, everyone is merely reacting in more or less the same manner to a clearly discernible external event. More to the point, if an instance of mass illness can be shown to be due to a gas leak, bacterial contamination, or sudden overwhelming stress, we would not consider these examples of psychogenic illness nor of contagion. Thus, in investigating mass illness, the first focus is always on whether some physical cause exists, and the outbreak then has little psychological or sociological interest unless people have greatly overreacted to the situation.

A second process that may be more difficult to distinguish from contagion is usually called conformity. The individual does what other members of the group do because of social pressure. In the typical instance of conformity, everyone in a group acts in one way and a new member or someone who

has not yet had the opportunity to perform the behavior, also acts that way. Note that in this case, most or all of the group is already performing the act, and it is this that puts pressure on others. In contrast, contagion occurs when only one person or at most a small percentage of the group perform the action and the rest go along with it. In other words, it is quite different from conformity in that in contagion a small number affect the majority, while in conformity, it is the majority that affects the rest. However, it is possible that conformity could occur when one especially outstanding or influential member of a group behaves in some manner, and the rest go along because of his position. That is, under some circumstances the norm might be established not by the majority - but by a small percentage of the group who happen to be particularly infuential.

This leads to a third process that has been called emergent norms (Turner, 1964). According to this idea, there are times when everyone ends up behaving the same because the group has established a norm for which a particular form of behavior is both acceptable and desirable. The group need not get together and have a vote on this. Rather, and more typically, the norm emerges gradually and each member identifies it for himself. This occurs because the members observe what others are doing, decide for themselves what seems to be appropriate under the circumstances, decide further than everyone agrees on what is appropriate, and then behave accordingly. If they decide that the group is storming the Bastille, they storm the Bastille; if they decide that the group is running into a burning building to save someone, they do that; and if they decide that the group is having headaches or laughing fits, they do that. In other words, these are instances of conformity to group norms. Each individual is exposed to these norms, is under pressure to go along with them,and thus we get similar behavior from the group members. Although this obviously occurs under some circumstances, it seems not to be applicable to most instances of mass illness. The notion that there is a norm to become seriously ill is highly implausible, and this theory does not explain how such a norm might develop in the first place. Surely, one or two people exhibiting physical symptoms would not ordinarily cause anyone to think that this was the norm. Thus, although emergent norm theory may account for the latter stages of contagion, when a large number of members are already exhibiting the behavior, it is not helpful in explaining why contagion occurs in the first place.

Let us be clear then that contagion occurs when, and only when, the behavior of one individual or at most a few individuals is observed by others, and these others then perform the behavior. Moreover, in order for it to be contagion, the following criteria must also be met: 1) there is no sufficient reason for the people to be behaving in this manner apart from the

fact that others are; 2) closely related to the first, those who observe the others would be unlikely to perform the behavior by themselves - that is, watching the others behave increases the likelihood of the behavior occurring; 3) it is not merely a case of members of a group imitating or being otherwise influenced by an especially powerful or popular individual, though such factors may play a role (this is to distinguish contagion from instances where leadership is involved, such as when an inflential member says "Lets all go swimming" or merely goes off to swim - and everyone follows.) Finally, it is perhaps important to note that in most instances of contagion, maybe all, the behavior in question involves some conflict. The individual may have some reason to perform it, but also some reason not to - he is, in traditional psychological terminology, in an approach-avoidance conflict. For example, the Frenchman who storms the Bastille probably has reasons for wanting to destroy this symbol of the government, but is also afraid of the consequences and may feel some hesitiation due to moral restraints against breaking the law. Similarly, people who stand in approval at the end of a piano concert want to show how much they enjoyed the performance, but each person is also reluctant to be the first to stand from fear of embarrassment (if no one follows suit) or simply from not wanting to be too conspicuous. In the case of psychogenic mass illness, the reasons for displaying the symptoms are less obvious, but according to the case studies, there always seem to be some particular stress present or other causes; while the pressures against displaying the behavior are clearer, involving social norms, needs of work and so on.

Taking these as criteria for contagion, it seems that mass illness typically satisfies them quite well. First, in all of the instances that have been described in any detail, the illness appears in a small number of individuals, and then many others exhibit the symptoms. That is, the illness does not break out in a great many people simultaneously - rather it starts with a few and "spreads". Second, there is no known physical cause for the illness, nor are there any sufficient psychological causes such as stress. Often, of course, the individuals are under considerable stress, but they have been for some time before the outbreak and are again after the outbreak. Thus, the stress is not enough to cause the illness by itself - people can experience this level of stress (or other) pressures) without showing the particular symptoms of the illness; and indeed, the very individuals who become "ill" have experienced identical conditions before without becoming sick. Third, as far as we can tell, the outbreaks are not organized by any person or persons, nor are they due to people following a particularly influential person who wants others to copy him or her. Instead, a few people get sick, they do not try to make others get sick also, and the others are not consciously imitating them. Those

involved appear to believe that they are sick; they are not deliberately exhibiting the symptoms. There may be other ways of considering psychogenic illness, but clearly it is one of the most dramatic instances of contagion. Therefore, theoretical explanations of contagion may help us understand the causes of mass psychogenic illness.

The earliest explanation of contagion was that being in a group in some way released primitive impulses by suppressing the individual's normal restraints. Both Le Bon (1895) and in somewhat different terms Freud (1923) subscribed to this view. The idea was that the usual moral sense and system of controls that we each carry around with us depends on feeling to some extent responsible for our own actions. When we are no longer responsible for ourselves, when we can blame others for what we do, or when for some reason others take responsibility for our behavior, our control system is weakened or even breaks down entirely. The superego, which ordinarily specifies what actions are allowable, becomes irrelevant when we feel that behavior is either beyond our control (we are forced to do it) or when no one, including ourselves, could blame us for what we do (one cannot feel guilty if the action is not freely performed, or when someone says that they take full responsibility - it is their guilt, not ours). Being in a group makes us feel less responsible for our actions. Instead of acting as individuals, we act as a group, and the responsibility is shared. Moreover, the group comes to act as a unit, submerging each individual's freedom of action and moral sense in the "group mind". Thus, not only is the individual less responsible because of shared responsibility, but he is carried along by the group mind. Moreover, and perhaps most important, once the superego is submerged or weakened, the more primitive impulses of the id (in Freudian terms) are released. They have been held in check by the superego and are now free to be expressed more fully. Thus, aggressive, destructive and sexual impulses, that are usually carefully restrained, are more likely to be expressed in groups.

Although this is a fascinating idea, there is no research directly supporting it. The only work that is possibly relevant will be discussed below in the context of a slightly different theory. However, whether or not the explanation is correct, it would seem to offer us little regarding our current concerns. In most instances of mass illness, the individuals affected are not displaying especially primitive behavior. They are not acting aggressively, destructively; or sexually. If, for example, there were an outbreak of wanton destruction in a factory, that would fit the Freudian model; or if a group of female workers suddenly threw off their clothes and leapt on the available males or on each other, there would be some reason to consider the Freudian view relevant. Indeed, it has been suggested that the outbreaks sometimes seem to have sexual overtones, but far from being

overt, the sexuality, if it is there, is generally disguised. Thus, it is difficult to see how the notion that being in the group releases primitive impulses or relaxes the hold of the superego would account for mass illnesses. One would have to assume that people unconsciously want to become ill or experience the particular symptoms that appear, and that they have held these desires in check, and now are expressing them because of the group effect. This seems highly implausible.

On the other hand, perhaps it could be argued that this is exactly what happens with one limitation - the impulses are being expressed more openly, but the superego still has some strength and thus the impulses, which usually are fully suppressed, appear in disguised form. Nevertheless, this viewpoint still leaves rather vague why the contagion itself occurs. That is, it gives a reason why people in groups may engage in bahaviors they would not engage in by themselves, but it does not explain why, at some point, the behavior of one person spreads to the rest. Nor, for our purposes, does it explain why, after exposure to the same conditions for many months or years, the people suddenly exhibit the symptoms. Thus, this early view is helpful mainly as a description of people in groups but not as an explanation of the specific phenomenon with which we are concerned.

More recently, a number of psychologists have translated these ideas into slightly different terms. Leon Festinger used the term "deindividuation" to refer to the phenomenon in which an individual's personality and will is submerged in a group. According to this view, people in a group tend to lose their sense of personal identity. Instead of thinking of themselves as individuals, there is a tendency for them to think of themselves as group members. This causes them to identify closely with the group, with the other members of the group, and with the goals and action of the group. At the same time, because of this close identification plus the loss of the sense of their own identity, they feel less responsible for their own actions. Whatever the others do, they do, and if one person does something, each individual is likely to do the same merely because they are all members of the same group and because each person's control is weaker than it would be outside the group.

The emphasis in this view is not on the release of primitive impulses, but rather on the lowering of the normal restraint and instead of using psychoanalytic concepts, it discusses the phenomenon in the terminology of research psychologists. According to the deindividuation explanation, it is not that new impulses appear, but that various reasons for restricting behavior are weakened. The individual need fear punishment less, because the blame is spread throughout the group; he need feel guilt less, because in some sense others are causing him to act the way he did; he need not fear social ostracism

or rejection, because he has the support of the group; and so on. All of this allows the person to act in ways he would not, if alone.

The evidence for this view and the somewhat similar views of Le Bon and Freud comes from two kinds of research. The first, concerns how being in a group affects the amount of aggression or anti-social behavior expressed. This work consists of comparing people's behavior when they are alone with when they are in a group. There is very little systematic work, and somewhat surprisingly, what there is runs exactly counter to what would be expected from the theory. Rather than being more aggressive or anti-social in groups, people tend to be less aggressive and anti-social. For example, one study (Diener, 1976) observed young adults in a situation in which they were free to act aggressively toward someone who had not harmed them in any way. In all conditions, the adults observed someone else acting aggressively toward the "victim", thus providing a situation that allowed contagion to occur. However, adults who were alone with the victim expressed much more aggression than those who were in a group. In summarizing all of the research on this issue, Diener (1977) concludes that the evidence does not generally support the idea that group membership increases anti-social behavior. Sometimes it does, but there is no reason to believe that in general people act more "primitively" in groups than when they are alone.

In fact, both research and observation indicate that membership in a group often is an important factor producing compliance with social norms. Group pressure tends to make individuals act in accordance with the norms and rules of that group, and also generally to act in more socially-accepted ways than when they are alone. Thus, being in a group appears to have varied effects on individual behavior. Under most circumstances it causes people to behave more conservatively, more acceptable, and therefore less "primitively". In contrast, under some circumstances it may have the opposite effect. The crucial factor is that the individual will tend to do what he or she thinks the group thinks is correct. Therefore, if the group for some reason holds anti-social values (as in some delinquent groups), someone joining the group may behave more anti-socially than he would alone; whereas if the group holds more socially acceptable values, the individual will behave in socially acceptable ways. The group does not appear to release any primitive impulses - rather it generally puts pressure on the individual to behave in certain ways determined by the group. Perhaps, under some unusual conditions, a normally pro-social group will become anti-social and exhibit an unusual amount of aggressive or destructive behavior; but this is not typical. Thus, the idea that groups necessarily cause individuals to behave in primitive ways seems to be unfounded, and this aspect of the

theories is probably incorrect.

The second source of evidence, especially for the idea of deindividuation, concerns the effect of anonymity on behavior. The argument is that being in a group makes each person stand out less, causes him to be less easily identified, and this results in his feeling deindividuated. At one level, this certainly makes sense intuitively. Certainly when we are in a crowd of fifty people or even in a group of five, we are to some extent considered part of the group rather than as individuals. Someone alone on the street, with no one near him, is more visible and more easily identified than someone walking in a crowd. He stands out more, his behavior is more noticeable- whatever he does, it is clearly the individual doing it. When a group does something, it is often difficult to know just who in the group participated. We know the group did it, but often cannot attribute the actions to any given individual. Similarly, in a factory or office, someone who is alone in a room or is the only one who has a particular job is more easily identifiable than someone in a room with others or who has a job similar to that held by many others. Thus, group membership does tend to reduce individual recognition. The second step in the argument is that this reduced recognition leads to the individual feeling less responsible for his own behavior, feeling deindividuated, and therefore freer to act in ways he would not by himself. Therefore, degree of anonymity becomes a crucial variable.

Quite a few studies have investigated the effect of anonymity on behavior. Although the research is not terribly convincing, the results do generally indicate that people who feel anonymous are more likely to engage in anti-social behavior. Several experiments showed that not mentioning people's names or actually hiding their faces had this effect (Festinger, Pepitone and Newcomb, 1952; Singer, Brush, and Lublin; Zimbardo, 1969). One study demonstrated that children who were trick-or-treating and wearing Halloween masks were less likely to steal extra candy when an adult first asked their names (Diener et al., 1976). And Watson (1973) reported that cultures in which warriors wore masks or otherwise disguised their faces were more likely to engage in especially aggressive acts during warfare than cultures in which warriors did not disguise themselves. On the other hand, at least one study (Diener, 1976) found that anonymity had no effect on aggressiveness.

In any case, there is a serious difficulty with this work. The original idea was that anonymity would lead to deindividuation, but none of these studies provides evidence for this. An easy alternative explanation of increased anti-social behavior is simply that anonymous people are much less likely to worry about actual consequences of their actions. Since they cannot be identified, they are unlikely to be

punished for what they do. That is, no complex psychological explanation is required - the effect may be due to the realistic, practical consideration of whether you can get away with it. Thus, there is little or no evidence to support the existence of a state that might be called deindividuation, and certainly no evidence to show that it is involved in contagion.

The "primitive expression" and "deindividuation" theories are quite similar even though they use different terminology. Both view the individual in a group as feeling less responsible for his actions, and to some extent "caught up" in the actions of others. His own built-in controls are weakened, and he is therefore more likely to do what others do, and especially more likely to engage in anti-social behavior and behavior that runs counter to his own moral and ethical sense. Although the evidence for this view is at best meager, it remains an interesting idea.

Assuming for the moment that being in a group does reduce one's sense of personal responsibility, and that this in turn leads to a tendency to do what others in the group are doing, and that it also allows some behavior that might otherwise not occur, the crucial question is what factors determine when all of this will happen. Clearly, not all groups produce these effects all the time. Returning to our concern, incidents of mass psychogenic illness are extremely rare. People work in groups a great deal of the time, they are exposed to stress much of the time, and an occasional person in these situations must exhibit dramatic psychogenic symptoms. That is, isolated cases of fainting, feeling sick, becoming nauseaous and so on probably appear with some regularity in factories, offices, and other group settings. Yet, the contagion of these symptoms resulting in mass illness occurs in only a miniscule percentage of the cases. What are the crucial factors that trigger the spread of the symptoms? Even if being in the group has the effects described above - of reducing responsibility and increasing imitation within the group, we do not yet have an explanation of why sometimes, quite rarely, this situation leads to mass illness. Thus, this general theoretical approach, while it may provide some framework, offers no answers.

Another theory that may have some explanatory power is in terms of imitation. As proposed by Redl (1949) and Wheeler (1966) the idea is that contagion is essentially imitation that is mediated by the reduction of restraint caused by observing someone else's behavior. As noted previously, the individual is in a conflict situation - he has some reasons for behaving in a particular way, but other reasons for not behaving in that way. He may experience stress, feel somewhat angry, and want to throw a rock through a window; but

he is afraid of being punished by the law, afraid of looking foolish, worried about acting anti-socially, and so on. Under most circumstances, for most people, the restraints are stronger than the impulse and so the rock is not thrown. However, once he observes someone else throw a rock, the idea is that this weakens the restraints and then he is freer to perform the action.

Certainly, there is evidence that observing someone perform an action and not get punished for it, increases the likelihood that others will imitate the act; whereas if the first person is punished, this reduces the likelihood of others performing it (Bandura, Ross and Ross, 1963, etc.). The act need not be especially anti-social to fit this model. For example, many people may be reluctant to be the first one to step onto a dance floor in a night club because they may be afraid of looking foolish or just standing out. Once someone else "takes the plunge" and starts dancing, others join in. Similarly, once someone has applauded at a performance, others tend to applaud; once someone offers help to someone in need, others are more likely to help, etc. The general point is that once you have observed someone else performing an action which you are somewhat inclined to perform yourself, but which you are for whatever reason reluctant to perform, your reluctance may be reduced and you would then be more likely to perform the action. Wheeler (1966) has provided some evidence that this occurs, and other research (Freedman et al., unpublished) also demonstrates the effect.

The question is whether this explains the incidents of mass illness. Clearly, at some level these are incidents that involve imitation. One person exhibits symptoms, often bizarre symptoms, and then others exhibit the identical or similar symptoms. It could be argued that what has occurred is that many people are inclined to show their displeasure at their situation, or to express their negative feelings due to stress, or in some way make a statement about their dislike of where they are or the conditions under which they work. They have probably not specifically considered throwing a fit, fainting, or any particular symptom. But they are tempted to do something. Against this are a variety of reasons for not doing anything along these lines because they will be disciplined by their superiors, embarrassed in front of their peers, or whatever. Once someone, for some reason, does exhibit the symptoms, these other people are less afraid of the negative consequences and they too want to express themselves. Although they would not have considered this particular form of expression, it is a convenient way of expressing themselves and they therefore imitate the initiator.

All of this makes sense and probably does play a role in mass illness. However, just as with the previous theory, it leaves

unexplained why the illness breaks out at a particular time and not at others, why it occurs rarely rather than often, and also offers little in the way of suggestions for reducing the incidence of mass illness.

A final theoretical approach to contagion is quite different from the others. Rather than focus on the mechanisms involved, it concerns the conditions under which contagion is likely to occur. In particular, it is based on research on how the amount of space and the number of people present affect people's behavior. The basic idea is that increased density (i.e., less space available per person) tends to intensify people's reactions to each other. This so-called intensification theory (Freedman, 1975) states that as density increases, other people become a more important and salient aspect of the environment, the actions of others are more likely to be noticed and to impinge on the individual, and therefore reactions are magnified. An analogy might be our reaction to music. If we like the music, as it is played louder, we notice it more, it becomes a more important stimulus, and we like it more; in contrast, if we dislike the music, as it becomes louder, we dislike it more. Similarly, our reactions to other people become stronger as the density increases. Whatever our reactions under low density, they are stronger under high density. If we respond positively under low density, we respond more positively under high density; if we respond negative under low density, we respond more negatively under high density. A considerable amount of research has supported this theory.

This intensification effect has been shown also to apply to contagion. The idea is that contagion depends in part on observing what someone else is doing, on being affected by it, and the more important the other people are, the more contagion should occur. That is, just as other reactions to the people are intensified by high density, so the likelihood of contagion is magnified. This has been supported in two studies - one showing that the contagion of laughter was increased by high density (Freedman and Perlick, 1979), the other that the contagion of applause was increased by high density and also, to a lesser extent, by larger groups (Freedman, Birsky and Caroukian, unpublished).

The application of this theory to mass illness would be that we would expect the possibility of contagion to be greater when people are under relatively high density, and also when they are in larger groups. Thus, working conditions which provide little space per person, and in which many people work, should be more likely to lead to incidents of contagion. If we assume that mass psychogenic illness is due to contagion, it should occur more under these conditions.

It should be clear from this review that none of the theories

of contagion provides a full account of the phenomenon, and certainly does not explain in detail why mass psychogenic illness occurs. The various theories give us some hints as to why contagion occurs at all, and what factors may increase or decrease the chance of contagion. Taking all of the theories at face value and accepting them for purposes of the argument, would lead to the following hypotheses: 1) mass illness should occur more when individuals feel part of a group, than when they feel relatively anonymous. Situations in which people are all working at similar jobs, when they are treated with relatively little individual attention, when they are "cogs" in a machine or part of a large institution, should all increase contagion. Moreover, people who are in this situation should be more likely to be involved in mass illness than people who are more differentiated in terms of jobs, special attention, or whatever. It would follow from this that anything which increases people's sense of being individuals, which causes them to think of themselves as separate from the rest of the group in some way (not necessarily opposed to the group, but merely not just one of many), should reduce the chances of mass illness occurring. 2) Assuming that contagion is due in part to imitation mediated by restraint reduction, the response to the first instance of illness should play a role in reduced contagion. When someone exhibits symptoms, the idea is that this reduces the restraint others may feel and therefore increases the chance that they will show similar symptoms. However, this assumes that the first person showing the symptoms suffers no negative consequences. If, instead, the first person were disciplined, treated harshly, rejected socially, or given negative feedback of any kind, this would presumably reduce the likelihood of further contagion. Of course, this is usually not a practical or ethical possibility. When someone is sick, they are and should be given good treatment that is nevertheless not reinforcing. For example, the "sick" individual could be immediately isolated from the others, thus reducing the chance of contagion and perhaps also implying some negative consequences. Whether this is practical or would work is questionable, but at least a range of responses should be considered. 3) High density, especially when combined with large groups, should increase contagion and the likelihood of mass illness. It should be clear that high density working conditions are not necessarily bad - the evidence is that high density does not generally have negative effects on people. High density may lead to more positive group feelings and higher productivity under some circumstances. Indeed, the stronger group feelings induced by high density are generally considered a positive outcome, though in the present context they would also increase the possibility of mass illness. On the other hand, it does seem that lowering densities and reducing the size of groups should reduce the chance of mass illness by reducing contagion. However, this change is not necessarily

a good one overall, considering that it may also reduce positive group feelings. Perhaps an optimal level of density and group size could be found for each environment such that it was high enough to encourage positive social interactions, but low enough to keep contagion to a minimum.

Considering all of the theories, and the factors just mentioned, we still do not understand why mass illness occurs at a particular time when similar conditions have existed previously. The theories provide some framework within which to consider the phenomenon and offer some suggestions as to factors that affect the likelihood of mass illness occurring. But chance elements almost certainly play a role, as do many various factors that have not yet been specified. It is important to keep in mind that mass illness is a very rare event. The theories specify factors that increase contagion and therefore increase the likelihood of mass illness; but mass illness will not occur often no matter what the circumstances. Thus, these factors should be seen only to increase the chance slightly, perhaps in the way that in golf holding the club just right, and having a perfect swing increase the chance of a hole in one, but holes-in-one are highly unlikely events regardless of what one does.

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SOCIAL AND PERCEPTUAL FACTORS AFFECTING SYMPTOM REPORTING AND HYSTERICAL CONTAGION

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Evidence is presented indicating that the perception of physical sensations and internal state parallels the processes involved in the perception of external environmental events. Two factors influencing the awareness of internal state are presented. If the external environment is lacking in information, the probability of attending to internal sources of information increases. In addition, the schemas or hypotheses we adopt about our health can result in highly specific monitoring of internal sensations. Further, individuals are much more likely to become aware of sensations that can verify an illness schema than sensations that could disconfirm such a schema. Ways in which illness schemas are established and result in selective search of internal state are presented. Finally, implications for the understanding and prevention of hysterical contagion episodes are discussed.

A fundamental issue in understanding psychogenic disorders concerns how individuals perceive internal sensations and physical symptoms. The present work addresses when individuals are most likely to attend to sensations and how these sensations can be interpreted. This approach is based on a perceptual perspective. That is, the processes that have traditionally dealt with the perception of external environmental events can also be applied to the perception of internal state.

In many respects, the perceptual approach supplements many of the perspectives that have been advanced at this conference. Social comparison, coping, labeling, and other theories often begin with the assumption that individuals are aware of internal arousal or physical sensations. I am concerned with the factors influencing the initial awareness of the sensations themselves. In other words, the perceptual approach is limited to peoples' experiences of their sensations on a more molecular level. The subsequent contagion of the sensations or symptoms is more directly addressed by many of the broader social influence theories.

Much of the work to be presented is the product of several experiments my students and I have conducted over the last two years. Many of the studies have dealt with relatively minor symptoms--coughing, itching, nasal congestion, perceptions of cold hands, and racing hearts. Obviously, ethical considerations have limited the domain of symptoms that we can study in the laboratory. However, our findings concerning the various mundane complaints should generalize to more serious physical problems. One reason we are confident about the generalizations of our findings is that we have found divergent symptoms to be influenced in similar ways by common perceptual factors. For example, perceptions of nasal congestion, heartrate, itching, etc. can be manipulated and influenced in comparable ways using similar instructions. In other words, the perception of divergent physiological systems--whether under the control of sympathetic, para-

sympathetic, of even the central nervous systems--is highly dependent on attentional and interpretive factors. Consequently, a pardigm that can promote itching or nasal congestion could, in theory, also increase vomiting, fainting, or other more serious physical problems.

The present paper is divided into three sections. The first discusses the perceptual process in general. The second section deals with attentional and interpretive factors implicated in the perception of internal sensations. The final section will link the perceptual approach to the contagion of psychogenic disorders and recommend techniques that should be effective in limiting their occurrence.

All organisms seek to learn about their worlds. In this learning process, we take in information about objects, events, etc. Although this information is apprehended by visual, auditory, or other receptors, it must be processed at a higher level within the brain. Perception represents a highly active and cyclical process. We do not passively wait for information or stimuli to bombard our receptors and brain. Rather, we actively and selectively seek information.

Many theorists and researchers have addressed how the perceptual process works in dealing with the external environment (cf., Gibson, 1966; Neisser, 1976; Newtson, 1976; Lazarus, 1966). We can study the process from two perspectives. The first is by looking at how external stimulus information is attended to and encoded into categories, schemas, sets, or tentative hypotheses. This view is unidirectional from external events to our understanding of them. The second approach concerns how these categories, schemas, etc. direct attention or search behavior for external information. Clearly, we do not encode everything in our visual world. Instead, we selectively look for information based on our expectations. Our selective search behavior, then, restricts the type of information that we may glean from the environment.

An example of this process can be seen in our walking in the woods and hearing a rustle in some bushes. The rustle may draw our attention because of its novelty, motion, relative complexity (e.g., Berlyne, 1960), or because there are no other competing stimuli present at the time. We may then develop a tentative schema or hypothesis such as "is it a snake?" Given such a schema,we may selectively look at the base of the bushes for a long, slithering object. Another equally plausible hypothesis, i.e., "is it the wind?", will result in our looking for different information (leaves shaking, sensation of coolness on skin, etc.). The type of information that we find may confirm or disconfirm our schema or hypothesis. If it confirms the snake hypothesis, we may then form new sets and begin looking for information (e.g., is it poisonous,

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can I run away or cope effectively?). In sum, the perception of external events is an ongoing process involving both taking in external information and creation or modification of schemas which direct out search behavior. To understand the process, we can study factors that affect the initial awareness of the stimulus as well as the schemas we adopt and the subsequent search behavior deriving from the schemas in order to learn more about the stimulus.

Of particular relevance to the present research are factors which can bias or distort the information we gain from stimuli. One problem is the role of competing stimuli in our noticing a given stimulus. For example, we are more likely to notice a rustle in the bushes if we are sitting alone in the quiet woods than if we are actively engaged in a conversation with a fellow camper. In other words, the probability of noticing any given stimulus is inversely related to the number of competing stimuli in the environment at the time. In addition, recent evidence by perceptually-oriented social psychologists indicate that the schemas that we hold will restrict the information that we can glean from the environment. Specifically, we are much more likely to find schema-consistent or relevant information than schema-inconsistent information (cf., Crocker & Taylor, 1978; Snyder, 1979). Using the above example, if we adopt the snake hypothesis, we would be more likely to notice a long object at the base of the bushes and believe it to be a snake (even if it were a stick), than if we did not initially hold the snake hypothesis. In addition, the snake hypothesis would restrict our encoding information that the wind had caused the rustle.

A basic premise of the present paper is that the perception on internal sensations represents the same processes of perception implicated in our learning of external events. Individuals are more likely to become aware of physical sensations at some times more than others. Further, the schemas or sets to which we have access will guide our search for particular sensations. Such a perceptual view has several implications for symptom-reporting in general and incidences of mass psychogenic illness in particular. The following section will be devoted to some recent findings supporting this perceptual approach. Evidence will be presented demonstrating how lack of external information will increase the probability of becoming aware of internal sensations. Secondly, data supporting the contention that illness-relevant schemas result in the selective encoding of internal sensations will be offered.

In order to experience and report physical symptoms, individuals must attend to their bodies. As discussed above, becoming aware of internal stimuli will be dependent, in part, on the nature of competing external information. In addition, the schemas we hold concerning our bodies will greatly influence our awareness of sensations via selective search. For

future reference, schema, as used here, is defined as a hypothetical entity which readies the perceiver for perceptual experience by providing a structure for assimilating new information. Hence, an illness schema will prompt the perceiver to selectively search his/her body for illness-relevant sensations. The present section is divided into two parts. The first concentrates on factors that will increase the probability of attending to internal sensations. Before we can define sensations as illness related, we must first become aware of them. The second part focuses on the role of selective search as a function of the schemas we hold. This section will explore both selective search and factors that can establish an illness schema in the first place.

Historically, perceptual psychologists have been interested in why we are more likely to attend to certain stimuli than others. For example, Berlyne (1960) notes that stimuli that are novel, complex, or display motion will be examined in greater detail than redundant, simple or stationary arrays. If we assume that attention or search can alternate between external and internal stimuli (cf., Wicklund, 1975), the probability of attending to internal sensations will depend, in part, on the lack of external information and/or heightened salience of the physical cues themselves. In other words, given that both external and internal sources of information are potentially available at most times, the individual must selectively process data. Given the high number of available sources of informations, the processing of any given internal or external stimulus will restrict the encoding of competing stimuli. This implies that external information can be used at the expense of internal information and vice versa.

Logically, in order to experience a sensation, the individual must attend to the body. Several demographic studies indicate that if persons' living or working environments are not stimulating, they are much more likely to report physical symptoms. For example, individuals living alone, in a rural area, or in an environment characterized by social isolation, report a heightened number of symptoms and claim to be in worse health (Wan, 1976; Moos & Van Dort, 1976; Pennebaker & Funkhouser, 1979). Work environments perceived as repititious and boring are positively related to health center and sick call visits (Moos, 1975; Greenley & Mechanic, 1976), and often typify incidences of hysterical contagion (Colligan & Murphy, 1979; Stahl & Lebedun, 1974). Comparable findings have been reported by person-environment fit researchers. That is, individuals whose abilities exceed the demands of their jobs evidence an increased degree of somatic complaints (Harrison, 1976), symptom reports (Coburn, 1975), and signs of somatic depression (French, 1973). Wright, et al (1977) found that smelter and mine workers who found their jobs uninteresting over-reported respiratory symptoms relative to an objective measure of respiratory dysfunction. In other words, lack of

external stimulation is associated with reporting of physical complaints. Of course, each of these studies were correlational, thereby making it difficult to establish causation. However, the converging evidence suggests that when external demands are low, the probability of attending to internal sensations increases. Once aware of the sensations, individuals may become more attentive to internal state in general, exaggerate them, and ultimately seek medical attention.

Recently, we have begun to examine health-relevant behaviors as a function of external stimulus demands in experimental settings. The first line of research on this problem has dealt with coughing and clearing-of-the-throat behavior. Coughing is a response to the perception of a tickling or irritated throat. In order to be aware of tickling or irritation, we must attend to our throat. In the first of several studies, we showed an 18 minute movie to 50 college students. They were asked to rate the movie on how interesting it was every 30 seconds, along a 7-point scale. We then showed the movie to three Introductory Psychology classes of 250, 120, and 60 students. While watching the movie, the number of coughs were counted and collapsed into the same 30-second blocks previously rated on their interest. The simple correlation between the number of coughs per 30-second interval and interest rating for the same intervals was a highly significant - .57. In other words, the less interesting parts of the movie were characterized by a larger number of coughs. Similarly, we have found strong negative correlations between students' liking of a given course and the number of coughs emitted during a typical lecture by the teacher of the course.

A second line of research substantiates this reasoning within a person-environment fit paradigm (Pennebaker & Brittingham, 1979). In these experiments, college students worked on serially-presented, simple arithmetic problems at one of three paces: slow, moderate, or fast. As expected, people reported the most physical symptoms (e.g., headache, racing heart, upset stomach) in the slow and fast conditions. In a second study, we sought to tease out factors influencing attention to body and actual autonomic arousal. Again, people worked on the arithmetic problems at one of the three paces. Skin conductance was continuously measured. In addition, subjects were told that they would receive short burst of air on their arm and none on their back. Symptom-reporting was again curvilinear with Moderate-paced subjects reporting the fewest symptoms. Skin conductance and accuracy of recall about the squirts served as measures of autonomic activity and attention to body, respectively. If differential autonomic activity accounts for the curvilinear pattern for symptom reporting, skin conductance should be higher in the Slow and Fast conditions and lowest in the Moderate cell. Conversely, if attention to body underlies symptom-reporting, we would expect

greatest accuracy of recall about the squirts in the Slow and Fast conditions. In fact, both processes appear to be working. In terms of recall about the squirts, only subjects in the Slow condition were accurate. The Moderate and Fast subjects were equally poor at recalling the number and location of the squirts. In terms of skin conductance, only Fast subjects maintained a high level throughout the experiment. Both Slow and Moderate subjects adapted equivalently.

These data, then, offer indirect evidence that an environment lacking in external stimulation allows for greater attention to body, thus resulting in increased probabilities for reporting physical symptoms. When a person must deal with an over-abundance of external information, he or she evinces heightened arousal or autonomic activity. Once the task is over, the person probably becomes aware of sweating hands, racing heart, etc. and accurately reports them. These data, together with other studies (e.g., Pennebaker & Lightner, 1979) indicate that situations lacking in external information result in attentional focus to the body and subsequent symptom awareness and reporting. The implications of this relationship will be discussed in the final section.

The approach described above represents an unguided search for information. That is, at certain times individuals have an increased probability of noticing internal sensations. Although our search for information about our bodies is often dependent on lack of external information, much of the time we are directly attempting to learn about specific sensations based on hypotheses, label, or schemas we hold. These schemas are particularly important in that they result in highly specific attention to certain sensations. Further, the sensations will be experienced in very different ways as a function of the schemas.

Before discussing our research concerning selective search, it may be instructive to report a case study related to contagion which occurred in a summer school class I taught last year. The class of 25 students met from 10:30 to 1:30 each day in a biology laboratory classroom. The large room was equipped with gas outlets. On one day the airconditioner was broken, causing the room temperature to increase to about 85° F. Both the students and I became extremely tired as my lecture droned on. Had I passed out a symptom checklist, virtually everyone would have reported that they felt drowsy and sweaty because of the heat. In other words, our perception of the heat resulted in our monitoring specific heat-relevant sensations. Midway into the lecture, a woman on the first row raised her hand and announced that she smelled gas escaping from one of the jets. Immediately, I noticed that I felt nauseous, dizzy, and on the verge of fainting. These perceptions were shared by over half of the students (In fact, those who did not experience these symptoms assumed

that I had set up the entire scenario as a class experiment!) As an aside, there was no escaping gas.

This mini-contagion episode exemplifies the role of selective search as a function of available schemas. First, the heat had produced a variety of common but ambiguous sensations. Having experienced heat, we all were familiar with certain sensations that accompany it. In a hot room, then, we selectively attend to parts of our body that are relevant--sweating, drowziness, etc. We typically do not look for irrelevant sensations. However, the introduction of a new schema or label will cause us to monitor other sensations that may be consistent with the new schema. In other words, we are selectively gathering new sensory information and organizing it differently. From the example, switching from a heat to an asphyxiation mode causes us to interpret drowziness as faintness, or hunger pains as nausea. We ignore our heat-induced sweating and other asphyxiation-irrelevant sensations. In sum, the adoption of a new schema will result in our attending to different sensations and interpreting them differently.

This assumption has prompted two related lines of research. The first concerns how schemas result in the selective search and encoding of sensations. The second question deals with what factors can establish differing types of body-relevant schemas. These two problems will be discussed separately. For the past year, J.A. Skelton and I (Pennebaker & Skelton, 1978; 1979) have conducted several experiments on factors influencing the perceptions of nasal congestion, heartrate, and skin temperature. Our initial idea was that merely focusing attention on some part of the body would result in heightened symptom reports. In the first experiment dealing with nasal congestion, subjects were asked to pay attention either to their nose and nasal passages or to an irrelevant object. To our surprise, we found no differences in perceived nasal congestion following these attention instructions. Our next tack was to manipulate how people were to attend to nasal sensations. That is, we sought to establish congestion-relevant schemas so that the nasal sensations could be organized in a more specific way.

The second experiment manipulated focus of attention and the ways in which nasal sensations could be encoded. Subjects were randomly assigned to one of four experimental conditions. As in the first study, two of the conditions required subjects either to attend to their nose and nasal passages (Nose Neutral condition) or to an irrelevant object (Distraction condition). In addition, one group of subjects was instructed to attend to sensations indicating "congestion, nasal blockage, and difficulty in breathing through the nose" (Nose Negative). Finally, a fourth group of subjects attended to sensations indicating nasal clarity and air passing freely through the nose (Nose Positive). The Nose Negative and Nose Positive

subjects, then, had instructions to selectively attend to nasal sensations. As can be seen in Table 1, perceptions of congestion were influenced only in the two selective attention cells. Following the experiment, each subject was extensively debriefed. Three subjects--all in the Nose Negative condition--blew their nose during this time. No others did so. One subject in the Nose Positive cell remarked that her nose hadn't felt too clear in weeks despite her using various decongestants. Unfortunately, the nasal congestion study did not employ an actual measure of congestion. To learn if the schema related instructions actually produced somatic change, a third experiment was conducted.

Table 1. Self-reports of nasal congestion before and after the attention manipulation

Condition	Before	After	Difference
Nose Neutral	60	67	+7 ^{ab}
Nose Negative	56	77	+21 ^{a}
Nose Positive	74	61	-31 ^{b}
Distraction	45	45	0 ^{ab}

Note: Responses were along a 100 mm line, where 1= not at all congested, and 100= completely congested. N=12 per cell. Numbers with different superscripts differ at p .05.

The next study examined changes in perceptions of heartrate and employed an actual measure of heart beat. As in the Nose study, subjects were instructed to attend either to the heartrate (Neutral condition), a racing or accelerating heartrate (Acceleration condition), or a slowing or decelerating heartrate (Deceleration). Although there were no differential changes in actual heartrate, Acceleration subjects perceived an increase in their heart beat, Deceleration subjects perceived its slowing down, and Neutral subjects perceived no change (Means of the changes of the three groups: Acceleration = +11.0; Deceleration = -9.4; Neutral + -1.9; all groups were significantly different from each other at $p < .05$, except for the Deceleration versus Neutral contrast which differed at $p < .15$). The results of this study, then, indicate that how subjects attend to their internal sensations is crucial in bringing about changes of perceptions of physical symptoms. Further, significant changes in perception are not entirely dependent on physiological change.

A clear problem with the above studies is that we were placing heavy demands on the subjects to report sensations in one way or another. In addition, subjects could have distorted their self-reports knowing that we could not detect if they were accurate or not. Even in the Heartrate study, subjects were only told that we were measuring some physiological index and were not told that it was a measure of heartrate. Consequently, we conducted a fourth experiment looking at changes in skin temperature using a different paridigm. In this study, subjects were told that we were examining the aftereffects of ultrasonic noise on task behavior. Subjects expected to listen to a high frequency sound beyond their hearing range while we monitored skin temperature. In addition, we told the subjects that the (bogus) noise had been shown to cause vasodilation or warming of the hands, vasoconstriction or cooling, or have no effect on skin temperature. Although all subjects thought they were listening to ultrasonic noise over headphones, they actually heard nothing. Following the "noise" period, they completed a short questionnaire ascertaining the degree to which they noticed their finger becoming warmer or cooler. As expected, "Increase" subjects detected their fingers becoming warmer, "Decrease" subjects reported a drop in temperature, and "Control" subjects detected no change. (Means of the three groups along a 100 mm line, where 1 = becoming cooler, and 100 = becoming warmer: Increase = 69.6; Decrease = 48.1; Control = 57.8. All means were significantly different from each other at $p < .05$).

As in the Heartrate study, there were no differential changes in actual skin temperature. Although self-reports of change were not correlated with actual change, an interesting pattern of results emerged in examining fluctuations in skin temperature. A fluctuation was defined as a change in the direction of temperature during the noise period, such as temperature becoming warmer, then cooler, or vice versa. Simple within-cell correlations of fluctuations with self-reported skin temperature were +.48 in the Increase cell, -.49 in the Decrease cell, and a non-significant +.11 in the Control condition. These data indicate that subjects were only encoding or noticing changes that would verify their expectations and ignoring disconfirming skin temperature information.

Taken together, these studies demonstrate that merely focusing attention on the body is not a sufficient condition for changes in the perception of physical symptoms or sensations. Rather, people must have a tentative schema, hypothesis, or general way of organizing and encoding internal sensations for them to have meaning. In other words, the schema guides ones' focus of attention, resulting in the selective encoding of sensations. Further, this search is biased in that one is more likely to encode schema-verifying information than information that could disconfirm the schema.

Given that the schemas we hold are important factors in the perception of internal state, it is critical to examine how schemas are adopted. That is, under what conditions are we most likely to encode internal sensations within an "illness" or perhaps "hysterical contagion" mode? Although there are a variety of developmental, cultural and dispositional variables implicated in this process, I will restrict my discussion to social factors that can establish body-relevant schemas. It must be noted, that at this point many of the approaches that are being presented at this conference directly bear on the factors which can induce an illness schema. Although our terminologies may differ, we are addressing the same factors.

For the past several months, my students and I have been a attempting to discern which factors are most likely to result in a person's selectively attending to the body. Examination of the hysterical contagion literature clearly indicates that seeing another person become sick or emit a bizarre behavior will increase the probability that others will behave likewise (cf., Markush, 1973). In other words, unambiguous overt behavior is more likely to spread than highly subtle illness behavior. An example of this is coughing behavior. In analyzing coughs during exams, we have found that coughs occur in "bunches". That is, there may be 30 seconds of silence and then 8-10 coughs in the space of five seconds. Just hearing another person cough, then results in a quick monitoring of one's throat and the emission of a cough.

One of our first questions concerning the establishment of a health or illness schema dealt with the probable cause of the illness. Recall that in many of the recent contagion incidents there has been a presumed cause--an invisible June Bug (Kerckhoff & Back, 1978), a mystery gas (Stahl & Lebedun, 1974), pollutants in the air (Rouche, 1978), etc. Is the attribution of a cause for the illness a necessary condition for the immediate spread of the symptoms? That is, after seeing a coworker collapse, does the individual need to go through the cognitive process: "Joe just collapsed; he must have come into contact with some toxic agent that caused it; I have probably also been exposed to the toxic agent; therefore I, too, will collapse". Some preliminary work suggests that this attribution process does not always occur.

Our first study examined scratching behavior, which we can assume is a response to an itching sensation. We were interested in learning the degree to which a possible cause of an itch would result in another person's also scratching. In the study which took place in various libraries on campus, two experimental confederates sat down by a person who was reading. After one minute, the confederate sitting next to the unsuspecting subject did and said one of four things:

she began scratching and announced to the other confederate (in a stage whisper) that she was just bitten by a mosquito (Mosquito condition); she scratched and said that she got sun poisoning at the lake (Sun condition); she scratched and said that she was hungry and wanted to get a hamburger (No Cause condition); she did not scratch and said she wanted to get a hamburger (No Scratch). The two confederates then left while an observer watched the subject for one minute to see if she or he scratched. In sum,the confederate scratched and gave a possible contagious cause (mosquitoes), an implausible contagious cause (sun poisoning), or no cause at all. Of the 16 subjects in each condition, 50% scratched in the No Scratch condition. In other words, people's scratching was unrelated to the purported cause of the confederate's itching.

In retrospect, these results are not surprising. For example, when my research assistants and I got together to talk about the study, we would all catch ourselves scratching feverishly even though there was no objective agent causing the itch. In various interviews, people note that if they see someone vomit, they too become nauseous. In other words, merely observing illness behavior sets up a tentative schema or hypothesis such as "Am I sick? or Do I itch somewhere?" This schema results in specific monitoring of relevant sensations.

It must be noted that such schemas can be established in ways other than observing overt behavior. For example, reading or hearing about an illness can prompt search behavior. Audrey Burnam and I (Burnam & Pennebaker, 1977) conducted two simple questionnaire studies examing the verbal encoding of schemas. In one study, we simply passed out a symptom questionnaire that was headed in one of two ways. One version--the Illness Set--simply noted, "As you know this is the time of the year when we are surrounded by cold and flu-producing viruses as well as other illness-related organisms." The other version omitted the statement. As we predicted, people reported significantly more physical symptoms in the Illness Set condition than the No Illness cell. This trend was especially strong for the flu-relevant symptoms (e.g., headache, upset stomach) than the symptoms unrelated to the flu(e.g., itching, ringing in ears). These data, then, indicate that illness schemas can also be established along modes other than overt behavior.

A particularly striking example of the establishment of illness schemas via verbal encoding can be seen in cases of medical students' disease wherein medical students adopt the physical symptoms of the diseases that they are studying (cf., Hunter, et al., 1965). Interestingly, as in cases of hysterical contagion, most incidences of medical students' disease occur when individuals are under great stress. That is, ambiguous internal sensations associated with heightened autonomic

activity can readily be encoded into the available illness schemas.

This section has broadly addressed factors which can influence the awareness and reporting of physical sensations: individuals constantly seek information about their world. If the external environment is lacking in meaningful information, individuals are more likely to attend to internal, sensory sources of information. Attention to internal sensations is also influenced by the schemas we adopt. These schemas can result in monitoring selective sensations to confirm or disconfirm the schemas. The search process is biased in that people are more likely to notice schema-relevant sensations than sensations that could disconfirm the schema. Finally, the schemas we adopt about our bodies can derive from seeing, hearing, or reading about illness. Further, the adoption of an illness schema need not be processed in a causal way. That is, individuals do not necessarily go through the process of assuming that the cause of a coworker's illness is the same cause of their own sensations. The following section will address some implications of the perceptual perspective for the hysterical contagion of psychogenic disorders.

As has been stressed, symptom awareness requires attention to the body. Any factors that promote monitoring of internal state, then, should increase the probability of perceived health problems which may result in higher absenteeism rates or causes of hysterical contagion. A job that is undemanding because of its repetitiveness, lack of intrinsic interest, or even slow pace may discourage external attentional focus. Lack of social interaction should also promote this process (cf., Cobb, 1976; Colligan & Murphy, 1979). Social support has the dual function of drawing attention away from the body as well as providing an information source for the person to better evaluate the sensations. Persons in settings lacking external information or social support have more time to notice and dwell upon subtle sensations. The more they inspect these sensations, they may begin to worry and define them in illness terms.

In practice, the establishment of environments that encourage external focus of attention may be difficult to engineer. Depending on demands of a job, music or other distractors may be feasible. However, such a task may only be effective in the short term. Ideally, of course, boring jobs should be made more interesting, some degree of social interaction should be encouraged, pacing of tasks should take the persons' abilities into consideration. Hopefully, management and labor could be told about the attentional factors involved and work out solutions that would be practical. If adjustments in the work setting could be accomplished, the payoff for all involved would be high.

The problems of illness schemas and selective search pose a more basic concern. Our research indicates that merely observing or hearing about an illness is likely to lead to an immediate search for comparable sensations. Although hypothesized physical agents such as odors, bug bites, etc. may prolong the contagion episodes, they may, on occasion, be secondary factors in the initial stages. That is, seeing someone vomit or faint may lead to an immediate monitoring of internal sensations. The assignment of a causal agent may well occur after the person has also detected signs of nausea, rather than before.

Virtually every episode of hysterical contagion occurs among workers or students who are tense, anxious, overworked, or in a state of increased autonomic activity. As has been suggested, this arousal could be the result of poor worker-management relations (cf., Colligan, 1978), poor lighting or temperatures (Colligan & Urtes, 1978), crowding (Freedman, 1979), loud noise (Shepard & Kroes, 1975), role or status incongruities (Kerckhoff & Back, 1968), family or financial pressures (Colligan & Murphy, 1979), or a combination of these and other stressors. No matter what the true cause of the arousal, the important fact is that the affected individuals are constantly experiencing a high number of diverse and ambiguous internal sensations. Based on our research with illness schemas, it would be predicted that the more physical sensations, present, the easier it would be to confirm any number of illness hypotheses. That is, with such a high number of available sensations to monitor, a person could easily confirm his or her being asphyxiated, succombing to the flu or more exotic ailment, radiation poisoning, etc. As an aside, the recent nuclear reactor problems may serve as particularly fertile bases for future illness schemas. As Robert Lifton (1967) has chronicled in his book concerning the Hiroshima victims, and as recent new media accounts surrounding the Three Mile Island accident imply (cattle deaths, illnesses, etc.) an ambiguous illness schema such as radiation can essentially account for any physical symptoms. Consequently, individuals exposed to radiation may well chronically attend to internal sensations at a much higher rate than if they were not exposed. Although this may not directly result in cases of hysterical contagion, it could have the effect of greatly increasing absenteeism and visits to medical personnel.

Obviously, the best way to circumvent the selective search process would be to reduce the true causes of anxiety and arousal. This would effectively diminish the number of sensations a person could use to confirm a given illness schema. Given the real world, however, the reduction of tension is not always a viable alternative. In such cases, an effective preventive technique may be to establish or alter peoples' schemas concerning their sensations. A possible way of accomplishing this would be to teach individuals which physi-

cal sensations are typically associated with tension, noise, or job-related stress. From my own talking with hundreds of undergraduate students, it is clear that most do not always associate stress with specific constellations of symptoms. We have found that most people usually associate only one or two sensations with tension--even though our physiological measure indicate they are evincing changes in skin conductance, heartrate, or skin temperature. In other words, when people are in an anxiety-provoking situation, they are attending to only a select group of possible sensations. In so doing, they are ignoring other tension-relevant sensations which could potentially verify other competing schemas. If individuals were trained to associate specific groups of sensations to the autonomic changes associated with their jobs, they might be less apt to later attribute them to illness. We are currently planning to test this idea with beginning college students this coming fall.

A final issue is how to deal with cases of hysterical contagion once they have begun. Simply telling the affected individuals that their symptoms are not really occurring or are purely psychosomatic would not be effective. They are experiencing the symptoms. They do have concrete sensory data to verify their illness schemas. Unfortunately, the attending physicians, psychologists, NIOSH researchers, or other personnel are in a major bind since, in some cases, the contagion may truly be the result of toxic agents rather than misperceptions of internal state. Consequently, only after medical or environmental tests have indicated no physiological bases for the disease can the workers be appraised of the possible psychological causes. Once aware of the perceptual processes implicated in the hysterical contagion incidents, similar outbreaks within the job or school setting may be prevented.

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THE STRESS AND COPING PARADIGM: AS APPLIED TO THE INVESTIGATION OF MASS PSYCHOGENIC ILLNESS

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The two tandem contributions to this conference volume (Lazarus & Folkman and Folkman & Lazarus) should be thought of as separate though interdependent components. The present paper deals exclusively with metatheoretical concerns and strategies of research on stress and coping. It focuses on transaction and process, levels of analysis, and on how to make the study of transaction and process possible. The present chapter also points toward our main substantive theoretical concepts, namely cognitive appraisal and coping, which, we propose, could be helpful in understanding mass psychogenic illness and which are examined in the second chapter (i.e., Folkman and Lazarus). Our overall paradigm approaches mass psychogenic illness from a rather different perspective, both theoretical and methodological, than has typified most previous work.

Research in mass psychogenic illness has been characterized by efforts to correlate illness on the one hand with environmental factors such as physical conditions, organizational structure, and conditions promoting boredom, and person factors such as education, sex role identification, and pattern of defense on the other. This style of research measures presumably stable psychological tendencies and chronic environmental conditions, that is, structure rather than process. The goal is to make probabilitistic statements of the sort: (1) certain types of people (with particular personalities and/or constitutional predispositions) are more likely than others to develop mass psychogenic illness; (2) any person is more likely to show mass psychogenic illness under certain kinds of environmental settings; (3) there is some form of interaction between (1) and (2) such that both sets of factors are necessary to produce mass psychogenic illness, or that both factors together increase the risk additively or in accord with some other mathematical function.

Although the structural research approach can define starting points by highlighting person-situation characteristics that seem related to vulnerability, it sheds no light on the psychological and social processes by which people become ill with psychogenic disorders. Yet it is these processes that hold the answers to the riddles of psychologically generated mass illnesses. We have developed this theme elsewhere (cf. Lazarus, Cohen, Folkman, Kanner, & Schaefer, in press; Lazarus 1978), but it deserves a brief clarification here. What is missing from structually oriented, strictly normative research is the study of what is actually happening socially, psychologically and physiologically, when a person develops a psychosomatic illness, or when such an illness improves or worsens. For example, about hypertension (which can serve as a model) the first author (Lazarus 1978) has written:

> Crucial to understanding hypertension is the com-

> arison of those psychological processes that lead to raised blood pressure and those that do not. We must ask what it is that the hypertensive is doing when his or her blood pressure is up and when it is down, and what the normotensive is doing to produce similar variations... The crux of the matter is to study the ebb and flow of blood pressure concurrently (Luborsky, Docherty, & Penick, 1973) with psychological processes such as cognitive appraisal and coping, so that these processes, the psychological and tissue, can be tied to each other. (p.37)

Likewise in the case of mass psychogenic illness, we must come to understand (for each individual involved) what is happening to him or her during the entire course of the episode from beginning to end at several levels of analysis. In effect, to the extent we can, we must study the ongoing social, psychological and physiological processes in affected and unaffected workers, something that antecedent and consequent research cannot do.

To more readily grasp the above assertion that traditional cause-and-effect, structurally oriented research cannot tell us about the processes accounting for adaptational outcomes such as somatic health/illness, morale and social functioning, consider the analogy of a cooking recipe. If one examines what a recipe does, the point becomes clearer. A recipe gives the cook a time-related, step-by-step set of things to do. It gives more than ingredients, that is, the causal variables that are required for eggplant parmigiana or a souffle. Just telling the cook the ingredients to use, even down to the last measure, does not work. This would not produce a constant outcome because what is needed is a detailed understanding of what has to be done, including mixing and cooking the ingredients in a precise sequence, cooking temperatures at each stage, timing, etc., in short, the processes whereby the ingredients are turned into the dish one expects. Except for the simplest dishes in which one simply mixes everything together at once and bakes (or whatever) for a given time at a given temperature, the recipe is a key statement of food processing without which no constant outcome is possible.

By the same token, scientific recipes are statements about the processes occurring as well as about the causal variables or ingredients. We do not have adequate understanding when we have, alone, identified the causal variables, though this is surely a step in the right direction. Such understanding, required for any kind of intervention for treatment or prevention, calls for the capacity to describe the processes whereby the variables (ingredients) turn into adaptational outcomes, in this instance, the outcome of mass psychogenic illness. The research that has been done to date has focused

on isolated sets of variables in a linear causation model, but has failed to help us understand the processes that take place in individuals, both affected and unaffected, when an outbreak of mass psychogenic illness occurs. We shall have more to say about process a bit later.

To understand the processes taking place in mass psychogenic illness, a style of research must be adopted which examines the dynamic relationship, the actual transactions, taking place between a given type of person (defined personalistically or in terms of physical constitution) and given types of environmental settings. We call this a transactional approach.

The conceptual differences between structural and transactional research can be illustrated by work in psychological stress. One of the notable characteristics of most stress research and theorizing is that it focuses separately on attributes of the environment or attributes of the person to explain stress phenomena. Thus, stress is most often defined in terms of stimulus events or environmental stressors external to the person, or in terms of intrapsychic factors related to increased person vulnerability to stress. The use of life events as a metric with which to predict health outcomes is an example of the former approach, while research and theory on Sely's General Adaptation Syndrome illustrates the latter.

Psychological stress, however, cannot be understood in terms of independent person and situation variables. Stress always refers to a troubled relationship between a person and the environment. In psychological stress, someone with certain commitments and belief systems is evaluating his or her environment as harmful, threatening or challenging, a process we have called "cognitive appraisal." Stress occurs only when an environmental demand, constraint, or opportunity is judged to tax or exceed the person's resources for dealing with it. It is, in short, a particular kind of person-environment relationship in which the environmental demand has relatively more power to harm than the person has the power to prevent or ameliorate that harm (as cognitively appraised).

In the case of mass psychogenic illness, symptom outbreak can be seen as evidence of such a troubled relationship. At first, then, the task would seem to be to understand the environmental demands, constraints or opportunities, the person's resources for coping with them, and the reasons the resources are not fully adequate. However, this would merely be another form of correlating person and situation factors with outcome, and would still miss what is critical to an understanding of both psychological stress and mass psychogenic illness, namely, the psychological and social processes that characterize the transactions people have with

their environment.

Four ideas are basic to the concept of transaction (see Pervin 1968; Dewey & Bentley 1949; Riegel & Meacham 1978). First, a transaction refers to an actual encounter with the environment, often taking place over a period of time. We are not speaking of a type or a trait, but of a specific interchange between the person and his or her social and physical environment. Also, causality is always a two-way street. Not only does the environment affect the person, as in a stimulus-response sequence, but the person also affects the environment. The interaction goes both ways in the course of an encounter, thus the term transactional.

Second, the identification of a variable as antecedent or consequent is provisional, and is determined arbitrarily by where one enters the flow of the transaction. For example, cognitive activity in the appraisal process results in some particular emotion such as fear, and the emotion in turn influences further cognitive activity. The answer to the question about which is causal, the cognition or the emotion, is nearly always "both," depending on where in a transactional sequence one chooses to look.

Third, the person and the environment do not remain in a static relationship; they influence or change each other during the course of an encounter. In an angry interchange between two people, there will be changes in emotions and cognitions on the part of both persons as each acts and reacts, creating a different relationship at time two than at time one. In effect, process is a key feature of a transactional perspective.

Fourth, variables can also fuse and integrate to spawn new relational or, as we call them, transactional variables. Threat is an example of this type of variable. Although threat depends on knowledge of environmental forces and the person's resources for dealing with them, it actually describes the relationship between the person and environment factors, creating a variable that represents a higher level of abstraction. Another example of a transactional variable is job involvement. Job involvement cannot be understood solely through knowledge of person factors such as motivation, or situation factors such as status or job complexity. Instead, it represents a higher-order variable in which these persons and environment factors are integrated, creating more than their sum (Lazarus, Note 1).

At first this definition of a higher-order variable sounds like the traditional definition of interaction used in the analysis of variance in which effects are explained by the interplay between person and situation variables. An interaction is said to occur when independent variables, such as a trait and a situational characteristic, together account for

more variance than is accounted for by adding the separate independent variables. However,this definition of interaction does not imply any type of change in the contributing person and situation variables, nor does it imply an integration to create new higher-order relational variables. In interaction, the interacting variables can always be identified in their origianl forms. As such, it differs fundamentally from a transactional formulation (cf. Olweus 1977; Lazarus, Note 1).

The four major ideas in transaction--that it refers to an actual encounter, that there is mutual causality between the person and the environment, that the variables change, and that higher-order variables evolve--share an emphasis on dynamics and change or, as this is commonly referred to, process. It is through process that the transactional relationship the person has with the environment can be studied.

The study of process has long been the concern of clinical cognitive psychologists. Zane (1978) provides a good illustration of intraindividual process in a case study of a phobic patient. He asks the patient to record her thoughts and feelings on a tape recorder during a threatening three-minute period when she has been left alone in a room. The protocol reflects the dynamic interplay of cognition and emotion. Zane tells us to notice how the patient's phobic behavior changes as her body reacts automatically to changing thoughts and images, and to perceptions that change with them. A plus sign in parentheses means that the phobic behavior gets better, a minus sign that it gets worse, a $\pm$ that there is no change. For example:

> (Door shuts, -) "right now... I feel panic... cause I'm realizing that only...not even one minute has gone by and I have to deal with over another two minutes and I don't really know where he is and I feel like I"m getting very hot and sweaty (-)...But just talking is making the time go. If I just look at this watch... like every minute is an hour...Now if I look at it on a larger scale three minutes is nothing and that would make me relax. My level has just gone down (+). Now if I imagine him really walking down the corner someplace ...even a split second now my heart didn't beat right. I couldn't, I wouldn't know where to go to reach him (-). But for some reason it doesn't scare me because I see almost two minutes is up. (Breathes heavily, +) All I know is that I'm glad I only said three minutes. But I'm holding on,even though my stomach is tight (=). I really am alone in this whole house. The only security I have right now is that I said three minutes (-). Now he should be starting to walk back soon (+). (Breathes, Clicks watch) I stopped it. Three minutes ($\pm$) Oh, yes. And here he comes. (Noise, door shuts) Right on time (+)." (pp. 340-341)

Cognitive psychologists have used similar "think out loud" method to find the cognitive processes through which individuals learn to solve problems. Anzai and Simon (1979), for example, had a subject think out loud while attempting several solutions to a puzzle. They analyzed the subject's protocol to provide a detailed picture of the transformation process--in particular, the sequence of cues she noticed in the problem structure and the use made of the cues in modifying her strategy. Without knowledge of the subject's cognitive processes, they would have had no way to understand the links between the presentation of the puzzle and the outcome.

A study by Cohen and Lazarus (1973) compared the usefulness of the process and trait approaches in predicting an outcome in a specific encounter, surgery. Cohen and Lazarus interviewed patients the night before an operation to determine how much they knew about their disorder and the nature of the surgery. This knowledge reflected whether they were coping by vigilance (seeking information) or avoidance (avoiding information). They found some patients were extremely vigilant while others were extremely avoidant. In addition, a trait measure of the same tendency was employed (Byrne 1964). The trait measure was distinguished from the process measure by assessing subjects' usual ways of reacting, rather than their actual reaction to the specific encounter, surgery.

The process measure and the trait measure were found to be unrelated, and only the process measure predicted post-surgery status. To make an accurate prediction required assessing how the person was actually handling the situation, in other words, it required an assessment of process.

In a naturalistic investigation of process and transaction, one of the first tasks is to define the unit to be analyzed. In Zane's (1978) study the unit was a phobic episode, in Anzai and Simon's (1979) study it was a puzzle solution, and in Cohen and Lazarus' (1973) study it was the imminence and threat of surgery. In our current research on stress and coping, the unit is recently experienced stressful encounters. We asked our subjects to identify the most stressful event they had experienced during recent weeks, and through interview or questionnaire we had them reconstruct the event by telling what happened, who was involved, the background, emotions experienced throughout the encounter, and the thoughts and actions the subjects used to deal with the situation. The units of analysis in the four examples above share in common the focus on an actual encounter and the psychological and/or social processes that unfolded during it.

The unfolding we speak of can take two forms: First, it can refer to changes in the process (say, cognitive appraisal or coping) across various stressful encounters, that is, different stressful occasions or episodes; second, it can also refer to

changes that occur within a single episode as when a person makes coping efforts that change the nature of the encounter. Some stressful episodes are short-lived, while others are very lengthy, as in bereavement, which may last for many months or even years.

Although in our discussion above we have emphasized the second form of unfolding or process, that is, what happens in a given encounter, the importance of looking at process across encounters, especially if they are united by a common theme, cannot be minimized. A good example of this is bereavement. Bereavement may actually extend over a very long time, sometimes years, and it encompasses many encounters and psychological changes. What happens at the outset when discovery of the loss occurs--the shock, dazed state, confusion, bustle of activity of family and friends taking care of necessary tasks such as the funeral and giving emotional support--changes greatly as time passes and different stressful encounters are experienced. Ultimately, the full realization of the loss must occur, then its acceptance, and finally, after perhaps a period of depression and withdrawal, a reinvestment in living and a search for new commitments. The process of grieving can be captured only by describing the transformation in multiple person-environment relationships as each encounter gets underway, proceeds and ends. To study coping as it manifests itself only at one point or occasion in grieving will reveal a pattern quite different from what is manifested on another.

A second task is to decide what kinds of process to examine--physiological, psychological, and/or social. This is a particularly pertinent question for research in mass psychogenic illness, where all three processes are salient. Physiological process is manifest in illness, psychological process has to do with the person's cognitive appraisals and patterns of coping, and social process plays key roles as providing some of the environmental sources of stress and in the "contagion" feature. The importance of all three levels of analysis in mass psychogenic illness demands that particular attention be given to how each level influences the other.

Research and theory in stress, coping and adaptational outcome (e.g., health/illness, morale, social functioning) occur at all these levels, but findings are confusing because the rules that link the levels are not clear. For example, the levels are often considered dependent or interchangeable, with the manifestation of stress at one level implying stress at another (Lazarus 1966; Lazarus & Cohen, Note 2). Thus, it is assumed that if a person is showing physiological signs of stress, he or she is experiencing stress psychologically as well. However, a person may show marked physical stress reactions while jogging, but be in a happy, relaxed state of mind. Or, conversely, a person may show minimal levels of

stress physiologically, but be observed as troubled psychologically, as in the case of a person who uses intellectualization (distancing, detachment) to deal with a difficult problem. In an even more complex example, and one that is particularly relevant to mass psychogenic illness, the onset of an illness (physiological stress) can bring psychological relief to a person who uses illness to escape a troublesome work situation while causing stress at the social (family or work) level. On the other hand, a decision to divorce may be threatening to a husband or wife at the individual (psychological) level and eventuate in illness, but relieve strain at the social (family) level by resulting in fewer interpersonal conflicts. Later, however, there may be cessation of stress at the psychological level when time has permitted effective adaptation, showing that all such relationships are time-contingent, varying greatly within it.

Although we must be wary of assuming that stress at one level implies an analogous process at another, often they occur together and, similarly, what is happening physiologically in the social stress context will not make sense without recourse to psychological mediators. It is important and legitimate to cross levels of analysis as long as these are dealt with and measured independently. Two recent studies illustrate how this can be done effectively.

The first example comes from some research of Pearlin (1975), who attempted to study a problem that has generated great interest in sociology, namely, that of status inequality between spouses as a source of stress in marriage. By means of interviews, Pearlin obtained information about the social status of both the husband and wife's parents (ascribed status), as well as about the importance of status to the respondents, some of the details of their married life and the amount of marital stress they experienced. In this way, he was able to relate a social system variable, namely inequality of ascribed status, to stress reactions via psychological mediation.

Perlin's findings include three points of importance to us here. First, status inequality by itself did not produce stress in the marriage unless the higher-status partner considered status striving highly important (the psychological mediator),in which case that partner felt cheated. Second, status inequality resulted in subjective stress by disrupting four aspects of the couple's interaction, namely, reciprocity, exchange of expressiveness, exchange of affection, and the sharing of values. Third, in partners for whom status was important, status inequality created a lack of affection in the relationship, and inadequacy both in the communication and in the sharing of values.

Pearlin summarizes his findings as follows: "...people who marry partners of lower status while valuing status advance-

ment are relatively likely to form daily marital exchanges marked by disaffection and a sense of loss and these conditions, in turn, contribute to stress." And further: "To account for marital problems and marital stress, it needs to be recognized first of all that status inequality by itself is of little or no consequence. The importance of such inequality to marital problems and marital stress depends on the meaning and value that one attaches to it." (p. 356)

We can see in this research not only the mixing of levels of stress analysis but also how going back and forth between the social level and the psychological level clarifies what is happening. For example, the social variable, status inequality, does not by itself create stress; it only does so in those individuals for whom status striving (the psychological variable) is important. Without taking into account the psychological mediators accounting for individual differences (e.g., individual values, appraisals), we can see that the social variable would not be as useful as a predictor of stress in marriage. Moreover, in order to adequately understand the stress reactions that are generated in individual marriages, it is also necessary to examine the interpersonal processes that generate it, such as the quality of the byplay between husband and wife. In a situation where the partners feel they have been cheated and with impairment of their sense of reciprocity, expressive exchanges, affection and sharing of values, partners then experience their marital relationship as stressful.

Also,this is not a one-way street, that is, from the social level to psychological. On the contrary, it would be equally inadequate to try to understand what is going on by looking solely at process and outcome at the psychological level without also viewing the social system matrix as a determinant. Thus the discovery that some couples experience stress in marriage as expressed in troubled reciprocity, expressiveness, affection and value sharing would not be meaningful if we did not also consider the social system variable, status inequality, that makes this stress comprehensible. By noting that the marital stress depended on social status inequality between the spouses, we have in effect embedded the stress reactions in the natural social context engendering the stress in the first place. Only those pairs where one spouse has internalized some of the values of that social context experience stress based on status inequality. As we have seen earlier, the relationship between the two sets of levels is not by any means automatic; quite the contrary, only attention to both levels simultaneously makes it possible to make sense out of what is happening.

The second example comes from recent research by Mason (1974) on endocrine profiles in monkeys and men under various physical stressor conditions including exercise, fasting, heat and cold. Mason sought to determine whether or not these divergent

physical stressors produce a general endocrine reaction (in the fashion suggested by Selye's General Adaptation Syndrome), or produce reaction patterns specific to each type of stressor. From our present perspective, the most interesting feature of Mason's research was his attempt to "clean up the independent variable," as he puts it. That is, psychological threat reactions are usually confounded with physical stresses, so it is difficult to tell what it is that is producing elevated adrenal corticosteriods.

In exercise trials with young human males, in fasting trials with monkeys, and in heat and cold trials with monkeys, Mason took care to minimize novelty, uncertainty, extraneous psychosocial stimuli, and discomfort, in short, to reduce as much as possible any psychological threats that might be engendered by the physical procedures in the laboratory. For example, by minimizing competitive concerns and avoiding severe exertion, the danger that young men would be psychologically threatened in the treadmill exercise experiment was reduced; and by providing the subject monkeys with a non-nutritive placebo, they were less likely to be upset when bypassed by their trainer who provided food to the control monkeys in the fasting experiment; and in the heat and cold exposure experiments, temperature changes were made very gradually rather than suddenly so as not to alarm the monkey subjects.

Under these conditions of minimal psychological threat, it was found that each physical stressor produced its own unique profile of hormonal change (eight hormones were measured), and there was no general or non-specific hormonal response to all stimuli despite expectations to the contrary based on GAS theory. Even more remarkable, when psychological threats were kept at a minimum, there was no elevation of the 17-hydroxicorticosteroid response except for the cold condition.

Here, as before, there has been a crossing over of two levels of stress stimulus analysis, the physiological and the psychological; only in this case, the mixing of levels had been hitherto inadvertent, the psychological level (threat) being confounded with the physiological (heat, fasting, cold exercise). Such confounding appears to have mislead previous researchers about the bodily response to various stress conditions.

The corticosteroid response appears to be particularly sensitive to psychological threat, which is often confounded with other, physical stressors (Lazarus 1966; see also Lazarus 1974). By systematically designing a study in which the contribution of each level can be separately distinguished, Mason seems to have substantially advanced our understanding of psychophysiological processes related to stress. The lesson to be larned is not only that intentionally mixing levels of stress analysis is desirable because it adds to our understand-

ing, but also that the inadvertent or careless mixing of levels (where the two are not differentiated by stimulus manipulation and/or by measurement) can lead to very serious confusion about what is actually happening.

In the literature on mass psychogenic illness there is confusion due to a lack of awareness of the differences between structural and process-oriented research, and of the relationships among the physiological, psychological and social levels of analysis. For example, in a recent review of mass psychogenic illness by Colligan and Murphy (1979) reference is made to a number of multileveled factors such as boredom (a psychological trait), physical stress (a physiological state or process, depending on how it is measured), interpersonal conflict (a social process), contagion (a social process), and a group of psychological and physiological reaction patterns including physical symptoms and subjective distress.

As we stated at the outset, structural and process-oriented research are both important in understanding mass psychogenic illness. However, care has to be taken to understand the nature of the variables being examined (structural or process), how to examine them, the information they can generate, and the place of the information in the whole schema. For example, as we pointed out in our other conference paper (Folkman & Lazarus), correlating sex and conditions of the work environment (e.g., organizational style) with illness outcome is a heuristic which can usefully narrow the focus of a study. However, these correlations do not tell us why some people become affected with mass psychogenic illness while others do not. To understand this, variables which shed light on psychological, social and physiological processes must be examined. Both forms of research are important; but the purpose of each style and the kinds of information they can generate must be clearly understood.

Similarly, intentional mixing of levels of stress analysis is desirable because it adds to our understanding. But inadvertent or careless mixing (where they are not differentiated by stimulus manipulation and/or by measurement) often leads to very serious confusion about what is going on because a simple, one-to-one relationship among levels does not exist, and the rules linking one level with another are rarely spelled out and clear.

Traditional psychological analysis and research tends to be normative in outlook, that is, it seeks principles that apply to all humans alike. That such a stance is often counterproductive has been well understood by clinicians concerned with individual adaptation. Comparing one person with another fails to take into account the actual circumstances under which a person functions and the variations within a person, a knowledge of which is essential to an understanding

of adaptional processes. Ironically, even personality research tends to be formulated from a normative standpoint. The effect of one or a few personality variables, usually assessed statically through single-occasion, trait-centered measurement, is measured on some consequent pattern of behavior, say, the occurrence of the symptoms of psychogenic disorder in an individual or group. This research style fragments the person into one or a limited number of traits, rather than attempting to capture the particular organizational properties (or facets) and functions that comprise a total individual. Even more to the point, such a style of research does not permit us to see the nature of the transactions between person and environment that are the necessary conditions for that person or group to react with a psychogenic disorder. The fragmentation of the person and the person-environment relationship is well recognized in the debates about idiographic versus nomothetic research styles (cf. Allport 1962).

However, such debates have missed a key point, that research need not be designed with either nomothetic or idiographic extremes in mind. In most epidemiological research, for example, a large number of cases are studied through survey methods to assess either personal or environmental causal antecedents of heightened illness risk, and we never learn the types of transactional processes that tend to generate the illness. What has been missed is that it is possible effectively to mix idiographic and nomothetic styles by studying transactions of various kinds in a modest number of persons across a variety of encounters.

The term "ipsative" has often been used to refer to the intraindividual perspective and "normative" to the interindividual (Broverman 1965; Marceil 1977; Lazarus, Note 1). "Ipsative" refers to many facets of the same person or, more to our purpose here, to how that person (or many such persons) functions (e.g., coping processes, sources of stress, areas of strength and vulnerability) in a variety of contexts or encounters. Research can be both ipsative and normative at the same time by reducing the N and studying the same persons from time to time or under various conditions. An ipsative-normative research style requires that we sacrifice the large number of (normative) cases needed for statistical significance when the source of variance studied is weak or one of many causal factors in favor of more intensive examination of the same persons or groups across occasions. Such designs, in which a heavy commitment is made to repeated measures on the same persons, requires--for the same cost--that we reduce the N across persons and increase it within persons (across occasions).

As we shall see in Folkman's paper, the person's pattern of coping is a key factor in illness and illness behavior, and in adaptational outcomes in general. If our concern is with

effective versus ineffective coping, or with the kinds of coping patterns that generate psychogenic disorder, we must be in a position to determine whether any given persons or group copes with a variety of stressful encounters in some consistent way, or uses effectual or ineffectual patterns in certain types of encounters but not in others. And we must learn how to analyze intraindividual data so that we can extract and compare meaningful patterns across diverse encounters and situational contexts, as well as from group to group. Suitable intervention designed to alter the person-environment relation in a way that lowers the risk of psychogenic disorder depends on precisely such knowledge. It is possible that the rules involved in such transactions vary from person to person, though there is some hope that we can find a shared set of transactional characteristics from which a common intervention strategy could emerge for a large group.

If one accepts this line of reasoning about stressful transactions and coping in individuals, then the term "mass psychogenic disorder" could be a red herring. That is, because large numbers of people fall ill with the same disorder, researchers in the field have become mesmerized by the contagion feature. Although contagion may be important, our hunch is that it would not take place unless it was based on a common set of transactional features or, put differently, common psychodynamics. The important element, then, would not be contagion--that would only create the numbers that imply high cost and social concern--but the particular, shared nature of the transactional process. It is our view that this process cannot be uncovered by searching for common personality traits, or common environmental (situational) elements, but only by discovering a common transactional pattern among those who display the psychogenic disorder. Contagion is a secondary social process which occurs only because of these common, or shared, transactional properties. Recourse to the concept deflects the researcher from a more powerful ipsative-normative research strategy in which each person is seen as undergoing a pattern of stressful transactions and/or a pattern of coping which generates the disorder within the same context and during the same time frame.

The key transactional processes involved in mass psychogenic illness cannot be identified without trying to reconstruct them in individuals who succumb to the disorder through in-depth study and comparing them to those who do not succumb. This calls for a rather different research strategy than has been typical. We must describe the social and psychological processes involved in each instance, not identifying separate person or environment variables, but looking for common forms of transaction.

As will be seen in Folkman's discussion of coping processes

in mass psychogenic illness, we think that stress and coping is a particularly strong candidate for causal honors. We need to discover the sources of stress to which the individual person with the psychogenic disorder is vulnerable, the pattern of coping generated, and the concurrent emotional states. The same questions must be asked about those who do not display the psychogenic disorder in the same work setting or context. Only then can our hypothesis that shared forms of transaction underlie the common symptomatology be evaluated.

In speaking of coping, it is worth a momentary digression to note that the older terminology, namely, mass hysteria, which seems to have been dropped in favor of mass psychogenic illness, implied something about coping which is now missing in the more neutral modern term. The term hysteria, which arose with Freud, implied use of a repressive mode of coping with a particular kind of stress emanating from unresolved Oedipal urges. Three troubling connotations probably led to its abandonment: First, that this was a disorder of women, with the denigration that this could imply, although it does appear indeed to occur preponderantly in women; second, the notion that the illness was tied to sexuality, specifically to the Freudian "family romance;" third, that its seeds were predominantly intrapsychic, so that one did not look hard at external or environmentally based causal circumstances. We are not so sure that the new term, which is pretty much devoid of specific meaning, is much of an improvement; after all, "psychogenic" only means having psychological causation, which we already are pretty sure of. What has been lost, we think, is the implication that there is a close link between the illness and particular psychodynamics, especially that implicating the coping process.

Another possibility that seems buried in the new term is that there are two kinds of psychosomatic ailments, one of the type Selye (1956, 1976) pointed to, "diseases of adaption", the other having a very different flavor, so-called "conversation hysteria", in which "wear and tear" or actual bodily disorder is not present, only a special kind of dysfunction. A prime example of disorders of the first type are ulcers; there is actual bodily disease, and it can ultimately become irreversible if allowed to progress too far. Prime examples of the second type (see Watson & Buranen 1979) include hysterical paralyses, anesthesias, paresthesias and hyperasthesias, fainting, dizziness, headache, blurred vision, paresis, chest pain, etc. The processes involved here are hardly well described by reference to stress-induced cellular disease at the site, but exclusively to something not yet understood happening in the central nervous system. In fact, a dose of the hypnotic drug sodium amytal will temporarily reverse the symptoms of the latter: While under the drug, paralysis, blindness, etc., disappear.

Moreover, research long ago demonstrated how to distinguish between actual tissue disorder and the hysterias (cf. Hilgard & Wendt 1933; Cohen, Hilgard, & Wendt 1933). The above issue also points to a role for specific coping processes in the etiology of the two kinds of psychosomatic ailments, including mass psychogenic illness, which is of interest to us here.

In any event, the hypotheses that shared forms of stressful transactions, including the coping processes used, underlie mass psychogenic illness is more in tune with Colligan and Murphy's (1979) concept of convergence than it is to contagion. We are suggesting that those who succumb to psychogenic disorder are struggling with certain stressful transactions at work, and trying to resolve them with a common pattern of coping. It is this shared struggle or transactional pattern that is the important feature rather than contagion per se. We should now proceed to the substantive theoretical concepts of our stress and coping formulation and attempt to show their relevance to the phenomenon of mass psychogenic illness.

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COPING THEORY AS APPLIED TO THE INVESTIGATION OF MASS PSYCHOGENIC ILLNESS

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One of the most interesting questions about mass psychogenic illness is why some people become affected while others do not. Investigations of this question have focused on characteristics of the physical and social environment, and on state and trait characteristics of the person that may render him or her susceptible to psychogenic illness (cf. review of Colligan & Murphy, 1979). There is a missing link in these investigations, however, namely, an examination of the ways people actually cope in the problematic setting. Evidence from the laboratory (cf. reviews in Lazarus, Averill, & Opton 1974; Lazarus & Launier 1978), from clinical settings (e.g., review in Holroyd, in press; Weisman & Worden 1975; Janis 1958; Cohen & Lazarus 1973) and from work settings (e.g., Howard, Rechnitzer, & Cunningham 1975) indicates that coping is a critical variable mediating the relationship between stressful events and somatic, psychological and social outcomes. A systematic study of the ways workers actually cope in the work setting should shed light on the question of why some people are more susceptible than others to mass psychogenic illness.

That there has been little coping research in psychogenic illness is not surprising, for despite the widespread importance attributed to coping in virtually every area of the behavioral and social sciences, little is known about coping processes, especially in adequately functioning populations. There are three major reasons for this state of the art. First, many investigations are motivated by an interest in the adaptive demands of highly unusual situations such as doctoral examinations (Mechanic 1963), specific illnesses such as polio (Visotsky, Hamburg, Goss, & Lebovits 1961) or cancer (Weisman & Worden 1975), tramas such as burns (Andreasen, Noyes, & Hartford 1972), or disasters such as war (Grinker & Spiegel 1945). The purpose of these investigations is to understand coping that is adaptive and maladaptive in the specific situation of concern.

A second set of investigations is motivated by an interest in unusual groups of people such as those exhibiting some form of neurotic or psychopathologic behavior or who seem psychologically unhealthy, rather than ordinary people facing the normal trials and tribulations of living. Examples include studies of people who are phobic (e.g., Zone 1978), angry (e.g., Novaco, in press), or schizophrenic (Zubin & Spring 1977), and adolescents of outstanding competence (Silber, Hamburg, Coelho, Murphy, Rosenberg, & Pearlin 1961) or "self-adjusted" college students (Grinker 1962; Vaillant 1977). In industry, too, research on coping generally focuses on a select group, namely, managers (e.g., Hall & Mansfield 1971; Kobasa 1979). Although there is research on stress and stress responese in non-management workers (e.g., Frankenhaeuser 1975, there is an absence of information on the ways these people cope with their stressful environment. With the exception of

the work of Perlin and Schooler (1978) and our own current investigation, there has been little research about the coping processes of ordinary people who exist between the extremes of pathology on the one hand and "superperson" status on the other.

Third, research in coping has been severely limited by the measurement approach typically used. Most measures of coping are designed to asses a trait, that is a stable personality disposition (e.g., Byren 1964: Goldstein 1959). The purpose is to place the person somewhere on a bi-polar unidimensional continuum, and by virtue of this placement, to predict coping or other forms of behavior. Generally these continua reflect ego defense processes (cf. Menninger 1963; Haan 1977) --a somewhat narrow aspect of coping--anchored at one end by a "primitive" defense such as repression or denial and at the other by a "mature" defense such as isolation or intellectualization. Trait measures have not been very useful in predicting coping for several reasons. In personality research there is a growing consensus that knowledge of a trait is by itself not enough to predict behavior; there must also be information about the situation and its demands, and about the transactions between the person and the situation (cf. review in Pervin & Lewis 1978). Second, naturalistic description of the ways people handle real-life stressful events, show that coping is not a simple, unidemensional process. It is a complex, shifting amalgam of thoughts and behaviors, some of which may be defensive in nature, but others of which can be restitutive or oriented towards problem-solving and mastery. This complexity is not captured in unidemensional trait measures.

What has been needed is a strategy of measurement that is appropriate in a wide variety of situations having different adaptional requirements, based on "normal" as well as disturbed people, and capable of describing the actual range of thoughts and behaviors people use in coping. We have been developing such an approach in our investigation of the ways adequately functioning middle-aged adults cope with the ordinary as well as unusual events of their daily lives. Over the course of a year, one hundred middle-aged men and women are interviewed seven times about diverse, recently experienced stressful events. In each of the interviews subjects were asked to reconstruct what had happened, who had been involved, their thoughts and feelings during the encounter, its background, and how they coped with it. In the month intervals between interviews, subjects also filled out questionnaires in which they briefly reconstructed another recent event and indicated on a checklist the ways they had coped.

The coping checklist consisted of 68 items indicating various strategies used in the stressful encounter, including forms of direct action, information-seeking, cognitive coping

activity (to avoid, minimize, or positively interpret an event), and inhibition of action. At the end of the assessment period we had an average of 13.3 stressful events described for each subject, allowing us to examine the ways people actually coped with a variety of real-life situations, intra-individual patterns of coping across events, as well as normative comparisons based on situation and person factors. As noted in our other conference statement (Lazarus & Folkman; see also Lazarus 1978; Note 1; Lazarus & Cohen, Note 2), this style of research, describing the ways people actually cope with events they appraise as stressful, is essential to help us understand the processes mediating between the person and the environment. Although an important goal in coping research is eventually to be able to talk about coping styles, it is first necessary to get a process-centered data base from which such abstractions can be made.

Over and above process-centered research strategy, a theoretical framework is also needed to organize findings and to guide further research efforts in a systematic, meaningful manner. The theory of psychological stress and coping developed by Lazarus (1966; Lazarus, Averill, & Opton 1974; Lazarus & Launier 1978; Folkman, Schaefer, & Lazarus, in press) provides a useful framework with which to consider coping and its relationship to mass psychogenic illness. Because it may not be familiar to many readers, we describe it in the next section before relating it to mass psychogenic illness.

Our theory of stress and coping is a transactional, process-oriented conceptualization in that the person and the environment are seen in a dynamic relationship, each influencing and in turn being influenced by the other. The relationship between the person and the environment is constantly mediated by the way the individual appraises the environment and his or her relationship to it, and by coping. It is these processes of cognitive appraisal and coping that comprise and shape the experience of psychological stress and the reactions to it.

There are three types of cognitive appraisal, each of which serves in its own fashion to mediate the person-environment relationship: primary appraisal, which is the process of evalating an encounter with respect to its significance for one's well-being; secondary appraisal, which is the process of evaluating coping resources and options; and reappraisal, which occurs as new information is obtained from internal changes, changes in the environment (often from coping efforts), from further reflection, and from cognitive coping processes.

Primary appraisal is directed at the question "Am I okay or in trouble?" A situation can be evaluated as irrelevant or benign. Such appraisals are accompanied either by no emotion

or by positive emotion. If, however, an encounter is appraised as involving harm (or loss), threat, or challenge, there is psychological stress. Harm/loss and threat appraisals are accompanied by negatively toned emotions such as fear, anger or guilt. A challenge appraisal marks an event as carrying the potential for mastery or gain, yet the person's adaptive resources are still taxed and, as such, it too involves stress. Challenge is characterized by the presence of predominantly positively toned emotions such as excitement or pride, even though negatively toned emotions might also occur (cf. Lazarus, Kanner, & Folkman, in press).

Secondary appraisal evaluates the question "What can I do about the stressful encounter?" and therefore it involves judgments about the accessibility and efficacy of coping resources. Such resources include general health and energy, good morale, social support systems and help-giving institutions, money, problem-solving skills, and meaning systems on which people can draw in sustaining their efforts.

Throughout the appraisal process, primary and secondary appraisal each influence the other. The extent to which a person feels threatened, for example, will be influenced not only by what is at stake (primary appraisal), but also by evaluation of available coping resources with respect to the requirements of the taxing situation (secondary appraisal). The more a person judges his or her coping resources to be adequate for a specific situation, the less threatening it will appear. The resources on which people draw--health, energy, morale, social supports and money--have been discussed elsewhere (for reviews see Folkman et al., in press; Berkman 1977) and need not be elaborated here. What we mean by problem-solving skills and meaning systems as resources, however, may not be as clear, and since they are relevant to our discussion of coping and psychogenic illness, a brief description of them seems in order.

Problem-solving skills include the ability to analyze situations for the purpose of identifying the problem, searching for information, generating alternative courses of action, weighing alternatives with respect to desired or undesired outcomes, and selecting and implementing an appropriate plan of action (Janis 1974; Janis & Mann 1977). Such generalized, abstract skills ultimately must be actualized through specific acts, in themselves often skills, such as changing a flat tire, managing a boat in a storm, presenting oneself to a prospective employer, preparing for an exam, and so on. Some writers conceptualize skills in global terms, such as dealing with moral dilemmas (Schwartz 1970), with interpersonal problems (Shure & Spivack 1978), with emergency situations (for reviews see Appley & Trumbull 1967: Baker & Chapman 1976; Coelho, Hamburg, & Adams 1974; Janis 1958; Lazarus 1966), with role conflict (Katz & Kahn 1966), with marital conflict

(Levinger 1966; Parsons & Bales 1955) or with ambuiguity (Haan 1977). Others favor more concrete day-to-day actions such as one might use in a training manual (cf. Meichenbaum 1977; Rogers 1977; Yates 1976). Both levels of abstraction are important for understanding and for practical intervention.

Meaning systems (e.g., beliefs about oneself and the world) are also highly relevant to coping. An important belief concerns a person's self-efficacy (Bandura 1977). To disbelieve in one's own efficacy should generate passivity and disengagement, and at its extreme, should be associated with a sense of helplessness and hopelessness, which are in turn probably linked to depression (e.g., Engel 1962; Lefcourt 1976; Schmale & Iker 1966; Seligman 1975). This concept seems related to locus of control as discussed by Rotter (1966) and Lefcourt(1976), that is, the conviction that reinforcements are contingent on one's own behavior or, alternatively, are the result of external factors such as luck, chance or fate. The importance of self-efficacy as a coping resource has been demonstrated recently by Meichenbaum, Henshaw, & Himmel (in press) in a study of the cognitive strategies subjects used to tolerate the pain of holding their arm in a tub of cold circulating water (2? C). Tolerance of pain was not related to the cognitive strategies used to cope with the experience, but rather to the subject's general sense of efficacy in using the strategies required to cope with the stressor. Subjects who least tolerated the cold tended to see themselves as being overwhelmed and as having little ability to do anything to manage the pain.

Using a quite different approach, Stewart (1978) identified a set of beliefs having to do with the ways people organize experience, and demonstrated a relationship between these beliefs and the ways women cope with stressful events. She describes two contracting patterns of cognitive organization: self-definition and social definition. A self-defining woman is relatively indifferent to sex role norms (and perhaps to norms associated with other social roles), capable of emotional "distance" and objectivity, maintains a small number of intense, personal relationships, is active and interested in broad social movements and issues, and capable of vigorous instrumental activity. A socially defined woman behaves in accord with sex role norms, shows rather superficial emotional attachments to a large number of people, has little capacity to achieve detachment, is preoccupied with her own emotional and social life, and reveals a greater inclination toward expressive and instrumental activity. In a longitudinal study of 51 female college graduates, self-defining women were more likely than socially defined women to see their problems as external but the solutions as within their grasp (or internal), to see causes of problems as multiple, and to explain those causes clearly. In our terms, this type of self-related meaning or belief system should influence

primary appraisal through the values and commitments expressed in the definition of sex role, and secondary appraisal through its impact on the development and use of instrumental, problem-solving skills.

These are just two of many meaning systems relevant to coping much has been written, for example, about existential meaning systems, such as faith in God, fate, or some higher natural order, that enable people to create meaning out of damaging experiences (cf. Benner, Roskies, & Lazarus, in press; Dimsdale 1974; Frankl 1963; Bulman & Wortman 1977; Moos & Tsu 1977). Our purpose here, however, is to give examples of meaning systems that have particular relevance to coping with day-to-day demands.

A person's coping resources are usually not constant over time, that is, they are likely to expand and contract, some more erratically than others, as a function of experience and the requirements of adaptation associated with different events, styles of life, or periods in the life course. Furthermore, the presence in a person of a general resource such as problem-solving skills does not mean that the appropriate resource will be available or used at the time and in the transaction in which coping is required. Every major stressful transaction has its own task requirements: a problem on the production line makes different coping demands than problems with an aging parent. Even a person who is likely to be effective across a variety of circumstances will be confronted by situations for which the requisite skills are simply not there or where some hidden personal agenda or psychological deficit will prevent effective use of available resources.

Some resources such as health, energy, morale and beliefs in one's efficacy seem to facilitate the use of other resources such as problem solving skills or material goods. If, for example, one has learned problem-solving skills but does not have any conviction in his or her ability to use them, the skills will not be well utilized. Or if a person has a wealth of resources such as material goods, problem-solving skills and a general sense of self-efficacy but is low in physical energy and morale, the wealth of resources is less likely to be of much use. An interesting question concerns the mix of coping resources that one must have to meet the demands of various kinds of encounters, or that is optimal in diverse contexts.

Coping is determined by both primary and secondary appraisal. Primary appraisal is made in the light of the person's values, goals, commitments and beliefs, and generates information about the importance of the event to the person, its likely shape, and its potential implications for the person's well-being. This aspect of primary appraisal influences coping directly by affecting effort and mobilization. Generally, the

more something matters, the more a person will put forth and sustain coping effort (cf. Atkinson 1957; 1965; Klinger 1977). Primary appraisal also influences coping indirectly through its impact on secondary appraisal; the definition of the task demands provided by primary appraisal guides the memory search, retrieval and evaluation of coping options that constitute secondary appraisal (cf. Janis & Mann 1977; Miller, Galanter, & Pribram 1960; Folkman et al., in press). Secondary appraisal is thus shaped by primary appraisal and vice-versa. Convergence of the two appraisal processes determines the coping strategies a person uses and the effort he or she is willing to put forth.

Coping refers to "efforts, both action-oriented and intrapsychic, to manage (i.e., master, tolerate, reduce, minimize) environmental and internal demands, and conflicts among them, which tax or exceed a person's resources." (Lazarus & Launier 1978, p. 311) We are particularly interested in two types of coping because they have different but equally important functions. In the first, the person attempts to alter the troubled relationship with the environment by making changes in the person, in the environment, or both. We call this problem-focused coping. The second type is emotion-focused coping, which refers to efforts to regulate the emotional response to the problem. Emotion-focused coping includes cognitive and behavioral efforts to make oneself feel better through defensive activity, through expression of feeling, or through other "palliative" activities such as eating, drinking, smoking or talking with friends. These two coping functions have also been noted by others (e.g., Mechanic 1962; Kahn, Wolfe, Quinn, & Snoek 1964), and provide a useful way of describing broad coping functions in specific events.

Problem and emotion-focused coping can be used simultaneously, as when a worker experiences anxiety while confronting a supervisor and needs to deal with both the anxiety and the precipitating problem. They can also be used sequentially as, for example, when a worker tries to get him or herself "together", that is,emotionally under control, before an interaction with a supervisor that is anticipated as stressful. In both cases one coping function can facilitate the other. Throughout a stressful event, as emotions ebb and flow, a person may use now emotion-focused coping, now problem-focused coping, in some combination or other.

There are at least four basic modes of coping, namely, direct action, information-seeking, inhibition of behavior, and cognitive processes, that can each be directed at the problem or at the emotional response (Lazarus & Launier 1978). For example, direct action can be problem-focused, as in fixing a car when it breaks down, or it can be emotion-focused, as in drinking, taking tranquilizers, or practicing relaxation or meditation. Information-seeking can be used to provide

a basis for action directed at the problem, but it may also be used to bolster or confirm a decision that has already been made (cf. Janis & Mann 1977), thereby regulating anxiety. Similarly, inhibition of action can be applied to both the problem and the emotion, as can cognitive modes of coping. In the case of the latter, cognitive efforts can be directed at problem-solving or a palliation. Further, each of the coping modes can be oriented towards events that have happened, that are happening, or that are anticipated.

Whether coping functions are classified into broad categories, for example, according to whether they are emotion-or problem-focused, or in the specific modes discussed above, the point to remember is that we are not talking about coping "styles" or "traits", but of the numerous processes of coping that a person engages in throughout a stressful event.

Psychogenic illness can be interpreted as an outcome of a chronic stressful situation that has been mediated by inadequate coping, the form of coping influencing the symptom pattern (cf. Schafer 1954; Shapiro 1965; Menninger 1963). For coping to be successful, a person must: (1) control the situation instrumentally as effectively as possible so that he or she can move towards important goals or master obstacles; and/or (2) tolerate, minimize or accept damaging environmental pressures; (3) maintain value integrity and morale; and (4) keep the internal environment sufficiently close to homeostasis to maintain bodily health despite having to mobilize to cope instrumentally or with the emotional response (cf. Roskies & Lazarus, in press; Folkman etaal., in press).

Often a person does well in one area, but pays a price in one or more of the others. For example, a worker might decide to increase his or her production pace to meet a quota, thereby being effective instrumentally, but doing so at the expense of the family. This could cause anger or resentment towards the supervisor, guilt and/or loss with respect to the family, as well as fatigue. If similar transactions occur on a frequent basis, the continued mobilization necessary for both the instrumental response and for dealing with heightened emotions could lead to increases in hormonal secretions and eventually cause damage as described in Selye's (1946; 1976) General Adaptation Syndrome. Epidemiological research has also implicated mobilization in disorders such as hypertension (Syme, Note 3; Ostfeld 1967) and coronary heart disease (Roseman, Jenkins, Brand, Friedman, Straus, & Wrum 1975).

Although the work forces in which mass psychogenic illness appears are predominantly female, apparently more females than would be expected become ill. This suggests a sex difference in vulnerability and/or coping pattern. If coping is indeed related to somatic and psychological outcomes, as so much research indicates it is, (see earlier comments) then

a reasonable starting place for the investigation of coping processes in affected and non-affected workers is to ask if there are sex differences in coping processes in the work setting.

There is a widely held belief that men are socialized to be instrumental, analytic and goal-directed in their thoughts and behaviors, whereas women are socialized to be expressive, compliant, dependent and deferential (cf. Broverman, Vogel, Broverman, Clarkson, & Rosenkrantz 1972; Epstein 1971; Ginzberg & Yohalem 1966; Papanek 1973; Colligan & Murphy, 1979). If these beliefs are transformed into coping terms, we would expect men to engage in more problem-focused coping than women to engage in more emotion-focused coping than men.

Our findings (Folkman 1979) partially confirm these expectations. There were sex differences, but only in the use of problem-focused coping, and not in the use of emotion-focused coping. The sex difference in problem-focused coping, however, is limited to stressful events taking place in the work context; we found no sex differences in the use of problem-focused coping in the contexts of health and family. In other words, the obtained sex differences do not reflect a general disposition on the part of males to use more problem-focused coping than females in all settings, but only in the work setting. Here we have a good example of the importance of a transactional perspective of type of person (male-female) in commerce with a type of setting (work vs. health and family).

A similar pattern emerged when we examined sex differences in coping according to how situations were appraised. After reconstructing each coping episode, our subjects were asked to indicate which of the following statements best described the incident:

In general, is this situation one:

1. That you could change or do something about?
2. That must be accepted or gotten used to?
3. That you needed to know more about before you could act?
4. In which you had to hold yourself back from doing what you wanted to do?

There were no sex differences in the use of these appraisals. Males, for example, did not appraise situations as ones which could be changed more often than females, nor did females appraise situations as ones which must be accepted or gotten used to more than males, as is usually assumed to be their sex role disposition. However, there were sex differences in the use of problem-focused coping as a function of such appraisals. Once again, men used more problem-focused coping

than women, but only in situations that must be accepted, or in which more information was needed. And there were no sex differences in the use of emotion-focused coping as a function of the different kinds of appraisals.

These findings are particularly interesting in light of the kinds of problems that occur in work settings where mass psychogenic illness has occurred. These work settings are characterized by one or more of the following: highly authoritarian management styles, production pressures and unwanted overtime, physical stressors, boredom, and lack of two-way communication with peers and supervisors (cf. Colligan & Murphy, 1979). Such descriptions suggest that many workers are faced with work-related problems over which they perceive little personal control. Given our findings that men use more problem-focused coping at work and in situations appraised as having to be accepted than women, and given that men are less affected by mass psychogenic illness, it is possible that the tendency to use problem-focused coping helps in some way with the psychological stress generated in these settings. Or, conversely, the disposition of women not to rely as much on problem-focuses coping in the work setting increases their vulnerability or is reflected in somatization.

That men use more problem-focused coping than women at work, however, does not necessarily mean that the differences in coping are a function of sex. It may well be that women more often than men are placed in jobs where there are fewer opportunities to exercise personal control. If this is the case, differences in coping are not so much a function of sex as they are of the job. This is an important distinction, for measures to reduce susceptibility to mass psychogenic illness would differ in their emphasis if the determinant of coping is primarily the person (sex) or the environment(job). In the former case, for example, the emphasis could be on teaching skills for problem-focused coping. In the latter, the emphasis would be on changing the environment so as to provide more coping options for the worker.

Even though women are apparently more vulnerable to mass psychogenic illness than men, there are women who remain non-affected. If problem-focused coping is related to psychogenic illness, then it is reasonable to ask whether non-affected women use more problem-focused coping than affected women. Findings reviewed by Colligan and Murphy (1979) indeed suggest this may be the case. They cite Kerckhoff and Back's (1968) study in which affected workers were more reluctant to discuss work-related problems with their supervisors compared to non-affected workers. Kerckhoff and Back also found that affected workers received little "information"support prior to an outbreak, although it is not clear whether non-affected workers received more.

With respect to emotion-focused coping, we found in our own research that situations appraised as having to be accepted called for significantly more emotion-focused coping than situations about which it was felt that something could be done. Given the chronic nature of many of the problems of industrial workers, the role of emotion-focused coping as a mediator of psychogenic illness should be taken seriously and studied systematically.

A variety of specific coping activities are subsumed under emotion-focused coping. For example, a person can avoid thinking about a problem, minimize its importance, or engage physically in a diversionary activity (cf. Gal & Lazarus 1975). One can also engage in emotion-focused coping by expressing feelings, using social supports to gain sympathy or understanding, interpreting an event in a positive light (Lazarus, Note 4), or engaging in wishful thinking. In our study, people used an average of fourteen emotion-focused strategies in each stressful episode they described. Although we have not yet analyzed the use of specific emotion-focused strategies or their patterns, the fact that they are so common as a class suggests that people actually use multiple modes in handling everyday stresses. The interesting question, then, is whether affected and non-affected workers differ in the combinations or mix of coping activities and modes they use.

It is essential to look at the combinations of coping modes individuals use at different times during an encounter, in effect, as stressful encounters unfold. Earlier, we identified four criteria which must be satisfied for coping to mediate a troubled transaction successfully. We also pointed out that coping that satisfies one criterion may be exacting a price in one or more of the others. In the same manner, a coping mode or pattern that satisfies one or more criteria at one stage of an encounter may not do so at another. For example, at the outset of a stressful encounter, denial can reduce psychological stress, but if its use prevents the person from taking action such as seeking needed medical care (cf. Katz, Weiner, Gallagher, & Hellman 1970), it can prove costly in the long run. On the other hand, at an early stage in some stressful encounters (e.g., disabling illness), denial is often valuable by buying time until strength can be mobilized for rehabilitative efforts (cf. Hamburg & Adams 1967). In the work setting, a person's feeling of frustration and anger can be reduced by minimizing the significance of troubling, boring or noxious aspects of a job that cannot be changed, thereby reducing psychological stress. In the long run, however, this can lead to alienation (Kanungo 1979; Dubin, Champoux, & Porter 1975; Hall & Schneider 1972; Hall, Schneider, & Nygren 1970) and be costly both instrumentally and to morale and value integrity.

In all likelihood, there are multiple sources of stress in the person and situation that contribute to mass psychogenic illness and, indeed, research has thus far focused on their identification. However, throughout these papers we have said that psychological stress refers to a relationship between the person and the environment. We emphasized the importance of two processes, cognitive appraisal and coping, as mediators of this relationship. We think an exploration of coping process, looking at coping patterns, their outcomes, and the manner in which they might be tied to symptom development cessation, hold particular promise for research in mass psychogenic illness. However, until a transactional perspective is adopted, and a process-oriented methodology employed, the processes through which sources of stress eventuate in somatic outcome are not likely to be understood.

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QUALITY OF THE WORKING ENVIRONMENT: PROTECTION AND PROMOTION OF OCCUPATIONAL MENTAL HEALTH

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1. Holistic approach to work environment and health

The past few decades have seen a rapidly growing awareness of the impact of the physical and chemical work environment on man's physical health and wellbeing. Much less attention has been paid to possible effects of such environmental factors in terms of mental stress and the ensuing mental and psychosomatic ill health. Even less interest has been devoted to corresponding effects of psychosocial factors at work, for good or bad.

To us, physico-chemical and psychosocial factors must be of equal concern, as they all focus on aspects of health and wellbeing in total man's interaction with total environment. What should interest us is the general question of whether present conditions and present trends in occupational environmental change do, in fact, pose a threat to human health or survival, to wellbeing and to the quality of life, and if so, the magnitude of the problem and what we could do about it (cf. Levi, 1977 and 1978).

2. Principal goals of working life

However, before discussing this in any depth, it may be worthwhile to review the principal goals of working life, not primarily in terms of economy or technology but with regard to satisfaction of human needs such as health. Health has been defined as not only an "absence of disease or infirmity" but also "a state of physical, mental and social wellbeing" (ECOSOC, 1946). The promotion of health in this broad sense (one could equally well refer to it as "quality of life") must be one of the principal aims of all social activity, both central and local, including the important sector of working life and its conditions.

Admittedly, working life also provides income and an output of goods and services. But these things are not ends in themselves; they can only be means of assuring optimum physical, mental and social wellbeing for the greatest possible majority and of promoting their health, development and self-realization. Thus, working life can satisfy human needs directly, through opportunities for creative and stimulating activities and social contacts, as well as indirectly, through the provision of income.

3. What workers have a right to expect

A good summary of what people have a right to expect from working life is given in a recent resolution from the International Labour Conference. It states:

- "that work should respect the worker's life and health; this is the problem of safety and healthiness in the workplace;

- that it would leave him free time for rest and leisure; this is the question of hours of work and their adaptation to an improved pattern for life outside work; and

- that it should enable him to serve society and achieve self-fulfillment by developing his personal capacities; this is the problem of the content and organization of work.

In essence, this means that work is made the servant and not the master.

However, this is not what work amounts to for hundreds of millions of workers all over the world.

4. Recent changes in working life

Generally, recent changes in working life in economically developed countries can be summarized as a trend towards:

-increasing mechanization and automation;
-increasing proportion of shift work;
-increasing proportion of large enterprises;
-increasing anonymity, division of labour, and heterogeneity of life;
-increasingly distant relationships between worker and management, worker and worker, worker and union officials, and producer and consumer.

All this has led to

-increasing advantages for the young, healthy, highly adaptable, well-educated high-performers, and corresponding disadvantages for those who are physically, mentally and socially handicapped.

In developing countries the picture looks different. Here, a series of dramatic transitions is taking place from agriculture to manufacturing, and from the latter to mass production. This is more or less the same sequence through which today's developed countries passed in the 19th and early 20th century. However, the velocity of these transitions differs greatly. Developing countries are undergoing the same process in one-tenth or less of the time taken by the developed countries. In addition, and this is the tragic thing, they tend not to learn from history and thus to repeat all the mistakes, even adding a few new ones.

5. Benefits - and costs

Although the economic benefits of present trends in working life are obvious, the satisfaction of some human needs has led to frustration of others. In addition, there is an increased over or underutilization of human abilities and a discrepancy between human expectations and perceived outcome, in short, a misfit between man and his environment.

Discussing various types of person-environment discrepancies some people argue that humans are adaptable. They are, indeed, but you can equally say that they are deformable. Deformation becomes the price.

This price can be expressed in terms of reactions on three levels:

- psychological reactions (e.g. anxiety, malaise, low-spiritedness, depression, feelings of helplessness, disrupted somatic awareness, defensive reactions of various kinds);

- behavioural reactions (e.g. increased consumption of alcohol, tobacco; drug abuse; prolonged sickness absence, suicide);

- physiological reactions (e.g. an increased activity of adrenal cortex and medulla; psychosomatic diseases).

It would seem to have been firmly established that considerable psychological, psychosomatic and social problems occur among workers in response to person-environment discrepancies at work. On the other hand, we lack evidence of who is at particularly high risk to such problems, how grave the problems are, and how they could be prevented.

Nor do we possess any reliable data concerning the components of the total situation at work and outside it which are particularly stressful, the extent to which they are amenable to environmental adjustment and/or psychosocial support and, if they are amenable, at what "price".

6. Common phenomena

But we know that all these reactions are exceedingly common. In a random sample of all Swedes who are gainfully employed, 37% found their work mentally stressful; two out of three said it was characterized by rush and tear; 18% felt it to be monotonous; and 14% reported that they felt mentally exhausted on arriving home from work (Institute for Social Research, 1978).

These figures, moreover, are only part of the story. As already emphasized, there are the other indicators of a bad person-environment fit at work and elsewhere, such as alcoholism, suicide, and mental and psychosomatic disorders. These, too, are very common phenomena. Although a causal connection between them and occupational stressors is not clearly proven, it is highly suspected (cf. Levi and Anderson, 1975; Levi and Alaby, 1979).

7. Man-made environments can be adapted - by man, for man

To all this, some people say that technological and economic development cannot be changed. In claiming this, they seem to forget that work environments and processes are man-made. It follows that they can also be adapted--by man, for man. The question is not if, but how.

As Trist (1974) indicates, we have two choices: The first is to leave the vast bulk of jobs that must still be done in manufacturing and service industries in the dull and monotonous state in which they exist at present, accepting the need to work as the primary curse, a necessary evil which we must endure; the principal aim then becomes to reduce the amount that has to be done, shortening both working hours and the working week, while maintaining a scale of pay which enables satisfactions to be sought elsewhere.

The alternative would be to redesign jobs and organizational forms so that the majority rather than merely the privileged few can do work which is meaningful and fulfilling, while maintaining a high level of performance.

8. Meeting people's psychological requirements

Such a redesignation would imply meeting people's psychological requirements at work other than those specified in a contract of employment (such as wages, hours, safety, security of tenure, etc.). Six such requirements have been listed (Emery, 1963) that pertain to the content of a job and which must be met if a new work ethic is to develop:

- the need for the job to be reasonably demanding in terms other than sheer endurance, and to provide at least a minimum of variety;
- the need to be able to learn on the job, and go on learning;
- the need for some area of decision-making that the individual can call his own;
- the need for some degree of social support and recogni-

tion in the workplace;

- the need to be able to relate what he does and what he produces to his social life;

- the need to feel that the job leads to some sort of desirable future.

9. Organizational consequences

Translated into organizational terms, measures to satisfy these needs appear to involve two fundamental ideas (Walker, 1974). The first is concerned with a reversal of the trend towards the division of jobs into smaller and smaller elements, each to be performed by a single worker, and its replacement by a trend towards putting together the various functions in a meaningful, integrated whole.

The second concerns modification of the hierarchic organizational structure of the enterprise by arranging for workers to work together in small face-to-face groups, which have a good deal of autonomy, and whose supervisor no longer gives detailed orders, but sees that the group has the resources it needs and handles the group's relations with the rest of the enterprise. Or, as put by Trist (1974), under the principle of self-regulation, only the critical interventions, desired outcomes, and organizational maintenance requirements need to be specified by those managing, leaving the remainder to those doing.

None of these measures (nor any others) provide a patent solution to all the problems. There are no patent solutions. What is good in one respect for one individual need not be good in another respect for another individual. What we have to do is to find out what is good (and bad) for whom, in what way, when and under what conditions. A considerable amount of interest, of course, will then focus on measures which are more or less generally effective.

10. Four cardinal principles

To be effective, such measures should be based on four cardinal principles.

- First, on a holistic (overall) view of man and environment, i.e. equal and integral consideration for physical, mental, social and economic aspects.

- second, on an ecological strategy, i.e., consideration of the interaction between the entire individual and the entire environment (physicochemical and psychosocial)

and of the dynamics of the entire system,

- third, on a cybernetic strategy, with continuous evaluation of the effects (physical, mental, social and economic) of different working environments and of changes in the same, continuous feed-back, to decision makers and the public, and a continuous adaptation and reshaping of the working environment by all concerned in the light of these various types of experience,

- fourth, on a democratic strategy, giving the individual the greatest possible influence over his own situation and direct, efficient channels of communication to the various decision makers.

These are the strategies. The tactics to improve person-environment fit at work concern the adaptation of environmental demands and opportunities to workers' abilities and needs. Accordingly, our action must focus on several types of environmental factors, which can be summarized as follows.

11. Satisfaction of various types of human needs

Satisfaction of physiological needs and of security and safety is often referred to as "hygiene". Attention to such factors is important but not enough. Other environmental factors are connected with satisfaction of other human needs. These include, e.g., salary, number and quality of human contacts, supervision, security at work and physical factors in the work environment (cf. Baneryd, 1976). Unless these aspects are satisfied to some degree, dissatisfaction and unhappiness will occur. As soon as they have been satisfied, additional "improvements" will not yield any further substantial increment to satisfaction.

Another class of factors is referred to as "motivational", i.e., related to needs such as ego-experience, self-esteem and self-appreciation. These include advancement to more stimulating tasks, appreciation of work well done, to be allowed to complete a task, to take responsibility, inherent qualities of the task itself, etc. These factors have a positive influence on the involvement in, and the experience of satisfaction from, work.

It follows that, to be successful, an occupational mental health protection and promotion program must be comprehensive and take into account all types of human needs and their satisfaction through environmental adjustments, i.e., by creating a good person-environment fit.

For such a fit to be good, the general organization of the work process is no less important than hygiene factors such

as noise and illumination. Organizational factors (cf. Brannstrom et al., 1975) are those primarily concerned with:

- the workers knowledge of how his part of the work contributes to the finished whole;
- the independence and responsibility of the workers;
- their social contact and collaboration with other employees.

12. Part of a meaningful whole

The first point means that the work task should be seen to constitute a meaningful whole or at least form an essential part of a production process which is understandable and meaningful to the worker in terms of the relationship between his own contribution on the one hand and the ultimate goals of the production process on the other.

This could be achieved by letting the worker take part in the preplanning, realization and control of his task. Further, one might wish to consider the amalgamation of several fragmented pieces of the work process into meaningful sequences.

The worker should further be allowed to utilize and develop further his knowledge and skills through the work process and thereby enable him to take over increasingly qualified work tasks.

This can be facilitated by choosing the right man for the right job and by rotating workers who so wish between different tasks and positions. Job rotation and job enlargement can also include an increased participation for the worker in the planning and control of his own achievements. It should further include options - but not necessarily demands- for recurrent training and education.

One of the most important ways of learning from experience is through feed-back from the environment. In working life, every worker ought to know the result of his endeavours, i.e., the quality and quantity of his achievements. This is true not only for the individual worker but for the work group as well.

This can be achieved in several complementary ways. One is to allow continuous contact with the preceding and following link in the production chain. Another is to allow the worker to control and check his own results. To this might be added regular reports and evaluations from his supervisors

about the quantity and quality of his performance. There should also be some opportunity to discuss with fellow workers and managment how work is and should be carried out (cf. Brannstrom et al., 1975).

13. Independence and autonomy

Our second point concerns independence and autonomy. This does not mean that every single worker should do whatever he likes, whenever he likes, in any way he likes. This would be to go to one extreme. The other extreme, where man is turned into a passive tool, is equally unfavourable. Work is made much more stimulating, rewarding and effective if it allows an optimal degree of participation; e.g., in planning the work, influencing the method and work pace and location of pauses.

This can be accomplished by making the individual worker or work group responsible for carrying out the job, for breaks and for temporary absence from work. Information on the progress of the work will then allow each unit to make the necessary decisions. This might mean that the work pace should be left to the individual or the group to decide. Responsibility and power should be delegated as much as possible to those directly concerned with the production process.

14. Social contact and collaboration

With regard to our third point it seems clear that one of the great advantages of working life is that it creates the social context for contact and collaboration with other human beings. These are basic human needs. Accordingly, conversation and contact between employees should be made a necessary part of the production process instead of being eliminated. Whenever possible, work should be planned in such a way that its various components could be allocated to relatively small groups of, say, 4-8 people. Speech and eye contact and collaboration between the group members should be encouraged. If this turns out to be impossible for practical reasons, human contact should be facilitated at least during breaks, and opportunities for friendly contact with supervisors and management should be promoted.

15. Needs satisfaction an integral part of production process

In these ways, satisfaction of human needs is made an integral part of the production process. Working life becomes humanized and, simultaneously, more smooth and efficient, with mutual solidarity and social support. Extreme, competi-

tive individualism is counteracted and a more communal approach is favoured. This may include tangible support, i.e., concrete assistance such as helping a person complete a task or sharing resources. Another type of support that is favoured is psychological-emotional support, as in lending a sympathetic ear, reassuring, or demonstrating concern and care (Pinneau, 1976). For these and other reasons, the mental health of the executive is of particular importance not only in itself but for its effects on other workers. For example, a mildly depressed manager cannot generate enthusiasm in his staff; and an irritable supervisor might be unable to provide any support and instead create stress for those under him. Man's most important environmental factor is--fellow man.

16. Deprofessionalization; sharing of responsibility

It is often taken for granted that responsibility for the humanization of working life is a matter for a few selected professionals. In our opinion it is more logical, and effective, to see this responsibility as given to each and everyone, although one naturally also has to allocate some specific responsibilities. It is in the interest of all concerned that the work environment is maintained or made optimal for all possible respects of improve person-environment fit and, consequently, health, wellbeing and productivity.

Much can be accomplished through two universally available tools of measurement and intervention, namely listening and speaking. People know best where their own shoe pinches and they should be allowed and encouraged to tell their own story. Wherever possible, they should further be allowed and encouraged to make their own adjustments with regard to the immediate environment, if this can be done without harm to others.

17. Implementation now - but with continuous evaluation

Some of these principles and measures are well supported by present knowledge and should be implemented immediately on a general level. Others may be good for some people, under some circumstances and in some respects, but not beneficial or even harmful for others. A third category concerns well-meant but untested, and therefore speculative approaches.

To implement such a program without delay but at the same time safeguard the public and increase the programme's effectiveness and efficiency, we need continuous evaluation. This will allow us to learn from experiences, our successes and mistakes, and thus enable us to correct and improve the program.

18. Acquisition of new knowledge

In many instances, however, our present state of knowledge simply does not allow rational health action even if combined with evaluation. To close such critical gaps, new knowledge must be acquired; i.e., before modifying, and evaluating our modifications, we need to identify three classes of phenomena, namely

- o high-risk situations (e.g. with regard to shift work, piece wages, sensory overload);
- o high-risk groups (e.g. within populations such as working mothers, migrant workers), and
- o high-risk reactions (e.g. various forms of psychological, physiological, and immunological dysfunction).

Whenever possible, our research strategy should combine

- o key hypothesis testing (basic research), with
- o evaluation of health actions (applied research), and
- o collection of quantified information on the interrelationship of various parts of the man-environment ecosystem (Kagan, 1978).

To provide the necessary data, research projects can often be carried out in three consecutive steps:

- o problem identification with survey techniques and morbidity data;
- o longitudinal, multidisciplinary intensive studies of the intersection of high-risk situations and high-risk groups as compared with controls.
- o controlled intervention, including laboratory experiments as well as therapeutic and/or preventive interventions in real-life settings (e.g. natural experiments; interdisciplinary evaluation of health action).

Some examples of the application of such strategy and tactics to the area of shift work; health and wellbeing and of their results are given below.

19. Examples of research on shift work, health and wellbeing

One source of problems in working life is the possible temporal misfit between man and his environment, arising from the altered rest/activity patterns required from subjects with work hours placed outside the conventional daytime range, e.g. shift workers (Akerstedt et al., 1977).

Such a misfit rests on the assumption of a conflict between endogenous, i.e. self-sustained biological rhythms in various psychophysiological functions on the one hand, and environmental demands on these functions on the other.

In our first series of studies we therefore wanted to look into the possibly endogenous properties of the temporal variation of some important physiological and psychological functions, i.e. their persistence in the absence of the normal time-cues. To this end, more than 100 normal healthy volunteers of both sexes were exposed to three days and three nights of continuous work. In spite of the strict standardization and equalization of environmental stimuli, the diurnal rhythms persisted throughout the vigil, with pronounced decreases in adrenaline excretion and in body temperature, shortfalls in performance and increases in fatigue ratings taking place in the small hours (Levi, 1972; Froberg et al., 1975 a & b; Frober, 1977; Akerstedt and Froberg, 1977).

This persistent oscillation under conditions of constant activity, light, food intake, etc. clearly indicates that there is an obvious endogeneous component in this oscillation (Akerstedt and Levi, 1978).

As a second step, several hundred shift workers presumably exposed to such conflicts between endogenous rhythms and environmental demands were studied with health questionnaire techniques. The results showed higher frequencies of sleep, mood, digestive and social disturbances among the shift workers than among the day workers. The complaints about wellbeing reached their peak during the night shift.

In a third step, we studied physiological, psychological, chronobiological and social reactions in response to the introduction of three weeks of night-work in habitual daytime workers. We found that, although the endocrine system does indeed adapt to the environmental demands induced by shift-work by "stepping on the gas" to keep awake in night-time and "slowing down" in the day to allow for some sleep, the usual one-week cycle does not suffice for a complete adaptation of turning night into day and vice versa. Not even three weeks of continuous night work are enough to cause an inversion of the circadian functions; the original circadian rhythms flatten out but still persist. In addition, switching from habitual day work to three weeks of night work is accompanied by increases in a number of indices of physiological stress and social problems in the workers and in their

families (Theorell and Akerstedt, 1976).

In a logical fourth step, night work was not intorduced but instead eliminated. A group of steel-workers was kept on a continuing 3-shift work whereas a comparable experimental group was switched to 2-shift work, everything else being held constant and equal. In a one-year follow-up we were able to demonstrate that the change to work schedules without night shift brings with it an improvement in physical, mental, and social wellbeing. The control group who remained on their habitual 3-shift work schedule did not change or even deteriorated with respect to wellbeing (Akerstedt and Torsvall, 1977, 1978).

20. Research and health action on the shop floor level

Accordingly, our program - not only nationally but also on each shop floor and in each office - must be as follows:

First, to identify the type and extent of the problems present, e.g. incidence of mental and psychosomatic disorders, absenteeism, alcohol abuse, labour turn-over, dissatisfaction and social unrest.

Second, to identify psychosocial and physical/chemical environmental correlates of the various problems

Third, management, labour unions, occupational safety and health workers, and authorities must consider, together with the workers concerned, which of these environmental influences are likely to be of greatest casual importance and at the same time accessible to change, and which of such changes are feasible and acceptable to all concerned.

Fourth, to change the work environment in the manner described above on a small and experimental scale to evaluate benefits and side effects in all possible terms, and on the basis of this to decide what kind of change can be implemented on a wider scale.

Fifth, such a wider application should be continuously monitored, evaluated and modified as occasion arises.

To be efficient, the whole procedure 1-5 must be carried out with "feed-back" of results and with full participation and understanding of all concerned.

21. Summary and conclusions

In summary, occupational mental health protection and promotion should be seen as a means to adapt work environments

to man's capacities, needs and expectations and to help man to find the job and the environment best suited to fit his personal requirements. The work environment should not only avoid exposing man to noxious physical, chemical, biological and psychosocial influences, but also promote health, well-being, development and self-realization. In addition, and most important, in all these endeavours man must be seen not as a passive object of benevolent expert supervision and action but as an active and respected subject, who often knows better than the experts where his shoe pinches and who is willing and able to make his own considerations and decisions.

When we treat machines badly, technocratic protest is immediate and strong. Human beings have been, and to a considerable extent still are, treated badly in working life and elsewhere. Protest is not very loud and response is far too weak. Now that we have rational guidelines for humanizing work, there should be no further delay in protecting human flesh and blood and - last but not least - the human mind.

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OCCUPATIONAL HEALTH AND THE DEVELOPING COUNTRIES

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Many publications are available on the impact of industrialization on health. Studies have also been carried out on health problems associated with psycho-social factors at work in the developing countries. However, the psychosocial factors at work in the developing countries have hardly been investigated.

It would not be totally correct to draw a parallel between stress encountered by workers of the industrial revolution of the 18th century, and those of workers in the developing countries nowadays. There may be similarities with respect to changing physical work exposure, causing occupational diseases. But the industrial revolution differed from the present industrial movement in developing countries in the fact that mechanized processes were a gradual development associated with innovation, thereby, probably providing a better opportunity for psycho-social adaptation.

The rapid introduction of complex work methods and tools in developing countries is associated with psycho-social stress. The main problem is that of adjustment to change.

To a large extent, workers in the developing countries have managed to adapt to the rather strange psycho-social environment of mechanized labor. They have been required to change from the quiet and more intimate rural life to the noisy speed of factories, from the traditional dependence on natural processes and manual labor to standardized production, precise timing, and dependence on energy, and from the old identification with land and crops to the impersonal environment of the machine.

They are likely to move away from their friendly work associates to work with busy strangers, different management, and a different hierarchy.

On the other hand, the benefits expected mainly from material regards may play an important role in motivation to meet such challenges.

In many instances, however, adaptation may fail, either because of too much stress or personal susceptibility, or both, resulting in desertion of work or in psychological or psychosomatic disorder.

In many of the developing countries, absence from work and labor turnover are rarely high. While these may be closely related to economic and employment problems, cases were found in association with failure to adapt to an alien psycho-social environment, and relatively large industrial undertaking.

When a new steel mill was first established in Burma in 1967--and although most of the newly recruited workers were familiar

with iron processing, with blacksmiths and private shops the annual labor turnover rate exceeded 200 percent in the first two years, and the mill was forced to slow down on production mainly because of the shortage of labor. The main causative factor was the difficulty in adaptation to the style of work, where workers were responsible for only limited parts in a line of production. Communication was difficult, and personal identification, despite the rewarding pay, was limited.

Another example, which illustrates the impact of the fear of imminent work hazards to health and life is shown in some of the South Korean mining operations. The isolation of mines in remote areas, coupled with reports on irreversible pneumoconiosis, and mortality in underground collapses, resulted in almost 300 percent labor turnover per year, despite such attractions as high pay, housing, free schooling and free meidcal care to workers and their families.

These illustrate inevitable breakdown of homeostasis, where escape from mounting psycho-social stress was difficult. Naturally, there is a gradual process of psychological disturbance that may appear in various manifestations of discontent and unsatisfaction.

In this respect, the experience of maladjusted or unhappy industrialized societies is not different. And while compensation may be found in these societies in habituation to alchohol or drugs or in behavioral anomalies, this has not so much been observed in the industrializing countries. Or at least there has been no evidence of serious occurrence.

There are also no data on the magnitude of prodromal manifestations of psychological disorder and frank psychosomatic disease. However, the striking nature of the latter led me to observe, and partly study, psycho-social factors associated with episodes of psychosomatic disorders among workers in rapidly developing societies.

In the early 60's, I had been notified of a relatively high prevalence of peptic ulcer in a glass factory in Egypt.

In investigating the problem, it was found that in addition to diet and minor physical stress in the work environment, the main associating factors, with what proved to be a significantly high incidence of acute ulcer, included a faulty management of payroll and strenuous provocation in seeking an increased output of individual glass blowers, who were the key workers in the overall production of that factory.

Very recently, the director of the Industrial Health Unit in Singapore has reported incidents of mass psychogenic illness in that country (see Phoon, present proceedings).

The preliminary investigation of the problem, which temporarily caused complete suspension of work in the affected factories, showed that there are several psycho-social stresses, such as job insecurity and monotonous tasks, lengthy hours, low pay. And these were factors common among the workers who also shared a common ethnic origin.

The positive effects of health associated with work should not be overlooked in referring to psycho-social stresses of working lives. As a matter of fact, it is my impression that many employers in developing countries are keeping with long inherited humane traditions.

The close and more intimate work relations exist in small work establishments, which employed physical hazards in these factories, that absenteeism from work is lower, and that this has been attributed to the comparatively better work relations in the small enterprise.

In many large establishments in developing countries, religious traditions are upheld, and the workers are allowed to go for prayers, in the temple on the factory's premises.

There are also examples where the workers themselves have actually induced changes in complex industrial processes to suit cultural and traditional habits. And that such changes did not only make them happier in what would otherwise be a stressful situation, but have also increased industrial productivity.

Systematic studies of psycho-social factors in the work environment in the developing countries are much needed and highly recommended with a view toward:

"Number, one, putting under control at an early stage of industrial development those factors that may lead to excessive strain and avoid the mistakes of older industrialized countries; and number two, identifying those psychosocial factors that play a role in health promotion and education of employers and health practitioners on utilization of such factors for betterment of health of the working populations in developing countries."

EVIDENCE AND INFERENCE IN RESEARCH ON MASS PSYCHOGENIC ILLNESS

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INTRODUCTION

At one point in his illustrious career, Niels Bohr announced that "Every sentence I utter must be understood not as an affirmation but as a question." It is tempting to make that view the theme of a paper on mass psychogenic illness. For what is known about the phenomena is rudimentary and often ambiguous. Moreover, I am not an expert in the area. One ought to be a little suspicious that such denial attempts to deter criticism. So I claim expertise in examining evidence on social and psychological problems and on their solution. It is as a methodologist that I testify, and I thank Michael Colligan for the opportunity to do so.*

This paper's main purpose is to discuss the quality of evidence and the link between evidence and inference in research on mass psychogenic illness. The direct resources exploited here are published case studies of the phenomena, and the excellent reviews by Sirois and Markish, each listed in the bibliography. This includes cases of illness in schools, factories, and hospitals, in the U.S. and abroad. Though the emphasis is on criticism, I do not mean this to be adverse commentary. The cases were compiled with vigor and astonishing skill in dramatically unsettled institutions. Rather the attempt is to identify what is admirable practice and what is not, to clarify evidentiary problems which have not been resolved, and to offer suggestions for their resolution. The scientific muses invoked generally concern access to information, especially for the sake of competing analysis, the quality of evidence, and reasonableness of inference.

Colligan & Murphy's (1979) definition of mass psychogenic illness is adopted here: the collective occurrence of physical symptoms and related beliefs among two or more persons in the absence of an identifiable pathogen. More formal definition, considered later, would attend to the ideas that onset is rapid, affecting several people in a short time period, that effects are temporary, and that symptoms of affected people are similar. Laughlin's (Kutach, 1965) theory of conversion is also adopted as a vehicle for understanding the phenomena at the individual level. His conception is that conversion is an unconscious process by which intrapsychic conflicts which otherwise give rise to anxiety, instead secure symbolic external expression. His typology of conversion types is based on the kinds of symptoms exhibited: somatic; physiologic; and psychologic. The adoption of definitions is tentative. Most of you recognize

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better than I that they are flawed, and one aim of this examination is to better understand how they might be improved.

SYMPTOMOLOGY AND MEASUREMENT

Several features of reporting on symptom patterns make it unnecessarily difficult for the researcher or lay reader to understand the phenomena. This includes published descriptions of the uniformity, verifiability, and recurrence of symptoms, and the influence exercised by observation itself on reported symptoms.

UNIFORMITY

Understanding whether each individual in an affected group exhibits the same symptoms is, of course, fundamental. To the extent that symptoms are quite similar, it becomes easier to establish the disease entity, to discriminate between psychogenic illness and other maladies, to chart the progress of the phenomena, and perhaps to control it. It is often very difficult to determine uniformity from published papers however.

Ebrahim (1968), for instance, appears to suggest that each of the 300 East Africans discussed in his case study were afflicted with running mania. But it is not clear whether each exhibited symptoms to the same extent, whether each also exhibitied other symptoms typically, or whether there were remarkable or atypical cases. Whether all cases were afflicted uniformly by headaches, burning eyes, nausea, weakness, etc., in Colligan and Stockton's (1978) study of a garment manufacturing plant, Stahl and Lebedun's (1974) work in the data computation center, and Chew, et al's (1976) investigation of the Singapore factory, is similarly ambiguous. Knight, Friedman, and Suliante (1976), on the other hand, provide slightly more evidence that uniformity in some respects and heterogeneity in others ought to be expected. Hyperventilation preceded the salient symptom, fainting, among some girls in the Louisiana school episode. Headache and dizziness preceded a faint among others; behavior akin to petit mal or catatonic posturing preceded still others. Colligan et al (1979) manage to provide the most information by condensing frequency of symptoms encountered in an electronics plant episode, but do not take the next natural step to identify cluster of symptoms.

We ought to know where regularity appears in episodes of mass psychogenic illness and where it does not. The implication then is that uniformity ought to be regarded as a legitimate topic of investigation during an episode. And for the sake of an interested scientific community, the regularity or

lack of it ought to be reported. Neither action appears to be common now, judging from published reports.

Whether such information is useful will depend on environmental insult. I gather from Sax (1979) that some toxic substances, such as lead, produce a variety of symptoms depending on the individual who ingests it. But other substances generate uniform symptoms. Consequently, a number of symptoms and regularity in their appearance is unlikely to be an adequate indicator of mass psychogenic illness in all cases. Examining cases on the basis of uniformity is still warranted if we want to establish utility of the information.

RELAPSE

Whether symptoms reappear spontaneously still appears to be debatable, perhaps because so few follow-up studies have been undertaken. Sirois (1974), based on his thoughtful examination of 78 cases of mass psychogenic illness, observes that "...relapses in the same person are not the rule." (p.15). He is a bit inconsistent in that his tables show that 19 out of 51 accessible cases involve relapses. That relapse does occur, that there may be multiple relapses, and that it is possible to document them reasonably well is illustrated by the Chew et al (1976) work in Singapore. Phoon's remarks at this conference suggest that reoccurrence of episodes in certain Singapore factories, is not uncommon.

A more complicated problem concerns an individual's relapse following attempts to investigate or treat the problem. The return visit of Knight, Friedman, and Sulianti to the Louisiana school in which girls blacked out in a mass episode was followed quickly by other blackouts. It is as reasonable to regard some of these as relapses as it is to regard them as a product of the intervention, a problem considered in a bit more detail later.

The obvious implication is that one ought to monitor systematically for relapse after a major attack. The monitoring would include individuals in a group previously affected, and the institution in which an attack occurred. The second, less obvious, implication concerns relapses which are plausibly caused by visits of the investigator, as in the Knight, Friedman, Sulianti case, or by attempts at treatment, as in the boss's visit to hospitalized women in the Colligan-Stockton work on a garment manufacturing plant. In particular, if such visits do indeed provoke relapse, and pathogens, toxic chemicals, allergies and the like are not present during visits, then it becomes more reasonable to diagnose at least the relapse as psychogenic and less reasonable to attribute the problem to environment. Indeed, the diagnostician may wish to introduce such visits deliberately in the interest of

a crude and cheap test of the hypothesis that the illness is psychogenic. The third implication is that relapse rates deserve attention because they are part of the symptomology. Because they help to identify the disease entity, and to build theory on immunity and sensitivity, and to identify the effects of ameliorative action, relapse rates are essential.

IRRELEVANT VARIATION IN OBSERVED SYMPTOMS

The quality of information about symptoms is influenced by the way one makes observations and by the setting itself. Factors which can plausibly degrade quality are not often recognized explicitly in published reports. Their appearance in other kinds of action research suggests that they ought to be considered at least in investigations of mass psychogenic illness.

It is, of course, possible that the mere observation of an individual's behavior will produce real changes in that behavior. So, for example, one of the interesting implications of the Knight, Friedman, Sulianti (1965) report on blackouts among Louisiana school girls is that active investigation may have produced attacks. The authors present a nice chart to illustrate the incidence of attacks as a function of time. The reader cannot help but notice a second cluster of attacks following a quiet period and the reentry of investigators at the school. The notion becomes more plausible when we recognize arguments that similar inflation of symptoms occur with other types of medical examination. So-called labile hypertension, for example, registered by temporarily elevated blood pressure, may be largely a function of nervousness produced by a visit to the physician's office (see Kolata, 1979 on Sokolow).

The Knight et al case is the only data-based illustration of the sort I've been able to find. The problem may have occurred in other cases of mass psychogenic illness, e.g. the Singapore Factory episodes described by Phoon. But pertinent data do not appear to have been published. What makes the problem more intriguing is that related problems are alleged to be a problem in other action research. For example, Kerckhoff, Back, Miller's (1976) June Bug investigation was said to have been delayed two months because plant managers were apprehensive about the possible effect of interviews. School districts, for instance, have refused to permit sociologists to ask students questions about drug abuse on account of fear that such questions would generate an interest in using drugs. Desegregation researchers have been asked to terminate studies ostensibly on grounds that squabbles and fights would be produced by their merely asking questions. Such claims are occasionally self-serving: consequently they may be entirely misleading. But we know so little about the way investigation affects behavior in disruptive settings, we cannot afford to ignore the matter.

It is also true that, irrespective of behavior, the information recorded during an investigation can be influenced by the views of the investigator. Knight, Friedman, and Sulianti, for example, also report that psychiatric interviews were informative. But from their test, it appears that interviews were not blind. The lack of blindness and their preconceptions could have led to psychiatrists' judgments that victims of attack were more "sexual," more willing to talk about illness, and so on, than were unaffected students. The problem of course, is not confined to investigation of mass psychogenic illness. It affects all mental health research and medical research at least in principle. Whether the bias induced is large and, if so, how bias can be reduced are important.

I know of no precise, easy way to gauge the effect of on-site observation on individuals' behavior. But I suppose that in this, as in other, settings that calm and less obtrusive observation will be less provocative than excited and more obtrusive approaches. Which one it is ought to be reported. I would also expect that such influences could be assayed in formal field experiments,but evidently there has been no attempt to examine feasibility of such tests.

Blind interviews and ratings are a natural device for avoiding biases attributable to prior views of interviewer and rater. The problems of assuring blindness are mechanical, but I should think that it is feasible to obtain blind observations in interviews if topics are prescribed and some topics are temporarily prescribed.

The Setting

The disruptive settings in which interviews often take place can affect the quality of observations. Knight, et al find no interesting results of their Rorshach testing and, assuming that such tests are informative in ideal settings, the failure to find anything interesting may be attributable to low reliability of such tests in disruptive settings. Similarly, the extraneous variation introduced to observations on the Minnesota Multiphasic Inventory, perhaps into electro-encephlagrams, and other measures reduces the likelihood of establishing interesting differences between an affected group and an unaffected one. That is, a failure to find such differences doesn't mean there are none; it may mean that test results are insufficiently reliable.

The possibility that results will be degraded by random variation suggests that conditions ought to be controlled so as to decrease external disturbance, e.g. testing an individual in isolation. Regardless of whether testing conditions can be controlled, the conditions of testing ought to be reported: they are a real part of the test itself. Further, the internal consistency and related forms of reliability

for some psychological inventories, rating forms, and physical measures can be computed from the results obtained on site. And it is not unreasonable to try to obtain test-retest stability in the interest of understanding temporal reliability of measures. Both types of statistic, internal consistency and stability, can help to determine whether further analyses are justified. There is not much point to even rudimentary comparisons of, say, affected and unaffected groups if the probability of finding differences is degraded drastically by random noise in the observations.

During this conference, Dr. Markush pointed out that it is also possible that an episode of mass psychogenic illness may itself affect test results systematically. For example, an individual's score on the hysteria scale may be inflated following an episode. Inferences about the personality of individuals who are susceptible to attack would then be misleading: the score is high because of attack, not necessarily because the trait was present in greater degree prior to the event. If indeed measured personality dimensions are not robust against situational disturbance, if the trait is not as durable as IQ, then this problem, is I agree, severe. Establishing susceptibility would be virtually impossible. On the other hand, knowing that disruption does influence scores on such inventories, if that is in fact what occurs, is itself scientifically interesting.

It is not unreasonable to entertain a similar theory for social measures, especially if observations are made well after an actual episode of mass psychogenic illness. For instance, the Kerckhoff, Back, and Miller (1965) data on sociometric patterns appears to have been collected some two months after the June Bug episode. It is possible that friendship networks found at that point were influenced by the attack. That is, individuals who had been affected might feel some additional kinship as a result, and some friendships may evolve or be strengthened as a consequence of common experience. If so, inferences about the role of social networks in mass psychogenic illness will be misleading.

In the absence of any other information, several options are worth considering. It may, for example, be possible to select inventories on the basis of their robustness against extraneous influence such as an episode of psychogenic illness. The criteria for selection should be empirical, and this suggests a program of formal experimentation on the influence of high and low anxiety situations on scores. Some work is possible in the laboratory, and given the advances in the state-of-the-art in social experimentation, some research in the field may also be possible (Riecken, et al 1974). The generalizability theory available for design of such experiments is elaborate (Cronbach et al, 1972).

A second option is to develop new tests which, in theory and practice, provide accurate estimates of the level of traits despite environmental variations. I do not know if it is possible to do so. I do believe that some new latent trait models should be explored. For instance, so-called Rasch models might be pertinent in that tests constructed in accord with the model may yield better estimates of trait level independent of the systematic pressures engendered by a disruptive setting. For applications in achievement testing, where the objective is to estimate true ability independent of item difficulty, see Lord and Novick (1968).

A third option hinges on the idea that measuring level of a trait after an event is useless at best and misleading at worst. Comparing level of hysteria to a norm is unlikely to be helpful, for example, because there are so many unknown and unknowable differences between the norm sample and the group involved in an episode of mass psychogenic illness. Measurements taken before an episode, in the interest of establishing individual susceptibility, are more desirable, but of course no such measures are typically available. The implication is that routine monitoring systems be set up to obtain such information regularly. Implementing such a system would be difficult, I recognize. But if the objective is to predict outbreaks, to understand the effect of outbreaks, and to assay susceptibility without formal side experiments of the sort described earlier, I see no other way to meet the objectives.

A fourth option is applicable at least to social network inventories and is deceptively simple. One may elicit retrospective reports about friendship patterns before the episode, for instance, as well as reports about contemporary friendships when delay in eliciting information is unavoidable. The problem here, of course, is that the quality of retrospective reports may not be sufficient to justify analysis. It is reasonable to develop a rough idea about the validity of such data from other investigations in which it is possible to obtain friendship information during and well after the episode. A discrepancy would be presumptive evidence on the degree to which memory failure, the attack itself, and other factors influence the accuracy of retrospective reports. And such evidence is invaluable to the investigator who must rely solely on the latter.

It would be presumptuous to declare that problems of the sort described here are important in every case. I do not know enough about investigations of mass psychogenic illness to make that judgment. It is far from presumptuous however, to regard the problems as plausible sources of irrelevant variation in the data, and to ask how they have been recognized at least and recognized at best in a case at hand. It is reasonable to ask that the problems be considered in published reports, even if

they've not been resolved. Certainly, investigators ought to report controls on reliability of observation, report reliabilities where possible, and take reliabilities into account when searching for group differences.

I surmise that some problems, notably identification or development of inventories and interview methods which are robust against environmental disruption require a program of research. Moreover, there is fine precedent for methodological field research on interviewer effects, on methods of rating, and other related topics in the U.S. Census Bureau's work. Advances are being made as well in research by the National Center for Health Statistics. Cooperative university-based laboratory research in the same spirit is worth considering to understand how current observation schemes can be improved.

Methods for Research in Disruptive Settings

Several speakers expressed concern about the difficulty of doing high quality research amid the chaos characteristic of mass psychogenic illness. As I understand, there are difficulties in gaining admission to the organization, in interviewing individuals upset by attack, and in interviewing nonvictims who may view victimization with some hostility. Time for the research is scarce. There may be resistance to research in general and psychology or psychiatry in particular.

It is puzzling that such difficulties and their consequences are not better documented, since in the end, they affect the quality of information collected about the outbreak. The Colligan et al paper (1979) is an exception in this respect. They report that questionnaires could not be distributed in accord with a specified sampling plan, hence response rate is about 50%. The implications for making even simple judgments about who exhibited what symptoms are serious, judging from the sensitivity and analysis of count data (below). Only some victims were given medical tests and that too may produce flawed conclusions about the nature of the event.

There is small comfort in knowing that similar problems are encountered in social research on prison riots, campus protests, picket lines and unions, street gang violence, desegregation squabbles, natural disasters, and other disruptions. The difficulty may be dramatic in these instances. But it is chronic (Boruch & Cecil, 1979; Beals, 1969).

We are justified in regarding such difficulties as a legitimate arena for methodological research and development, primarily because they influence the quality of data. But not much serious intellectual attention has been given the problem by academic or government researchers. For mass psychogenic illness, it is not unreasonable to consolidate the experience of re-

searchers who have been successful, noting for example, the importance of gaining access through personal contact with managers, developing rapport through conventional devices, minimizing the time demanded of respondents, minimizing the provocativeness of inquiry, accommodating typical criticism, and so on. Manuals which emphasize evidence and methods may also be helpful. Developing a reward system, including paying victims and especially nonvictims, to compensate for their participation in their research is likely to help in this arena of research as it does in others (Riecken, et al 1974). It is doubtful that science or methodology will be served well by ignoring the opportunity to advance the state of the art in this arena. But I know of no concerted effort to do so, or to link understanding in this arena to what we know from research efforts in other disruptive settings.

MEDICAL AND ENVIRONMENTAL TESTING

REPORTING RESULTS OF MEDICAL TESTING

Early investigators report that medical tests were undertaken on at least some victims of psychogenic illness. But they rarely report the specific tests, nor are reports which contain test results cited. So, for example, Knight, Friedman, and Sulianti (1965) report that some students "were subjected to thorough physical examination, including lumbar puncture". (p. 858). But the authors of this fine report still fail to specify the tests and the number of students involved in each. Similarly, Stahl and Lebedun (1974) refer to "excessive blood and urine tests of women" in the computation center case, but they provide no detail. To his credit, Ebrahim (1968) reports that laboratory studies were carried out for 32 patients of 300 affected by running mania in Mbale, and that cerebrospinal and blood examinations were carried out in 17 and 15 cases respectively. But the specific tests are not specified. Colligan, et al (1979) are exceptional in reporting on tests of 28 victims in an outbreak at an electronics plant. But neither Ebrahim nor Colligan et al tell us the basis for selecting individuals for tests: some victims in each case underwent no medical tests and the rate of nonparticipation in tests is unspecified.

Reporting such tests in journal articles or citing research reports in which details are given, is important of course, for several reasons. First, if the tests were pertinent enough, in some physicians' judgment, to have been done, they are pertinent enough to be reported. Knowing that specific tests yielded no evidence of a particular malady is information. We know what causes of symptoms can be ruled out of speculation, and we narrow the territory of the unknown. The second reason for reporting lies in the idea that we should

have a better understanding than we have now of why we follow false leads, why our initial investigations or tests may be misleading, unnecessary, or silly. Understanding the traps in simple testing for gonorrhea, for example, led Mausner and Gezon (1967) to avoid relying on vaginal smears alone, to seek cultural confirmation, and ultimately to their development of a remarkable case study. More generally, at least some medical tests undertaken during an epidemic may be imperfect, useless or harmful; and we ought to know about them as well. The third reason for more adequate reporting of medical results is that such data can be important in secondary analysis by independent researchers. If at Time 1, malady A is ruled out by such tests, at Time 2 one might rule out malady B based on the same test or regard B as a possibility based on the test and new information.

REPORTING ENVIRONMENTAL TESTS

The inquiring reader of reports on epidemic hysteria encounters two problems bearing on environmental monitoring. First, the nature of environmental tests is often unspecified. And second, the nature of samples taken to identify toxic environmental influences is unclear.

More specifically, though many analysts report that environmental tests were done as a consequence of an outbreak, few report the specific nature of tests or cite original reports on the topic if they exist. So, for example, Colligan and Stockton's (1978) cite Meyer's observation that "massive environmental sampling" of air, water, and noise was undertaken, but no details are provided. Despite the attention paid by Stahl and Lebedun (1974) to noise of a diesel engine in their case study of the data computation center, no detail on the nature of noise or tests for gas is provided. In other cases, claims by others that "samples of ambient air in different quarters, of solvents, chemicals, and glues, even of drinking water and food in vending machines" are not supported by reference to specific tests. Colligan et al (1979) illustrate what can be reported in brief, describing when tests were initiated (day of second incident), duration of testing, and the chemicals for which tests were made.

The reasons these tests ought to be specified, despite their negative results, are identical to those cataloged earlier for medical reporting. They include: ruling out competing explanations and limiting the unknown in the case at hand; identifying plausible explanations for epidemic which are incorrect; and permitting later secondary analysis of the case.

Herrik (1979) reports that reliability of air screening is such that readings are within +25% accuracy. The observation does suggest that reports of environmental tests routinely provide information about their reliability, just as one ought to

provide estimates of reliability in reporting results of using personality inventories, questionnaires, and the like. The reliability of environmental tests no doubt also depends on circumstances, so an experimental testing program for such tests, analogous to one outlined earlier for softer measures, is warrented, especially if estimates cannot be obtained in the unsettled context of an outbreak.

ENVIRONMENTAL SAMPLING

Very few reports in the social science literature provide any real information on the environmental sampling designs employed or employable during investigations. By "sample" design here I mean the plan which specifies when and in what areas or volumes sampling units will be drawn. I do not mean what also appears to be a vernacular definition of sampling technique used by Herrick (1979), i.e. the physical device used to capture the unit of gas, liquid, or solid. I expect that most samples are actually opportunistic, e.g. someone points out an area in which a peculiar odor was detected and the area is assayed once or twice. I understand from Herrick (1979) that obtaining a sample likely to exhibit extreme properties, in the interest of upperbound estimates is not uncommon, e.g. a rafter sample for certain precipitants. Perhaps this is the only way one can sample in such cases. But to label an event as taking a sample without further specification is, at best, frustrating to scientific readers. We know the word sample is used promiscuously and become suspicious in the absence of additional information. And so the nature of the sample ought to be specified.

More important perhaps is the absence of interesting work on statistical sampling plans for such cases. I can find no textbooks or quidelines which help answer questions such as: How often should one take a sample? What sample design ought to be exploited for assessing physical areas, or volumes of moving air? What kinds of systematic biases in sampling or in testing a sample at any given time can be expected? Answering such questions is reasonable given that odors emerge and dissipate, noise varies in intensity, and so on, and static tests are unlikely to be very useful. Developments along these lines are warranted if they have not already been made.

DIAGNOSIS AND MISDIAGNOSIS

SIMPLE THEORY OF DIAGNOSIS

Implicit in Sirois' (1974) remarkable review of epidemic hysteria is a basic theory of diagnosis and misdiagnosis. In the abstract of course, a theory of this sort is important for any social or medical research. As a matter of practice, no

explicit theory is treated or exploited in reports on mass psychogenic illness.

Sirois' simple theory implies first that establishing the nature of the phenomena entails recognizing that similar symptoms may generate two different forms of explanation. That is, the diagnosis may be that the problem is largely medical in origin e.g., there are toxic influences on physical well-being. Or, the diagnosis may be that the illness is, in this case, contagious hysteria. Either declaration might be wrong in interesting ways. Consequently, we ought to recognize the possibility, based on crude theory of how errors occur.

Clearly, the problem may be psychogenic in origin, despite declaration that it is organic. Sirois cites the McEvedy and Beard (1973) research to support the notion that some disease is iatrogenic in character and based more on physicians' expectations than biomedical evidence. Recall that the research involved outbreaks of neuro-myasthenia: symptoms similar to those exhibited by victims of poliomyelitis were also exhibited by individuals without the latter disease, and were regarded as a clinical syndrome when indeed the problem appears to have been psychogenic. Barnes' (1977) review of worthless surgery is at least as pertinent. He reminds us that ptosis was characterized early in this century as a condition in which the position of the internal organs was abnormal. The abnormality was thought by surgeons to be the cause of a wide variety of symptoms which incidentally are quite similar to those exhibitied by victims of contagious hysteria. Kidney displacement was alleged to give rise to neuroticism, back pain, vomiting, and so on. We know now that ptosis is not an organic problem, that surgery was unwarranted, and that diagnosis and etiology were nonsense.

This class of misdiagnosis implies that we ought to catalog stereotypical errors of the sort, and estimate base rates of misdiagnosis. The former would help to avoid problems in actual cases I should think, and the latter would help to characterize misdiagnosis better statistically. More generally, we do not know what the base rate for accurate medical diagnosis in institutional settings is, and that itself would be interesting. For if estimates are constructed, the figures would provide us with a rough upper bound on incidence of hysteria. That is, the complement of the rate of accurate diagnosis includes a rate of psychogenic illness and the probably larger rate of misdiagnosed medical problems. I suspect that crude estimates could be based on contemporary research in physicians' second opinions, supported now by the National Center for Health Services Research, and perhaps on contemporary archives, e.g. insurance company records.

If the declared diagnosis in a particular case is contagious hysteria, this too, may be wrong in interesting ways. I know of

no good estimates of the rate at which hysteria is misdiagnosed at the individual level, but the statistic would be helpful in forming opinions about rate at the institutional level. There are lots of anecdotes to support the idea that misdiagnosis is possible. Young women, for instance, occasionally complain that their physicians regard some of their symptoms as hysterical when, they maintain, the problem is biomedical. Sirois relies on more reliable data to argue that epilepsy and rheumatic fever are examples of disease which are "...triggered by or mimic collective symptoms". Recall also the remarks at this conference, reminding us that diagnoses of acute schizophrenic reactions among textile workers have been made when in fact the origin of the symptoms are psychogenic but not tied directly to hysterical conversion. Sirois provides no examples; nor do I know of any research on the topic.

This implies that one ought to catalog chemicals, gases, etc. partly on the basis of whether they produce symptoms similar or identical to those encountered in mass psychogenic illness. Sirois observes that the latter is benign in having no permanent effects. I should think that it is possible to classify toxic agents then partly on the basis of indurability of their effects. Then one may identify those which produce similarly diffuse symptoms, and exercise their influence very quickly.

AUGMENTING DIAGNOSTIC THEORY

Much of this implies that at least one other category of outcomes be adjoined to the ones which Sirois proposed: unknown. That is, one admits the possibility that evidence, for either medical or psychological diagnoses, is simply too puny. This category may be abused: it is possible for a manufacturer, employee, or physician to take refuge in a declaration of ignorance. But I suspect that the pressure to diagnose the malady as something with a name is sufficiently high to make declaration of ignorance difficult. Lawsuits doubtless increase that pressure. The point is that theory of diagnosis should include criteria for determining when data are not sufficient. Rhetoric to sustain that judgment and to make it palatable to a public which is often unwilling to take "I don't know" for an answer needs to be articulated.

As a practical matter, formal recognition of dissenting opinion on the sufficiency of evidence and the accuracy of a diagnosis is justified. That is, union, management, or other interest groups are invitied to comment on the adequacy of diagnosis and the commentary is published as part of the main report. The practice does not guarantee diagnostic accuracy, but it may help to make competing explanations explicit. The latter are a basis for developing competing diagnostic categories.

CORRUPTION OF DIAGNOSIS

Judging from the remarks of other speakers at this conference, the diagnosis of contagious hysteria or mass psychogenic illness is in danger of corruption.

That is, the diagnosis provides a way of shifting the blame for illness from the institution, which may produce hysteria-inducing stresses, to the individual. It is in more danger of becoming a label for malady in which origins are simply unknown. That is, the real chemical or pathogenic causes of symptoms remain undiscovered despite our efforts, and the exclusionary portion of the definition of mass psychogenic illness (no pathogenic origins) invites the diagnosis. Or, the diagnosis may become stylish, as in the case of neuromyasthemia, the need for hysterectomy, and the like. The problem, of course, is not confined to this arena of research. Artificial inflation of body counts in war (Knightley, 1975) of policy arrest and conviction rates in new administrations (Campbell, 1975) and the like appear frequently. If Campbell's theory of corruption of social indicators is at all accurate, the inflation occurs whenever the social system adopts the indicator for administrative purposes.

GENERAL THEORY

Few methodologists are well informed enough about mass psychogenic illness to dictate changes in the way the theoretician views the problem, and I am not among those few. Nonetheless, it would be careless to ignore some theoretical features of work on the topic which are particularly nettlesome. A few of those bearing on diagnosis and evidence are discussed briefly in the following remarks.

Gender Differences

The emphasis on susceptibility of women to contagious hysteria seems to be more historical accident than conscious planning and overadvertised as well. Moreover, it conflicts with evidence which, though admittedly scarce, is provocative. Contagious hysteria in wartime, alluded to by Sirois (1974) and by Elesh at this conference, fainting in blood donation stations and during military vaccination, some epidemic suicide, some outbreaks of violence, and the like, appear to the lay observer to fall legitimately into the category of mass psychogenic illness, and do involve men. The lay observer may be wrong in interpreting this as contagious hysteria and concluding that risk is notable, if not high for men. But I've seen no interesting attempt to show why the interpretation is incorrect. There are also, as we know, one or two men involved in some industrial cases, and at least one well documented case involving boys (Sirois, 1974). It is doubtful that by ignoring gender similarities we will be in any better position

to understand what may be more fundamental differences between victims and nonvictims.

Analogues and Relations

Investigators of contagious hysteria often cite the after-dinner yawn as a commonplace example of contagious behavior: one yawn appears to trigger others. The polite cough during a play or concert is also mentioned frequently.

It is not clear that the mechanism involved in such cases is identical or even similar to one which results in mass psychogenic illness. It does seem to me that, on the evidence of appearance alone, the extensions ought to be explored. There are, for instance, rather closer analogues. There is some evidence that when a single individual faints in a blood donation center, during military vaccination, and so on, the event triggers other fainting. That evidence ought to be consolidated and examined for its relation with theory in this area. The rather more upsetting triggers for contagious (or at least similar) behavior are no less interesting. The vomit produced by one individual in a group in turn is likely to produce nausea among at least a few other members, and may produce other symptoms as well. Blood, a sharp cry, a thump on the head, and so on may also produce sympathetic reactions. The analogues suggest a rather larger framework of behavioral phenomena than is typically considered in research on contagious hysteria. And more importantly, I think, at least a few are experimentally manipulable. Pennebaker's (1979) reports are particularly interesting in this respect. It <u>is</u> remarkable to know that perception of temperature, coughing, scratching, sneezing, and so on can be produced among some by merely coughing oneself, or by merely talking about an itch. And both reprocibility of the phenomena and its relation to more violent behavior are important to understand. I would suppose that Pennebaker's experiments can be extended to explore a large response surface, using both social and chemical triggers in ways which are not dissimilar from research strategies in marketing, product testing, and chemical engineering. I suppose also that field experiments such as the one's he has described can help to illuminate currently debatable topics, such as the susceptibility of women, the role of isolates and friendship networks, and the temporal sequence of events.

Synthesis and Quality of Evidence

It is obvious, once said, that theory-oriented synthesis of research on contagious hysteria should take the quality of evidence into account. That is one ought to recognize that validity and reliability of information varies, in developing taxonomy of symptoms, progress, probable causes, and competing explanations.

Sirois (1974) and Markush (1973) recognize the principle, and that is admirable. But the Sirois review and others are less satisfying in that quality of evidence is defined in practice solely by accessibility. To be sure, one should consider some reports of illness separately because reliable information is inaccessible. This is a fundamental, albeit limited, standard. It is reasonable on cases characterized by high quality information rather than solely on cases with evidence of mixed quality. This will, of course, reduce the number of cases that can be dealt with, and perhaps make generalization more difficult. But there is lots of precedent for educing misleading theory from faulty data. This includes medical research, where poor experimental design has led to grave errors in judgment about the effectiveness of surgical care (Barnes, 1977), in economics where poor control over quality of information leads to flaws in policy (Morgenstern, 1963), and so on.

Taxonomy

Sirois (1974) appears to be among the few to have tried classifying processes of epidemic hysteria. The classes proposed, based on his review, include: explosive; explosive, preceded by isolated cases; cumulative (slow chain reaction); rebound (massive episode preceded by a small one); massive and diffuse.

The taxonomy is useful in simple description, i.e., identifying the forms an outbreak may take. But in the form offered by Sirois, it does not do much more. It is not unreasonable to consider extending it to accommodate variations in predictability, e.g. massive diffuse outbreaks are quite rare, as I understand, while other forms, notably explosive preceded by isolated cases seems to be more common. Nor is it unreasonable to create competing taxonomies which may be more useful in organization (e.g. identification by symptom type or severity), or in prediction, or in explanation. The Sirois taxonomy does have the merit of forcing one's attention away from the explosive outbreaks and toward other types. It is not clear, however, that it can be linked readily to other taxonomies created to understand hysteria at the individual level, e.g. Laughlin's (Kutash, 1965). Establishing such linkages is worth considering if only to simplify the way we look at the phenomena.

DIAGNOSIS, THEORY, AND MEASUREMENT

Given the difficulty of acquiring information about symptoms during an outbreak, it is no wonder that debate over usefulness of symptoms as an indicator of the nature of illness is vigorous. But even accurate well-articulated information will add only slightly to what one may understand. That is, we will not achieve much by relying on symptoms alone.

In poking through Sax (1979) for example, I find that carbon

disulphide produces "nausea, vomiting, headache...depression or irritability...insomnia and chronic fatigue". The same source reports that lead poisoning, of the alimentary form, also produces nausea, vomiting, headache, dizziness, and insomnia frequently. I learn from the National Institute of Mental Health (1978) that earthquakes and other natural disasters also produce similar reactions among some people. And, of course, there are plenty of reports of contagious hysteria, viral infections, etc. producing the same reactions.

Sax warns against educing the origins of the problem from symptoms alone for the case of dangerous industrial materials. There is no good reason to ignore that advice for illness with psychogenic origins. That leads us to theory and what information one needs to discriminate between psychogenic and other illness. To be sure, behavioral symptoms are important. Some origins can be ruled out because their effects are more specific, more dramatic, or different, a matter of contemporary toxicology as I understand it.

Some theory, explored by Kerckhoff, Back, and Miller (1965) among others, suggests that contagion operates through friendship networks, at least at later stages. To the extent that it does so regularly, then network itself becomes a useful element in diagnosis. And the theory certainly implies that information about networks ought to be obtained. That networks may be confounded with other characteristics, such as seating arrangement, is also clear. This implies obtaining information about physical proximity and traffic patterns as well.

At least in principle, inducement of relapse, discussed earlier, is a useful device for discriminating between illness with psychogenic origins and illness caused by other problems. That is, if in the absence of toxic environmental influence, episodes occur with visits of investigators, supervisors, or reproduction of earlier conditions, it seems to me that the case for psychogenic illness can be made more persuasively.

The benign morbidity of contagious hysteria is, according to Sirois (1974) at least, a useful discriminator. It is consistent with his theory, and this suggests that follow-up studies are warranted to assure that effects are indeed insignificant. That theory, however, is not entirely consistent in that it ignores rather more serious forms of what some would regard as contagious hysteria, e.g. suicides which occur in clusters.

For Kutash (1965), some of the criteria to be used in distinguishing between hysterical and organic disturbances must include the "belle indifference of the hysteric in relation to his symptom, the frequent failure of the disfunction to follow a correct anatomical or mere distribution pattern, contradictions such as the person who is hysterically blind but avoids bumping into persons or objects, and the susceptibility of the symptoms

to removal by hypnosis or suggestion." (p. 964). This standard has some promise when the purported effect of toxic agents, the dysfunction, are well-understood, and the correct anatomic patterns know. Judging from the number of production of new chemicals whose effects are not understood, however, the standard is useful only for a small class of dysfunctions. The illustration of the use of suggestion which appears to be available is the data computation center case: weather inversion was labelled as the origin of illness (Stahl and Lebedun, 1974). The ethical implications of suggestion which is patently false or not clearly accurate have not been worked out. More to the point, resorting to suggestion, or recording the consequence of accidental suggestion is warranted, since it too may enhance identifiability of the illness.

At least some theory based on laboratory study as well as on field tests holds that individuals who are susceptible to hysteria score high on measures of neuroticism and at the intermediate level on measures of extroversion (Eysenck and Buch, 1971). Assuming that the evidence is adequate, the implication is that indicators of neuroticism and extroversion be used in investigations to verify the illness. Exploiting this approach is likely to be difficult with existing indicators; development of less obtrusive indicators may be possible, but I do not know the area well enough to predict accurately. Related theory also stresses the use of various forms of therapy and tests of therapy for chronic cases (Eysenck and Buch, 1971). Again, the effectiveness of the therapy may help to establish that the label attached to an illness is proper. But follow-up study of contagious hysteria is rare, and opportunities to employ therapies may be still more rare.

Physical contact or visual observation of an episode is purported to be a reasonable device for helping to discriminate between psychogenic and other illness, and is consistent with some theory of contagious hysteria (see Chew, et al, 1976 for instance). It is rather more complicated an indicator, of course, in that viruses and the like are as plausible a cause of illness in these instances; visual contact may be a surrogate indicator of proximity to a gas or chemical producing the result. To the extent that these can be ruled out, physical and visual observation are candidates for assisting discrimination and so ought to be recorded.

Finally, though stress is said to be a major influence on occurrence of mass psychogenic illness, it is not yet clear how useful a discriminator it is. That is, many environments are stressful, but no outbreaks occur. Perhaps they do occur and we are not informed. It may also be argued that episodes occur in environments which are not stressful. Because no one has any information about these at all, the argument cannot be resolved. Al this implies greater attention to surveys as a

vehicle for understanding how to characterize the problem. We are not entitled to blame stress, except tentatively, unless there are data which show that outbreaks are more likely in high stress settings than low stress settings. Even then, the stress adds incrementally to prediction. And stress itself is as likely to be a symptom as well as cause of outbreak.

Though a good deal is said about them, a few other indicators appear to be less useful than advertised at least in theory. This includes the purported role of sex: women,it is said, are more susceptible. Yet counterevidence, if not strong, is at least plausible (see earlier remarks). Similarly, education level and socioeconomic class are said to assist in discrimination. But we have illustrations of physicians and nurses being affected. And, to my knowledge, there has been no concerted attempt to examine, say, company middle managers to assay frequency of the event.

AMELIORATIVE ACTION AND ITS EFFECT

Over the last 300 years or so, the treatments invented for victims of contagious hysteria have had the merit of being entertaining if not always effective. Soldiers were ordered to succor a convent's newing nuns during the 18th centuuy by beating them with rods supplied, evidently, by the local sheriff (Markush, 1973). Individuals so equipped and so disposed organized themselves into a religious order to assist the convulsionnaires of St. Medard during the same period. Drowning, torture, hanging, burning, dismemberment, and similar remedies have each had their time in the limelight. Even if the remedies eliminated both victim and disease, there were elaborate efforts to describe treatment and at least a few attempts to rationalize why they worked (Kors & Peters, 1972). It is not unreasonable to expect similarly conscientious, if less lurid, attention to detail in reporting about contemporary treatment. It is doubtful that we will learn much about controlling mass psychogenic illness unless analysis of the probably effect of treatment is careful.

REPORTING

The specific actions taken to remedy a problem in the particular episode, irrespective of their probable effectiveness, are not often well reported. Yet knowing what was tried is important if we want to contain or control the problem, if we aspire to build reasonable theory and linkages between action and response. The rationale for action and uniformity in application are no less important. More specifically:

- Sedatives are given, it is said, but there is no specification about type, time, dosage level and so on;

no mention is made of placebo level doses.

- Oxygen is provided to some, but we have no information on whether it was provided to all; why some victims received it and others did not.

- Isolation is employed but no information about timing, duration, and level of isolation is given.

- Some victims are hospitalized but we know little about duration of hospitalization or therapies provided, and the basis for hospitalization.

- Rhetoric, conversation, public statements are used but little information is provided about source, authority attached to source, medium of communication, and so on.

In the absence of more specific information, it is very difficult to justify a major investment in understanding whether a particular action works to reduce the problem, in what sense it works, and how well it works.

AMELIORATIVE ACTION AND DELIVERY OF TREATMENT

One of the few reliable rules of social research is that treatments are rarely delivered as advertised. Ameliorative action may differ systematically from plan, as when many individuals fail to take a prescribed sedative because of memory failure, distrust, or superstition. Or the deviation may be asystematic, as when individuals are persuaded to varying degrees that their problem is psychological rather than chemical in origin. The phenomenon is, of course, not confined to attempts to cope with epidemic hysteria, though it may be especially difficult to assay in these cases. It occurs with fair regularity in medicine, clinical psychology, welfare programs, and education (Boruch & Gomez, 1978).

The rule implies that at least crude verification of treatment is necessary. Independent testimony about who took what action, how soon, how often, and for how long is reasonable in principle, if difficult in practice. But there is little recognition of the rule in reports on mass psychogenic illness.

THE RESPONSE EXPECTED UNDER TREATMENT

The effects generally anticipated from treatment are generally clearly specified in reports. One would like to see the individual calmed as soon as possible, the contagion minimized, a return to work, and minimum disruption following return to work.

But it is difficult to find more than temporary attention to

these. The absence of longer term studies will lead us inevitably into scientific sins if history is any standard. Barnes (1977), for example, argues that the popularity of useless surgical treatments for "displaced" organs (ptosis), for constipation, for control of kenetic drive (thyroid operations), and other problems, during the early 19th century is due in part to the absence of follow-up research. It is unlikely that investigations of the effectiveness of action taken to ameliorate psychogenic illness, mass or otherwise, will fare better if such studies are not executed.

The effects which are not anticipated are no less important especially if they are negative. Sirois (1974) seems to be among the few investigators to recognize this in a general way, by suggesting that some approaches to treatment, such as exorcism, may exacerbate the problem. But he does not attend to the matter in his detailed comments. To their credit, Knight, Friedman, and Sulianti (1965) present data which, though ambiguous, highlights the possibility that mere presence of investigative researchers may have produced blackouts among schoolgirls. The report of immediate relapse of hospitalized women after a visit from their supervisors is less ambiguous (Colligan & Stockton, 1978). The negative effects of treatment are not well-documented for clinical therapy in general much less for care of hysterics. Experts such as Mowrer (1965) issue periodic warnings about iatrogenic inflation of personality disorders, but experimental research is sparse (see for example Lieberman, Yalom, & Miles, 1973 on encounter groups for an exception). Admitting the possibility of negative effects of what is ostensibly benign treatment is important. For if we don't look for these, we won't find them, and we will have deceived ourselves unnecessarily.

The failure to consider, record, and report negative effects is not confined, of course, to investigations of mass psychogenic illness. There are lots of examples one could cite from tests of innovative surgery and therapeutic medicine. It is clear that one ought to seek ways to avoid the manager's embarrassment in these cases, and to avoid the embarrassment of the victims. Ignoring the possibility of negative effects is not a particularly humane way to meet the objective.

THE EFFECTS OF REMEDIAL ACTION

Reports of mass psychogenic illness often contain the authors' judgment about the effectiveness of action taken to remedy the problem. The grounds for judgment are not always very clear, however.

Chew's (1978) report, for instance, is confusing in that the effects of chlorpromazine on victims are said to have been negligible. But the author goes on to report that after the girls had calmed down, they were sent home, implying that

chlorpromazine could have been effective. Knight, Friedman, and Sulianti (1965) observe that "many students continued to have attacks in spite of taking the prescribed medication on a regular basis" but they provide no detail on how regularity was monitored, or on how many students in the medicated group had attacks. Public confrontation and exhortation are, according to Sirois (1974) "usually useless and aggravate the outbreak" (p. 19). That is plausible, but he provides no evidence. On the other hand, Chew (1978) maintains that dousing a victim with water discouraged fakery and implies that it may have impeded further attacks. Conversation itself is said to be helpful, e.g. Colligan and Stockton (1978) emphasize the psychologists' "making clear to the individual that symptoms are real,", and that too is plausible but the evidence is informal. Counterfeit explanation led to cessation of attacks in the data computation center case (Stahl & Lebedun, 1974) but we cannot tell from the report whether and for how long individuals were monitored after the first attack.

The point is that it is difficult to decide what works based on such evidence, unless one is willing to rely entirely on the investigator's expertise. Even with very good evidence, decisions about whether an action ameliorates a problem is not easy. Gaps in reporting exacerbate the problem.

It does make sense to arrange the evidence on impact of an action in order of persuasiveness. Consider, for example, treatment of the victim. To the extent that the effect of action is immediate and large, and there is at least crude theory to link action and response, and there are no other uncontrolled influences on the victim, attributing the result to action is not unreasonable. In the easiest cases, the effect of sedation, say, will be almost immediate, dramatic, and consistent with theory of how sedation works. And the victim will not have responded to placebo. Variation in individual response will complicate matters, of course, but conformity to the stereotype ought to be recognized. To the extent that response to remedial action is delayed, taking credit for control of the problem is risky: people become calm even without sedatives. To the extent that the observed effect is small, taking credit is riskier still, since small variations in human behavior are the rule. To the extent that there are a great many actions taken, etc., sedation, isolation, conversation, etc., attributing positive response to one is not likely to be warranted. The most ambiguous evidence--delayed, small response, etc--ought to be identified as such.

Most remedial action is cheap and so there is little justification for attempting, in small formal experiments, to isolate their relative effectiveness. There is, however, some theoretical justification. It is important, for example, to understand whether placebo works relative to drugs in sedation. And it does seem possible to design small tests if victims are

treated in isolation, and to recommend systematic introduction of treatment tests rather than ad hoc action as part of instruction to physicians who may have to cope with these problems. It also seems conscionable to manipulate leave. That is, assigning a victim to a week of home rest is ethical, but may encourage other attacks. Home rest is a reward of sorts. I suppose that it can be argued that requiring very early return to work, rather than home rest, is ethical too, in that getting victims back on their feet is important. And the implication is that one may be justified in running controlled trials of the alternatives in this arena as in other arenas when effectiveness of treatment is debatable. Though the design of small studies of the sort is worth considering, more thinking needs to be done on the topic. Without design, and in the absence of immediate large effects of treatment, it is doubtful that we will learn very much about the sense in which treatments work or produce negative effects for the individual victim.

Some investigators also allude to the on-site objective of minimizing or reducing the number of attacks. The objective makes little sense in that we usually do not know what level of contagion will appear in the absence of extraordinary action. Exceptions might include the Singapore work where reappearance of mass psychogenic illness is, judging from Phoon's remarks at this conference, not uncommon. It may then be reasonable to base one's judgment on peak levels of psychogenic illness encountered in the past. Standards weaker than this peak one are also possible, e.g. the limits implied by friendship networks in which attacks spread. But I can think of no stronger bases for evaluating statements about how attacks were prevented.

EXTERNAL THERAPY AND COMPARATIVE TESTS

Despite the large body of literature on individual hysteria and the presumption that a contagious variety underlies mass psychogenic illness, there does not appear to have been much research or treatment of victims following an outbreak. That is, no formal tests of, say, desensitization therapy or other strategies have been undertaken to illuminate the problem. It is not clear that available therapy would be useful: that is, the reason for suggesting comparative tests. Nor is it yet clear that a therapy program is justified on economic grounds. Assuming that it is, those tests might be undertaken in the laboratory, the therapeutic setting, or on site. Whatever their location, if some therapies are found to work they may provide clues about more effective action during an outbreak, or at least some indirect evidence that action is likely to have been helpful rather than harmful. They may have the benefit of increasing the individual's resistance to influences which provoked the outbreak in the first instance. And they will almost certainly help to understand the etiology of psychogenic illness.

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THE EFFECT OF NONRESPONSE ON COUNT DATA

The unsettled nature of an episode of mass psychogenic illness is almost certain to produce missing data. For example, despite the Colligan et al (1979) plan to sample victims and nonvictims in the electronics plant outbreak, they succeeded in obtaining information from only 81 of the 150 individuals to whom questionnaires had been given. They deserve credit for the attempt to sample and reporting results, especially since such reports are infrequent. The low response rate, however, does suggest that the analysis of the inferential biases engendered by missing data is warranted. The statistics presented on symptom patterns, for instance, are misleadingly precise because nonrespondents go unrecognized.

To illustrate what might be done in simple sensitivity analysis consider a situation in which response rate is less than 100% but the obtained sample is large and responses are accurate. The assumption about sample size and accuracy are temporary and made for simplicity's sake. Suppose that only 54% of the target group responds, as in the Colligan, et al case. Their report suggests that 58% of the 81 respondents had headaches during the outbreak. The suggestion is accurate (ignoring sampling error) if people who respond are random sample of the target of 150. But that is unlikely. Nonrespondents may differ from respondents in education level, compulsivity, and other ways, in this research as they often do in other action research. One may construct a less misleading description of headache rate by recognizing nonresponse and computing arithmetically permissible upper and lower bounds on headache rate in the target sample of 150. Doing so suggests that at minimum 31% had headaches and at maximum, 78% did so. Had we used the actual target sample size for Colligan et al, the range would have been larger, but the authors fail to specify it. The permissible range implies, of course, that statistics based on samples in which response rate is less than 50% are likely to be useless. Even the range we have computed here is considerably wider than we'd like, and the only way to reduce its size is to increase response rate.

Developing crude bounds on the possible value of a proportion for a particular ad hoc sample, or developing more general tables requires only simple arithmetic. One constructs a table cross-classifying Respondent/Nonrespondent against whether response is positive or negative. That is,

	Respondent	Nonrespondent	
Response Yes	a	b	a+b
Response No	c	d	c+d
	a+c	b+d	a+b+c+d

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The table states that of the observed a+c respondents, a said Yes to the inquiry (about,say headaches), and c said No. Further, of the b+d individuals who failed to respond, b would have said Yes and c would have said No. The numbers b and c are unknown of course, but we do have their total as well as total number in the target sample. This, and the number of respondents affirming the trait, a, is sufficient to compute minimum and maximum values. Specifically, the minimum number of nonrespondents who have the trait in question is zero, i.e., b=o, yeilding a minimum for the sample. The sample maximum occurs when all (b+d) nonrespondents have the trait. The minimum proportion of individuals in the original target sample than is a/(a+b+c+d) and review of proportions based on imperfect response rate. A response rate of, for example 50%, given in the first column of the table, and an observed proportion of 75%, given in the second column, yields a permissible arithmetic range of 45/85%. That is, between 45% and 8% of the target sample have the trait in question.

I have used the word arithmetic here because such tables though useful, have only an elementary relation with statistics. Indeed, one ought to construct little confidence intervals, if the target sample and actual sample is selected from some larger target population. Sample-to-sample variation ought to be recognized in Colligan et al (1979) original tables, as well as in a sensitivity analysis table of this sort. Computations are given in any standard statistics text. One can presume further that responses will be imperfect at some level; a respondent may affirm falsely that he has a symptom, or falsely deny that he has it. If the rate of false positives and false negatives can be guessed or estimated from side studies, then more elaborate tables can be built to help assay credibility of original data.

Comparisons are similarly complicated by nonresponse, especially if nonresponse rate differs across from one group to the next, or from one time to the next. Suppose, for example, that one is fortunate enough to have a headache count before an outbreak. Assume that response rate is 50% and the observed proportion of people in this sample who admit to headaches on a given day is 25%. After an outbreak one might obtain exactly the same response rate and headache rate; yet it is possible for headache rate to have changed from 15% (the minimum), to 63% (the maximum), the change being masked by poor response rate. It is similarly possible for a group of victims and a group of nonvictims to have exactly the same response rate of 50% and rate of affirming headaches, 25%, yet actual rate of headache can differ notably, as much as 50%.

As one might suppose, the arithmetic range becomes larger with higher level of nonresponse, lower validity in response, and smaller sample. There is no substitute for good data. But the ambiguity might be reduced if stereotypical forms of bias

are known. One may find, for example, that respondents and nonrespondents in a particular case do not differ from one another in education level or other characteristics. The information makes it more reasonable for some analysts to assume that a headache rate found in the respondent group would also be found in the nonrespondent group, for instance. And it reduces one's dependence on demoralizing of necessary computation of the sort described. That matching on the basis of observed variables such as education may itself produce biases analysis is also clear; variables may be imperfectly measured and may be incomplete. For stereotypical forms of this bias, see text and references to work in chemistry, education, and medicine in Campbell and Boruch (1975).

Table 1. Arithmetic range for the true proportion of individuals with a given trait when the overall response rate is 30%, 40%,...100%, and the proportion of observed Yes responses to inquiry is 0%, 25%...100%. The table assumes perfectly valid response and ignores sampling variation.

Overall Response Rate	Observed Proportion of Yes Responses	Minimum True Proportion of Yes Responses	Maximum True Proportion of Yes Responses
100%	50%	50%	50%
80%	100%	80%	100%
	75	60	80
	50	40	60
	25	20	40
	0	0	20
70%	100%	70%	100%
	75	53	83
	50	35	65
	25	18	48
	0	0	30
60%	100%	60%	100%
	75	45	85
	50	30	70
	25	15	45
	0	0	40
50%	100%	50%	100%
	75	38	88
	50	25	75
	25	13	63
	0	0	50
40%	100%	40%	100%
	75	30	90
	50	20	80
	25	10	70
	0	0	60
30%	100%	30%	100%
	75	23	93
	50	15	85
	25	8	78
	0	0	70

RELATIVE COUNTS

Although the number of people affected in an institutional episode are usually reported, the number is not always informative. The meaningfulness of the numbers depends on one's definition of who is affected of course, and that depends on exhibition of symptoms. Problems in determining and reporting the latter were discussed earlier. Apart from those problems, meaningfulness of reported incidence depends on the number of individuals who might be affected.

That is, the number of vitims relative to the total number in a group more or less at risk is important but rarely reported. That 25 individuals out of 25 were affected seems to imply something different from a case in which 25 out of 100 or 1000 are affected. In particular, I should think that physically dangerous environmental influences are more likely in the first case, and psychogenic illness, or environmental insult, or both are more likely in the second. Further, theories offered to explain hysterical contagion may hinge on isolation, and on the proportion of a group who are friends, e.g. Kerckhoff, Back, Miller (1965). In any case, a report of the absolute number affected is a bit misleading, constrained as it is by physical characteristics of the environment, the size of the administrative unit, and so on. A ratio helps direct attention to the unaffected group, and that is warranted scientifically. Finally, we ought to know whether the relation is reasonably stable from one case to the next, for this will help us to predict crudely at least, and may help us to understand better the effects of remedial action.

How base rates of the sort ought to be computed is not at all clear, however. Specifically, how should we define the total group, the denominator of the computation? Should we use the number of individuals within an administrative unit in which an epidemic occurs? Which administrative unit and under what rationale? Or, should we use the number of individuals who occupy the same room or floor, or who are otherwise in visual contact or physical proximity? Because any convention adopted as answer is likely to be premature, and because computation of each ratio is cheap, providing ratios based on each is probably warranted.

ARCHIVES AND SECONDARY ANALYSIS

The idea that one ought to store data generated by investigation of mass psychogenic illness, and to make it available for independent secondary analysis, is implicit in Colligan and Stockton's (1978) work. It is consistent with a scientific tradition which encourages skeptical review, and with a more recent tradition of regarding expensive data as a national re-

source. The idea has merit made explicit in the following.

It is plain that published reports will continue to be a major vehicle for scientific communication. Judging from my review, reporting can be improved. Put simply, details which ought to be available to the interested reader are often not provided, and reports which contain details are often not cited. The result is that we rely more frequently on the expertise of the author than we ought to--edifying for the author perhaps but not especially good for understanding the problem. Partly as a consequence, journals such as Anesthesiology, for example, occasionally publish the entire data set used as a basis for research along with the pertinent article. Others such as the Journal of Personality and Social Psychology, maintain an editorial policy which requires that authors make pertinent raw data available for reanalysis. Formal publication of second opinion, and especially of dissenting views, along with original analysis is similarly consistent with a principle of ready scientific access to information. Here too, there is reasonable precedent, e.g. the U.S. General Accounting Office's publication of criticism of GAO investigation.

Publication of all pertinent material is not justified, much less feasible. Yet logs, letters, memos, and microdata may be fundamental to competing analyses of a given data set by analysts outside the original group of investigators. An archive does help to resolve this latter problem to the extent that it provides flexible access to all relevant material. It may also be justified on grounds other than facilitating secondary analysis of particular cases. For example, the relatively small number of cases involved in each episode of contagious hysteria makes statistical analysis difficult if not gratuitous. An archive can help to inform a judgment to pool data across sites and can facilitate the pooling process in the interest of better statistical power. Further, the caliber of data available on episodes varies greatly as many of you know. Protocols for investigation will help to increase uniformity of reporting, and an archive is a reasonable vehicle for monitoring adherence to protocols, for integrating material, and for improving the quality and flexibility of protocols. In any event, secondary analysis makes good economic sense in making expensive data work repeatedly and at low cost. The reanalyses may be executed in the interest of policy analysis and facilitates the process in principle, and judging from economics, psychology, and criminology, in practice as well (Hedrick, Boruch, Ross, 1978).

More generally, the notion of statistical archives designed for such purposes is consistent with federal statistical policy (President's Reorganization Project, 1978). Judging from the report of the Privacy Protection Study Commission, the notion is consistent with the intent of the Freedom of Information Act (Pub. L. 89-554) and the constraints of the Privacy Act

(Pub. L. 93-579). The state of the art in development and maintenance of social research archives has advanced substantially over the past ten years, and applications are easy to find: see Boruch (1979) Boruch, Wortman, Cordray (1979), Hedrick, Boruch, and Ross (1978), for example. It may be possible to expand existing archives, in the interest of economy, as Dollar & Armbacher (1978) suggest in their description of the National Archives' machine-readable data program. The existence of pertinent professional organization, such as the International Federation of Data Organization, makes world-wide cooperation possible.

None of this implies, of course, that developing such an archive is easy. The problems in organization, management, and quality control of data are obvious. Furthermore, evaluating utility of any archive is complicated at best, and we know very little about the task. We will continue to be ignorant unless an archive is tried out in pilot test.

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MASS PSYCHOGENIC ILLNESS OR CHEMICALLY-INDUCED HYPERSUSCEPTIBILITY?

Claudia S. Miller

Chicago, Illinois

Dr. COLLIGAN: Our next speaker is Claudia Miller, who is a private consultant and an industrial hygienist. Claudia is presently authoring a book entitled Chemical Susceptibility and Sensitization and the title of her presentation is going to be Chemical Hypersusceptibility.

We are all familiar with the plight of the so-called "hypersusceptible" worker. He or she may have been employed at a particular job or operation for several years when suddenly the materials or chemicals involved in the work are no longer tolerated. Most familiar are those workers whose skin becomes sensitized to a particular agent and thereafter even the least contact with the offending substance triggers a massive skin reaction. There is the individual who works with urethane foams, but becomes so sensitive to the chemicals used in its manufacture that just walking by the plant percipitates an asthmatic attack.

Such skin and respiratory sensitivities are familiar to every industrial hygienist and physician. It is always impressive, if not somewhat unbelievable, how very little of the offending substance is required to spark a reaction. A few molecules seem to be all that is necessary.

Although respiratory and skin sensitization are widely-recognized they represent only the "tip of the iceberg". Many other kinds of chemical sensitization occur which affect the nervous system, cardiovascular system, digestive tract and other parts of the body. These effects have been well-documented among chemically susceptible patients who have been exposed to test concentrations of chemicals under controlled conditions. Of particular relevance to this conference on mass psychogenic illness is the fact that the symptoms which are induced in these patients mimic perfectly the "subjective" complaints of workers involved in these outbreaks.

A great diversity of worksites have been involved in these episodes, including electronics assembly plants, a frozen fish packing plant, a shoe manufacturing plant, a lawn furniture manufacturing plant, and garment making operations. Yet what is puzzling and invites speculation are the similar kinds of symptoms the workers experienced - headache, dizziness, lightheadedness, weakness, sleepiness, nausea, a taste in the mouth, blurred vision, dry mouth, chest tightness, abdominal pain, breathlessness, watery eyes, chest pain, difficulty swallowing, numbness, diarrhea, racing heart, tingling feeling, muscle soreness, fainting, ringing in ears, vomiting and fever - in that order, were reported in one detailed investigation.*

What conceivable etiology could account for such a rash of "subjective" symptoms in such dissimilar worksites? It is the purpose of this presentation to suggest that there may well be

concrete chemical causes underlying these psychological episodes.

A newly emerging approach to medicine, called "Clinical Ecology", has much to offer in increasing our understanding of the effects of chemicals in our environment upon us. Its practitioners are engaged in diagnosing and treating the hypersusceptibles in our population, and its theories and techniques are very relevant to this symposium. Clinical ecologists have successfully demonstrated in thousands of "hypersusceptible" patients that minute quantities of chemicals in the air, food, or water can be used to provoke major and minor reactions under controlled test conditions. The quantities of chemicals used are far below the concentrations in OSHA standards. Clinical ecologists work in units of molecules, not milligrams per cubic meter. The results of their tests on patients demonstrate conclusively that the majority of the chronic physical and psychological symptoms experienced by individuals results from exposure to chemicals in the environment.

Many patients who see clinical ecologists for help with their symptoms work, or formerly worked, in electronics assembly plants, plastics manufacturing, leather cutting, chemical operations, and other facilities. Many, if not most of these patients, are sensitive to one or more of the exposures on their jobs.

Clinical ecologists do not rely upon conventional skin "scratch tests", as do most allergists. Tests are performed by direct provocation; that is, challenging the patient with a test dose of a suspected substance, either by placing a drop or two of extremely dilute chemical under the tongue where absorption into the bloodstream is rapid or by inhalation.

In this manner, it has been amply demonstrated that minute exposures to a wide variety of chemicals and synthetic materials can cause headaches, dizziness, ringing in the ears, stiff joints, nausea, and a host of other familiar symptoms. All of the symptoms reported in mass psychogenic outbreaks in industry can and have been provoked by Clinical Ecologists in their patients hundreds of thousands of times. A listing of symptoms and conditions that have been elicited by test exposures to low levels of environmental agents (Table 1) shows that no part of the body escapes. Indeed the nervous system is just as vulnerable as the skin or respiratory system, if not more so.

Clinical Ecology is a new field, relatively speaking. However, its impact on the practice of medicine in this country is sure to be enormous. Unlike other approaches to illness, it explains why disease occurs. Clinical Ecology demonstrates, in a convincing manner, that there are distinct, reproducible

cause-and-effect relationships between chemical exposures and symptoms. More will be said on its techniques later.

It is generally believed that the "susceptible" worker is somewhat of a curiosity, that he or she represents only a tiny fraction of the workforce and should be handled as a special case. Indeed this is not true. Susceptibility to chemicals is widespread and incapacitates many workers in this country, but we have failed to recognize its occurrence. It is important that we revamp our thinking. Hypersusceptibility is not limited to skin reactions and respiratory sensitization. Skin conditions are readily visible, and respiratory difficulties can be measured with some certainty. This is true for animal toxicity testing as well. But "sensitization" of the nervous system, gastrointestinal tract or other portions of the body by chemicals is not so readily detected or measured. A guinea pig simply will not tell you when it is depressed, has a headache, feels nauseated or cannot concentrate. Subtle changes in neurological and other functions, which undoubtedly occur with exposure to many sutstances, thus cannot be detected or measured easily before a chemical reaches the workplace. For decades workers have served as the final proving ground for the effects of new materials introduced into commerce, and they will undoubtedly continue to do so for sometime into the future.

In this discussion, the term "allergy" has been avoided. Even the term "sensitization" is used with hesitation because of the widespread view among conventional allergists that these terms denote an antibody-antigen reaction only. To the contrary, "sensitization" is used here to describe the process, however, it occurs, by which an individual becomes intolerant of exposure to a substance, so that even minute quantities provoke an adverse reaction. Antibodies and antigens are only a piece of the story. Our biological defense systems involve several different classes of immunoglobuline, B-lymphocytes, T-lymphocytes, the bone marrow, thymus, the components of the complement system, the kinin system, histamine, serotonin, eosinophils, the hypothalamus and other participants. Because of all of the confusion over the meaning of the term "sensitization", the word "susceptibility" is often used by Clinical Ecologists to describe the same thing.

It is not known how this process occurs. It does occur and it is demonstrable. Theories abound. There is a great deal of information in allergy, toxicology and the neurosciences that is important to understanding the complexity of the process. One theory is that small amounts of chemicals disrupt the hypothalamus. Corwin* describes the complexity and delicacy of hypothalamic function:

> "Imagine, if you will, a chemical laboratory set up to monitor a stream of fluid continuously. This laboratory is equipped to perform analyses for simple

> substances such as acids, bases, and salts, ions such as sodium, potasium, calcium, and chloride, and more complex substances such as glucose and cholesterol, simple hormones such as adrenaline and thyroxine, more complex hormones such as the peptide hormones. This laboratory has a built-in computer which evaluates the balance between all these substances and controls this balance so that in case one is formed in excess, the valves can be tightened electrically to decrease the production or to generate an antogonist. In addition, this laboratory has the means for the synthesis of organic chemicals which can be released into the flowing stream of fluid at appropriate points to alter the action of the way stations producing the desired or undesired materials...The human body has its analytical laboratory, computer controller, and hormone factory compressed into a few grams of hypothalamus, a true marvel of microminiaturization.

The dynamic involvement of the hypothalamus in virtually every function of the body and its amazingly small size lead some to feel that it plays a part in hypersusceptibility. It is theorized that molecular quantities of synthetic organic compounds would easily disrupt the critical balance which the hypothalamus labors to maintain. Chemical interference with or "poisoning" of this critical tissue may account for the many diverse symptoms affecting every part of the body that are seen in chemically susceptible patients. Once harmed, the ability to compensate for the presence of other synthetic chemicals is lost or diminished and further exposures drastically rock a balance that is already out of kilter.

Bell* proposes that the hormone bradykinin is involved as an essential mediator of adverse reactions to chemicals and food. She further hypothesized that the olfactory system, together with the hypothalamus, plays a significant role in initiating these somatic and psychological reactions to chemical exposure.

Many scientists working on this problem feel that it is only recent in its origin. Prior to World War II, patients did not often see doctors about these problems, if they did at all. Today, most, if not all, of us exhibit at least sporadic symptoms of it; many are chronically ill. What has happened since World War II? Petrochemicals and synthetic materials have been introduced into the workplace, home and environment as a whole. Between the years 1950 and 1977, a span of only 27 years, the production of synthetic organic chemicals rose more than 623% to a level of 306.6 billion pounds.*

Twentieth century technology subjects all of us to a plethora of chemicals which, form an evolutionary standpoint, our bodies are not equipped to metabolize:**

"The human race has had millions of years to adjust to cold, light and other solar radiations, as well as various other physical exposures...Likewise, most of us are adequately adapted to common foods, plant pollens and oils, spores, insects and other animals...In contrast to these naturally occurring physical and biological exposures...the man-made chemical environment is a relatively new exposure, both racially and individually."

It is particularly interesting to note that many of the hypersusceptible patients whom practitioners in this field are seeing work or were working in operations involving plastics, electronics, soldering, and similar kinds of "light" industry. These are not the places where industrial hygienists would ordinarily concentrate their efforts since exposures are considered to be at relatively low levels and even "insignificant". Attention is, instead, given to more "major" exposures in other operations. Perhaps this emphasis is in need of overhauling. Not only are many "hypersusceptible" patients coming out of these plants, but this is also where a number of larger episodes of mass psychogenic illness have occurred.

It seems as though multiple "minor"exposures to a huge variety of esoteric organic compounds, (such as those found in plastics, resins, fluxes, and their oxidation products) can wreak havoc with the body's defense mechanisms. Do they interfere with hypothalamic function, thus creating a disturbing array of symptoms? For a moment, try to imagine the sheer biochemical complexity of all of these compounds entering the bloodstream, reaching the hypothalamus which tries hurriedly to compensate for their presence, or to somehow eliminate this harangue of chemical saboteurs! It may be more than a minor skirmish that takes place. And the hypothalamus must respond to this exposure and the next and the next, day after day, year after year. It is hard to imagine how it could not become exhausted or at least make mistakes with such a continuous onslaught.

Historically speaking, a convenient and productive approach to learning about medical conditions and problems has been to observe and study, in detail, those individuals who represent the extremes--for example, those who are most severely afflicted by a particular disease or condition or who practice extremes in diet or lifestyle.

The chemically susceptible patient affords us just such an opportunity for studying the subtle effects of chemicals on the nerves,heart, muscles, stomach and intestine, and other parts of the body.

The pioneer in the field of chemical susceptibility in this

country and the world is Dr. Theron G. Randolph who has studied and treated these individuals for over three decades. In his practice, he has seen a limitless range of medical conditions, all the way from arthritis and schizophrenia, to diabetes, multiple sclerosis, and a host of other chronic illnesses. Most of the patients he sees are referred to him only after conventional medical therapies have been exhaustively sampled, with little improvement. He recognized early the influence of the chemical environment upon health. He devised a logical and methodical approach for sorting out the environmental factors responsible for his patients' illness. It most certainly will emerge as one of medicine's most important contributions. In searching for the cause of a patient's illness, he first isolates the chemically susceptible patient in a ward that is specially constructed to be free of petrochemicals and other synthetic materials. Electric hot water heat, ceramic tile floors, cotton bedding and clothing, no use of perfumes, cosmetics, smoking materials, etc. are all facets of this approach which he terms "Comprehensive Environmental Control".* Rigorous control necessitates a detailed knowledge of construction materials, furnishings and supplies can be achieved only through painstaking effort. In addition to completely controlling all chemical "inputs" to the patient's systems, fasting is carried out for several days after admission by each patient. Experience has shown that chemically susceptible patients most often have developed some food sensitivities as well.**

The importance of a chemically controlled hospital environment cannot be overemphasized in diagnosing such cases. A patient's reactions to chemicals and foods are often chronic and lowgrade and symptoms from a single exposure may last for days. In persons who have developed widespread sensitivities, i.e., they have become susceptible to a multitude of substances or are constantly being reexposed to the same substance, the effects of one exposure overlaps with the next and the next until no one, neither patient nor physician can tell what is making the person ill. This effect of one exposure hiding or concealing the symptoms of another exposure is called "masking". In a controlled environment from which all substances troublesome to the patient have been removed, single test exposures* can be given and, for the first time, the patient will "see" a cause-and-effect relationship between his exposures and his symptoms.

After several days in this controlled environment, without food, most of the chronic, troublesome symptoms that brought the patient in diminish significantly or disappear entirely. A period of testing ensues during which patients are tested for one substance at a time, food by food, and chemical by chemical. All reactions are carefully monitored and recorded.

Examples of testing that have been performed on some chemically

susceptible individuals whose jobs contributed to their exposure follow. These cases are but a few of many that could have been selected for this purpose.** Note that many cerebral (or so-called "psychological") symptoms which are caused by chemical exposure and the constellation of other symptoms affecting every system of the body.

Case No. 1 - Mr. J.M., 43 year-old university instructor, whose office was located above a biology laboratory. He constantly smelled the odor of formalin arising from the floor below. He suffered from chronic progressive fatigue and exhaustion, mental confusion, poor concentration, memory deficits, irritability, depression and multiple system complaints for many years, according to his patient record. A psychiatric clinic diagnosed his complaints as due to "unrecognized or undefined emotional factors," and psychiatric treatment was recommended. Other complaints included chronically sore, still muscles (myalgias),chronic constipation, insomnia, "on edge" feeling as if fingernails were being grated on a blackboard, and little tolerance for stress. He also noted that alcoholic beverages helped to relieve his irritability and impaired concentration temporarily. He was aware that exposure to tobacco, paint solvents, floor wax, perfumes, inks, papers, books (printing ink or treated paper), and traffic exhaust all made him feel worse.

After several days in a hospital environment that was as free as possible of exposure to synthetic chemicals and materials, he was tested on formalin by briefly inhaling a small amount from a distance; this exposure made him lightheaded, foggy, his hands became cold, he felt dizzy and experienced difficulty thinking, and tightness in his leg muscles.

Similar testing with a common pesticide also caused difficulty thinking, mental fogginess, giddiness, and a feeling of being remote.

Exposure to auto exhaust provoked lightheadedness, difficulty in concentrating, thinking and pronouncing words, fatigue, and a "floating"feeling.

Case No. 2 - Mr. B.T. was 43 years old when first seen; his earliest symptoms occurred 5-6 years before when he felt himself becoming lightheaded and dizzy at work. As a senior repairman for a natural gas transmission company, he repaired, installed, and maintained plant equipment and did routine pipefitting and plumbing. Prior to this, he had spent 9 years working with gas compressors. He had no family history of allergy nor did he himself have any food, dust, mold, tree, grass, or insect allergies that he was aware of. He suffered from severe headaches which were not relieved by any of the 6 types of medication he was given by doctors. In addition,

after exposure to any of a number of chemicals, he would get a flush across his nose and cheeks, sometimes mental confusion, always severe headaches, blurred vision and nausea. Symptoms were provoked by truck exhaust, gas furnace, paint, smoke and burning leaves, detergent, hairspray, perfume and supermarket cleaning agents, among others. His symptoms would abate significantly while in Florida on vacation. He recalled doing some spray painting in an enclosed area at work with inadequate ventilation and no respiratory protection for several hours a day over a 2-3 week period, shortly before he became incapacitated. About the same time, he had refinished some furniture at home, strippint, sanding, and spraying it with lacquer.

Case No. 3 - Mrs. P.T. experienced episodes of confusion, uncoordination, mental dullness, fatigue, chest pain and chronic bronchitis. She commented that she felt as if she were "a walking Zombie". At age 29, she worked in the manufacture of teletype machines where her blood pressure increased and her weight went up to 245 pounds; she found that breathing or touching the oil she used provoked a skin rash, pains over the eyes, nausea and made it difficult for her to focus her vision. When she became a solderer, her constant cough required that she take lozenges continuously while at work. The company moved her under an air vent where she found she could work better. At least one doctor told her her reactions were due to her imagination. She was troubled by many common exposures, including newsprint, cosmetics, hairspray, traffic exhaust, cigarette smoke, detergents, the odor of many plastics. In a controlled hospital environment, she was tested using a single-blinded procedure with the flux and freon she used, and with a saline solution for a control. After the freon exposure, she complained that she was having difficulty thinking, felt as if her head were going in circles, her chest tightened up, her face and eyes itched, she had intermittent chest pains and pains in the calf of her legs. She felt pressure in her bladder, and developed a rocking feeling. Throughout the night she experienced further symptoms, including chills, and difficulty hearing and in the morning there was obvious swelling around her eyes and mouth. She stated that she associates both chest pains and urinary frequency with working with soldering wires, and that this problem went away when she stopped soldering for a few weeks. Ambient air inhalation test with the flux she used caused her pulse to beat faster and heavily, and by the end of the test she had begun to develop a rash that spread throughout her upper extremity. Exposure to the saline solution under identical circumstances, provoked no symptoms.

Case No. 4 - Mrs. Y.R., 48 years old, worked for a plastics manufacturer doing silk screening. She traced her symptoms, which include fatigue, insomnia, muscle stiffness, soreness

and spasms, arthritis in her knees, a skin rash, irritability, mental confusion, difficulty making decisions, urinary frequency and burning, to the time that a bottle of lacquer or paint thinner fell from the top of the machine at which she was working and landed spilling the chemical on her lap. She recalls no immediate reaction, except for local burning and feeling "very strange". She noticed that she stuck to the plastic stool when she tried to get up. She retired to the women's lounge until the end of the usual supper break and upon trying to get up suddenly felt awful, her legs became numb and tingling from her knees down. She tried to take water, but could not swallow; her eyes were red and bloodshot and vision blurred. She could not talk. Her throat and insides hurt and she broke out in hives all over. She was taken to the local hospital and made a slow recovery. Chronic fatigue become a major problem for her. She did return to work in another department, an inspecting and packing line, packing plastic containers into a box, but she would often get hives and itching while trying to perform this work. The company's physician did skin tests on her and told her she was allergic to foods and petrochemicals. She feels better when she stays in her home, which is all electric(no gas utilities). One evening, her husband came home with insulated overalls on that were permeated with gas odor. Her throat tightened up, her eyes became dripping and red, and she began to shake and tremble.

Many common exposures, including perfumes, aftershave and exhaust from engines give her headaches and other symptoms. When she develops a skin rash from an exposure, it begins in the area where the silkscreening solvent contacted her legs. She threw out her kitchen cleaners since they bothered her and she reports getting sick from most odors from combustion or synthetic materials.

Given a test exposure for natural gas in the hospital, she reacted with mental confusion, nausea, headache, low backache, and onset of chest congestion.

Exposure to carbonless carbon paper resulted in blurred vision, difficulty concentrating, and tingling around her mouth and neck. Carbon paper caused nasal and eye itching and hot flashes; she had no reaction to a control consisting of plain water.

Case No. 5 - Mr. F.V.55-years-old, worked as a leather cutter, for several years,until 8-9 years ago, and odorous leather (still cowhide) with a more supple texture was introduced. Her symptoms began gradually, and she began to experience a sensation that her tongue was swelling and prickling; her throat would feel like it was about to close, and she would develop headaches and dizzy spells. At night she would get cramps in the calfs of her leg and depressed feelings. She

had no known childhood allergies, no hayfever, asthma, articaria or eczema. Women adjacent to her working on the cutting machines also complained of dizziness and headaches while working with the leather; some men felt a "floating sensation", plus headache and dizziness.

She now suffers chronically from a facial rash, tongue paresthesias, burning around her eyes, headache, and conjunctivitis. She reacts to the synthetic yarn she crochets with, certain kitchen cleansers, soaps, auto exhaust, oil furnace (when the furnace "kicked on" her face became blotchy and red). She states, "I'm tired all the time--I wake up tired," and has felt this way over the past five years. She has no problem getting a skin rash from working with her hands unprotected at work. In order to test her in the hospital, one of several of the specific leathers which bothered her were cut up and twenty of each placed in 100 cc of water overnight. Serial 1:5 dilutions with water were made and she was given (0.1cc) drops of this diluted solution under her tongue (sublingually) to test for reaction. Plain water was used as a control. The extract from leather A provoked a headache, slight nausea and some facial burning. Leather B caused her face and tongue to burn, dizziness, head heavy, and throat tight; she also developed a headache, her face became flushed and warm and her neck and cheek reddened. Leather C extract made her tongue numb, face burn, her pulse jumped from 78 to 100; she experienced tightness in her head, her eyes burned and watered and her tongue felt thick. Controls administered in identical manner elicited no reaction or complaints. She too was told by doctors that her problem was psychosomatic.

Case No. 6 - Mr. T.D. Worked as manager of a farm chemical distributing plant in a small building consisting of an office and storage room only. In the storage area roughly 30 tons of insecticides and herbicides were stored: he stated that ventilation was poor. Among the chemicals in the warehouse were dieldrin, DDT, heptachlor, toxaphene, aldrin, chlordane, 2,4D. The chemicals were stored in stacks of paper bags 8-10 feet high. After a year he began to notice his thinking had slowed down; he was having trouble keeping up with correspondence, and he underwent tests for a brain tumor. After returning to work from the hospital, within 30 minutes after entering the office his arms went limp and he could barely walk. Although he left work quickly, for three days thereafter muscles in his arms and legs were very sore and lame. Now even the pesticide residues on foods that are commercially sold trigger symptoms of headache and facial swelling. The same foods raised without pesticides evoked no symptoms. He notices difficulty making quick decisions while driving a car; although he used to enjoy reading, his eyes now tire and he must read the same thing two or three times to understand it. His memory is poor. Another man who

worked with him for two weeks on the job left because of nausea and headaches. A test with some of the chemicals stored in the warehouse was conducted by dipping a small glass rod in a dilute solution of chlordane in olive oil and holding it one inch from the patient's nose. His eyes watered, left lid dropped, and he had needle-like pains in his left eye. Although exposure was discontinued after three minutes, pains, perspiring, chilling, drooping eyelid, tingling and muscle spasm continued for the next several hours. Olive oil by itself provoked no reaction. His speech is often slurred and others at work began to make fun of his spelling and writing, although he claims to have been a good writer previously. At times his speech was slurred so that he appeared to be drunk. Interestingly, one doctor diagnosed his difficulties as "a cover for latent depression and feelings of inadequacy."

Case No. 7 - Mrs. F. T. worked in a plant engaged in the molding and potting of various plastics. Work had just moved to a laboratory in a new building which, according to health and safety staff at the plant was not adequately ventilated for the work being performed. Nevertheless, a coworker came into the room to mix something and spilled it on the hotplate, which later was found to be polyurethane. She was in this room for thirty minutes when she began to vomit. She later passed out and was taken to the hospital for three days. She recalls little during this time. When she went home she continued to experience nausea and headaches. She took work which involved only crimping metal pins onto plastic covered wires, but continued to be ill. For about a year she continued to take headache and nausea pills to keep her going. Finally, she left this job and tried to go to business school, only to find that the typewriter ribbons, carbon paper, and other office supplies bothered her. She was twice referred to psychiatrists as a result of her complaints. She became unable to use any house-cleaning chemicals, such as detergents, floor waxes, ammonia, etc. and became intolerant of traffic exhausts and tar exposures on the highway. She complains that she will start to do something and forget what it was she started to do; she has trouble remembering names and making decisions; she starts to talk and forgets what she was going to say. She never raised her voice at her husband or children before, but now screams at them. Often she cries for no apparent reason and her depressions are followed by headaches. She had had trouble passing out in a grocery when it was being mopped with a detergent or disinfectant and while driving, although she managed to stop the car first. Less severe reactions to chemicals leaves her feeling wobbly, lightheaded, dizzy and confused.

Undoubtedly the single most impressive feature about testing patients, such as those whose cases have just been cited, is the manner in which "psychological" or"mental" symptoms can be provoked (and these are reproducible) by single exposures

to test substances. Patients themselves are astounded by how the anxiety, nervousness, depression, confused thinking, "feelings of appartness", difficulties in concentrating, speaking or writing can be recreated for them in the hospital. Even a disbelieving observer is impressed when a patient suddenly becomes stuporous, begins to weep "hysterically", or staggers as if drunk during a test, when only moments before that the patient appeared perfectly normal and well-adjusted.

The "average" hospital is not a suitable environment for testing chemically susceptible patients. One hospital dietician developed widespread disabling chemical sensitivities after working in a hospital kitchen that was routinely pesticided. Another hospital employee developed a massive chemical problem that has been tied to her use of a widely-used hospital disinfectant. It is more than coincidental that several of the outbreaks of mass hysteria reported by Markush in his extensive review of 100 cases of mental epidemics occurred in hospitals.* For the chemically susceptible, conventional hospitals can be dangerous places:

> "Chemical air contamination of hospitals is contributed to by the gas utilities of laboratories and kitchens; chemical deodorants, disinfectants and cleansers; the odors of ether and other volatile anesthetics blown off by recently operated patients; odors arising from the use of certain drugs; the odors from rubbing alcohol; perfumes worn by hospital personnel and visitors; rubber draw sheets and other rubber and plastic bedding and furniture."

For two years of my career as industrial hygienist, I worked for the University of California, San Francisco campus, a major medical research and teaching facility with two hospitals and about 5,000 employees. Over a two-week period, I had almost a dozen emergency calls from staff located in one particular wing of the hospital concerning a "strange odor". I was only a block from the facility, but by the time I arrived the odor had disappeared. Numerous individuals complained of headaches, nausea and a host of other symptoms. Every time I was called the description of the odor was different, a real "mystery gas". Mass hysteria would have been a tempting diagnosis for me as industrial hygienist for the University to make, except I didn't know about mass hysteria back then and I naively assumed that if there were complaints, there was an explanation for them. Relying on out-of-date blueprints and lots of footwork, at long last I traced the problem to an empty drain trap inside the air conditioning housing on the roof of the building. Every time someone in a research laboratory poured waste solvents or chemicals down the drain, these volatilized through the drain trap and entered the ventilation system! The odors changed every time because the

chemicals that were being disposed of varied. Discovering the source of problems like this which involve many different factors functioning or malfunctioning simultaneously required real sleuth work by the industrial hygienist involved. We have all been faced with these challenges; resolving such a mystery for once and for all can be a rewarding experience. "Artifacts and "glitches" in contemporary heating, air conditioning, and ventilating systems are commonplace. Undoubtedly, many of the episodes described in this symposium have cross connections, empty traps, unbalanced ventilation systems, defective ductwork, short-circuiting of exhaust air as the simple explanation behind them.

What evidence is there that chemical exposures actually have occurred in the mass psychogenic illness episodes that have been documented? Unfortunately, many of the studies lack sufficient details to reconstruct retrospectively what exposures may actually have occurred. Data on heating systems, pesticide spraying, nearby construction, and other relevant sources of air contamination which could trigger such episodes often were not considered relevant to the investigation.

However, for purposes of illustration, a few cases of "mass hysteria" from the literature are reviewed in this light. More than a chance number of the episodes which have occurred have taken place in electronics assembly plants, or similar operations where a vast array of solvents, adhesives, fluxes, solders, circuitboard resins, and other esoteric organic compounds are handled; generally, one in large quantity. We know that unwanted overtime was imposed upon the workforce in certain plants so probably they were operating at peak capacity in order to fill orders and levels of air contamination from soldering and other operations were at their highest. Further, in many such operations, pay is on a piecework basis. One of the hypersusceptible women, whose case history was cited earlier told me that when the solderers at her plant are in a hurry, instead of stripping off the plastic coating on the wires, they simply burn it off. Although this is not good practice and is not endorsed by the management of the company, it is a frequent expedient. Clearly, if this is the case, then it is piecework pay practices which should be placed on the couch and not the worker.

One English physician who has studied electronics workers in that country from the point of view, discussed in this paper, has found that some workers who solder on wires that are bonded with pine resin, a type of widely used wire in the industry, become sensitive to the pine resin.*

In two of the episodes involving electronics assembly for which I was called in, workers at both plants commented to me that the episodes always occurred toward the end of the week, as if the concentration of the material was building up.

Interestingly, Hosen points out that when hydrocarbons or other gases cause reactions in susceptible individuals "it takes from one to seven days to accumulate enough of the gases to start the allergic state in action again."***

Finally, among those electronics operations in which mass hysteria has occurred, perhaps the greatest single piéce of evidence that chemicals were the cause is the fact that when ventilation systems are installed or upgraded, the outbreaks cease.

In an article entitled "Sandy," that appeared in New Yorker Magazine*** an epidemic of mass hysteria among children in a Florida elementary school was described. Symptoms included headaches, dizziness, chills, abdominal pain, shortness of breath and weakness. An industrial hygienist was called in and was asked about the "funny smell" present in the school. He "laughed" and said that it came from an adhesive used to secure a new carpet in the library, but that "the adhesive" was in no way toxic. Anyway, the carpet had been laid a good two weeks earlier. Interestingly, this episode occurred on a sunny Monday morning in May. Was it a hot day? We cannot tell from the account. The students had been away from the exposure over the weekend and then returned. Chemically susceptible patients cannot tolerate such carpet adhesives in their homes; if present, they must be ripped up and the adhesive scraped or removed with solvents in their absence. During a period of absence from such a chronic exposure, and then a return to the exposure, reactions are greatly intensified. Many workers complain of acute reactions on Monday mornings or following vacations. The fact that the odor of the adhesive was so apparent two weeks after installation of the carpeting should immediately raise questions as to whether the children have become susceptible to the adhesive.

Finally, it is of interest to note that of the children who were affected most severely, "one of these had a clear history of hypochondria. Another was always sick or sickly. Another had a habit of hyperventilating in moments of stress. Another had come to school that day feeling vaguely ill." In addition, one of the boys was a "chronic discipline problem", and the other was considered "highly excitable". Clearly, these children are the ones who are "chemically susceptible", and they further confirm that chemicals were the cause of the outbreak.

Stahl and Lebedun* reported an episode of mass hysteria which involved 35 female data processing workers who were exposed to a mysterious gas that caused dizziness, vomiting, nausea, and fainting among a number of the workers. Two incidents occurred on successive days. The events are interpreted in a psychological and social context by the authors, despite the fact that, 1) the women sat in a glass enclosed room,

whose ventilation source and efficiency is not noted in the paper, and that 2) the University, whose data processing center it was, was also constructing a new power plant on an adjacent lot. The authors report:

> "The constant noise of heavy construction machinery was frequently interrupted each day for a two-week period prior to the mystery gas incident by unexpected dynamite blasts. The coupe de grace, as reported by the workers, seemed to be the moving of a large diesel engine to drive pneumatic hammers from one side of the construction site to a location under a window of the Data Center."

If indeed the ventilation system picked up any of the construction gases or fumes and circulated these into the Data Center, this could easily be the cause of the symptoms reported by the women. Diesel exhaust, in particular, even more so than gasoline exhaust, creates a severe problem for the chemically susceptible patient. Many such patients when trapped in traffic behind a diesel truck or automobile will make herculean efforts to get in front of them or at least upwind to avoid becoming ill.

A diesel exposure may also account for the "epidemic of overbreathing" reported by Pfeiffer, involving 8 girls of a high school band that had travelled by bus 56 miles to a football game on an unusually hot day.* During the game, they developed severe abdominal pains and numbness in their hands. Even though the author comments that "several of the girls were stable and sensible people", he goes on to state his conclusions about contributory social factors, including the fact that their team lost the game as being responsible for the episode.

Randolph, however, who has worked closely with chemically susceptible patients and meticulously documented their symptoms and the exposures which provoked them offers an alternative etiology:

> "Due to the tendency of a moving vehicle to suck in its own exhausts through cracks and open windows, riders sitting in the rear of rear-engine diesel buses are more exposed to exhaust than those sitting or standing in front. This exposure is increased if favored by wind direction. The tendency for buses to follow each other closely also favors the entrance of exhaust of the lead bus into the passenger compartment of the following vehicle. Although this danger exists for any type of motor vehicle, it is especially marked in diesel buses and trucks...
> "Passengers often develop headaches or other reactions during or following bus trips, without suspecting

exhaust exposures. These effects usually are cumulative, often manifesting only after a certain mileage or time in the bus. In less susceptible persons they may manifest only when riding in vehicles in poor mechanical repair or only when the rear windows are open."*

Randolph goes on to describe one especially susceptible patient who exhibited abdominal cramps and diarrhea after riding only a few blocks in a diesel bus! In addition, on a hot day, plastics and other synthetic materials inside a bus are highly volatile and will provoke acute symptoms in the chemically susceptible individual. "Rubber floor coverings, plastic upholstery, and sponge rubber cushions and padding are associated with reactions in susceptible persons..."***

With regard to the girls on the schoolbus, clearly the cautious investigator would find out where they sat on the bus, whether it had a diesel engine, whether the windows were open, whether there was a caravan of buses, and whether the bus was in good repair, or had an odorous plasticky interior.

One does not have to be hyper-susceptible to react after three consecutive sprayings in so short a period. With such liberal spraying, one wonders if it was intended to drown the bugs!

Case History No. 6 concerned a man who worked adjacent to a large storeroom containing numerous pesticides and who also exhibited many neurological aberrations as a consequence of his exposure. The symptoms reported by the women at this plant are characteristic of those experienced by the chemically susceptible individual who is encountering a pesticide exposure unwittingly. Rea et al reported the history of a 37-year-old woman who worked in the food preparation area of a country club.** An automatic aerosol spraying system with twelve different vents was installed which discharged the pesticide pyrethrum automatically every fifteen minutes. When working near the pesticide dispensers, she would feel her esophagus begin to burn, develop extreme nausea, numerous disruptions in her thinking, ability, shortness of breath, numbness in her legs, tachycardia and spontaneous bruising.

With respect to the chemically hypersusceptible patient, Theron G. Randolph cautioned against pesticide use:

"DDT and related chlorinated hydrocarbons being relatively insoluble in water are usually dispensed in kerosene or other solvents. Whether the deleterious effects of such mixtures in chemically susceptible persons are due to the active principles or vehicles is often difficult to determine. The exceedingly high degree of susceptibility of many persons to such mixtures containing lindane, methoxychlor, DDT,

> chlordane, malathione or thiocyanates precludes their use as aerosols indoors...." Toxic insecticides, such as dieldrin, chlordane or pentachlorphenol are often used by professional exterminators for the control of termites and ants. These chemicals should not be used indoors--that is, in the basement or attics of homes of patients known to be susceptible to other chemical exposures. Once these materials are applied, it is impossible to remove them. The only alternative in certain extreme instances had been for susceptible persons to abandon such homes in order to control their chronic symptoms.*

The chemically hypersusceptible individual offers a unique opportunity for understanding the subtle effects of chemicals upon sturdier individuals. If even a few individuals are injured by exposure to certain synthetic chemicals, then all are exposed to stress.

The importance of "susceptibles" in the workplace was recognized early by miners who took canaries along with them into the mines. They also knew that some canaries were more resistant to toxic gases than were the miners, so the prudent miner was careful to select a canary that was known to be quite susceptible, or, to take several canaries along with him. The chemically susceptible worker is today's canary in the workplace.

> "The chemically sensitive persons have been likened to early warning systems. They are alerting us all that something is wrong, and with continued use, the same problems could show up in the rest of us who are less sensitive, and have merely taken longer to react."*

It can be assumed that if some are being irreparably injured by exposures on the job, then everyone is jeopardized. Excluding or prescreening the susceptible worker from a job, even if this could be done with technical certainty in the future, would only serve to lessen the measure of protection available to other less susceptible workers. Their health presence in the workplace provides assurance all is well. No animal toxicity studies, epidemiological investigations, or other scientific affirmations of safety can offer the same degree of certainty.

If a "susceptible" worker faints, vomits, or becomes mentally confused because of exposure to chemicals, then it is likely that many others are feeling fatigue, indigestion or irritability as a result of the same exposure, though they may not realize it.

To summarize what is known about Chemically-Induced Hypersusceptibility:

1. It is characterized by diverse symptoms, not limited to any part or system of the body. Its effects are often chronic and subtle. Though hypersusceptibility prevents most persons from working, it can "make life miserable."

2. It is triggered by molecular quantities of chemicals.

3. The majority of workers are probably affected; however, different persons will react differently to the same substance. This makes epidemiological study difficult.

4. Once it develops in an individual, hypersusceptibility tends to spread to many common chemicals and foods.

5. Hypersusceptibility may involve a disruption in hypothalamic functioning, which in turn affects immunologic activity, pancreatic enzyme production, and other important functions.

6. Two kinds of exposures seem to promote hypersusceptibility:

   a. A massive, overwhelming exposure, such as a chemical fire, spill pesticide spraying or working with chemicals in a confined unventilated space.

   b. Repeated, low-level exposure to a complex array of synthetic organic compounds as occurs in handling plastics, resins, soldering, etc.

To assist in guiding future investigations of episodes of supposed mass hysteria, the following suggestions are offered:

1. Rule out physical and chemical causes of illness before ruling in psychological ones.

2. Listen carefully to all clues offered by affected workers, no matter how obscure.

3. Review heating/ventilation/air conditioning systems. Were there recent changes? Is natural gas, coal or oil used for heating, or other operations? Look for malfunctions, design deficiencies, worn parts. Do complaints fluctuate with heating/cooling or operation of hot processes? Is plant air recirculated?

4. Check out "minor" complaints about lighting, noise, temperature, ventilation, odors, nearby construction, vehicle exhaust. Diesel exhaust?

5. Was plant operating at peak capacity so that ventilation capacity was taxed?

6. What operations do the workers suspect? What materials? Who has symptoms and where do they work? Is fatigue or headaches a common complaint?

7. Has a pesticide, germicide, disinfectant, or other chemical recently been applied throughout the plant? Symptoms may take a few weeks to show.

8. Has anything new--new furnishings, carpeting, wall coverings, etc.--been introduced within the past few weeks?

9. Randolph* devised a questionnaire which could be administered to affected and non-affected workers that could provide some estimate as to their sensitivity to chemicals. He also has described a test using synthetic (ethylene-derived) ethanol for testing chemical susceptibility in particular individuals. These could be quite useful in establishing the role of chemical sensitivity in such outbreaks.

Listen, listen to the patient's story! He is telling you the diagnosis, John Benjamin Murphy.

The woman worked in an electronics assembly plant whose case history was described earlier, told me she had had dozens of "little things" going wrong with her for thirty years, but nothing major that could be confirmed by a physicians's diagnosis or given a name. There are literally millions like her in the workplace who are wondering why they never seem to feel good, why they are irritable when they get home, sleepy all day long, and exhausted when they get up in the morning. They are not hypochondriacs or hysterics. Most are simply susceptible to chemicals with which they are working, or to which they are exposed. Our metabolism did not evolve to cope with the onslaught of synthetic materials that has occurred in recent years.

It has been shown that "subjective" symptoms of mass hysteria are identical in every respect to symptoms experienced by persons tested for susceptibility to various petrochemicals. Unlike a diagnosis of mass hysteria, chemical susceptibility can be objectively demonstrated by testing affected individuals with suspected incidents. Future efforts should be directed toward this goal.

The task of differentiating between an illness of the body and one of the mind is most difficult. However, the price of making a mistake is deep and prolonged resentment on the part of the workers.

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THE DIAGNOSIS OF MASS PSYCHOGENIC ILLNESS: SOME CAUTIONS

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It is the role of NIOSH, which is after all funding this session, to look not only at the science but the way the science interacts with the cause of worker protection, and I think that interaction has to be looked at very carefully. We have to look not only at the scientific facts but the way those facts can be explained and used in that cause of worker protection.

Let me begin with one specific incident. In August of 1969, a worker at a textile plant in West Virginia suffered what was called a nervous breakdown. The company doctors diagnosed his condition as acute schizophrenic reaction. He was hospitalized for 30 days.

While he was in the hospital, he noticed that there were three other men from the same plant in the same hospital with the same symptoms and the same diagnosis of an acute schizophrenic reaction.

Eventually, he returned to work but under heavy medication; he was receiving more than a hundred milligrams of prescription tranquilizers, powerful tranquilizers, a day. His problems continued; a year later his wife divorced him. The worker explained the divorce this way. He said, "It was me, my temper, not cooperating with her. I was mean to her but I couldn't help myself. I didn't want to be that way".

Well, I'm happy to report that the man and his wife are back together because they now know what caused the problem. As it turned out, the plant produced viscose rayon, and some of you may have already guessed the problem. The problem was carbon disulfide poisoning which produces all of the symptoms that the man and his coworkers in the hospital--and there were more than three--experienced. And through the help of their union and through the help of an excellent NIOSH Health Hazard investigation which came in and identified the problem, conditions are now better in that plant; the workers have taken steps to force management to clean up.

Now I'm not claiming that the poisoning of those workers was ever claimed as a case of mass psychogenic illness. For one thing, that particular term wasn't in vogue in 1969 as far as I can tell, but the point is that the company doctor and other doctors in the community diagnosed those cases as psychoses that were not necessarily related to work place conditions; they never talked about carbon disulfide poisoning and they certainly must have known better.

Carbon disulfide poisoning was first described by Paen, a French researcher, in 1856. It was studied by Alice Hamilton in the 1930's. Dr. Hamilton at that time described the response of the medical profession to the problems of carbon disulfide, and I quote from her book on Exploring the Dangerous Trades,

"The usual physician knew absolutely nothing about this form of occupational disease, and even when the victims reached asylums for the insane and were examined by specialists, the cause was not revealed for carbon disulfide insanity simply was not recognized as a clinical entity by American psychiatrists. There must have been company doctors who knew, but they kept the secret. This prolonged neglect both by the medical profession and the state governmental officials can be explained only by the fact that the most important plants were situated in states where labor laws and labor inspection were practically non-existent or very imperfectly administered and where no matter what sickness the worker contracted, he could claim no compensation from the company employing him. Under such circumstances, it is always easy for the employer to take refuge behind an ignorance which has been encouraged by the very man whose duty it is to enlighten him."

I tell this story to illustrate one of the perils of occupational health research, and that is the degree to which it's tied up with the perceived economic interests of large and powerful corporations and other entities in American society, and I think that's a unique situation, one that people coming into occupational health research have to be aware of.

Occupational epidemiology is very different from infectious disease epidemiology. Occupational health research is different from mental health research. Bacteria and viruses don't hire lawyers. Nobody lobbies Congress in favor of mental disease. Viruses don't get together in a trade association to hire consultants to critique scientific papers implicating them as a cause of disease. The manufacturers of toxic chemicals do all those things.

Now, this is not to indict all physicians, all occupational physicians, all company physicians, certainly not to indict all industrial hygienists who work or consult for corporations. There are many people, many I count among my friends, who are truly concerned and dedicated and I'm happy to report their numbers are increasing and I think we have seen lots of evidence of that in this Conference and others. But I think all of us have to be honest and admit that any theory that can be interpreted--perhaps misinterpreted as saying it's not the chemicals that are to blame, it's the mental state of the workers' that such a theory will be accepted happily, rapidly and all too gleefully by many plant managers and vice presidents for industrial relations and probably funded very heavily as well.

If you don't think that's a problem, let me relate to you one incident involving an alleged mass psychogenic illness that occurred in a steel workers' local, in particular in a small plant that assemble speakers using a variety of solvents and some very complex glues that are very ill characterized in the literature.

A large group of workers on one occasion got sick. The company now admits, although it did not admit at the time, that it was a breakdown of the ventilation system which exposed the workers to fairly high levels of a group of solvents and other chemicals. When the problem recurred a week later after the ventilation had been partially--and I stress partially--fixed, certainly not completely fixed, it was attributed to mass psychogenic illness.

Now, we think that diagnosis was too hasty, but that's not the point I want to make. The point I want to make is that the plant manager, on subsequent occasions, any time any worker has complained about a work place hazard, has attributed the complaint to "menopause maidens", that is the line in that plant, at least it was until recently when they got another plant manager, I'm happy to report. I'm unhappy to report that NIOSH is not too popular in that plant right now because many of the workers perceive that it was NIOSH that gave the plant manager the clout to do that and that has made things more difficult in that plant for us in terms of gaining better conditions.

Now, does this mean that researchers shouldn't diagnose mass psychogenic illness where they honestly believe it to be the case? No, of course it doesn't. What it means is that very special care must be taken, rigorous scientific objectivity has to be maintained and all alternate explanations should be rigorously checked and excluded before a diagnosis of mass psychogenic illness. Unfortunately, in past cases, these conditions have not been met.

Let me very quickly quote to you from an article which I was able to read this morning which is by Paul Shodoff, M.D., published in the American Journal of Psychiatry in which he says, "The term hysterical personality has become one of the most loosely used phrases in the lexicon of a profession not famous for the scientific rigor of its concepts." I think when we start applying that term to the work place and the very political atmosphere of the work place, that tendency can be increased.

Let me give you a few examples of where that's been a problem. In 1976, there was an outbreak of what again was alleged to be mass psychogenic illness at another steel workers local. I believe this is not now considered mass psychogenic illness by NIOSH but I want to read you from the local newspaper which quoted, and from every indication we've got this is an accurate quote, an OSHA expert flew to the plant and made a statement to the press after a preliminary walk-through investigation. He said "My impression is that there is no toxicity problem at the plant. However, tests will be made to bear out my findings." In other words, before he made a single air test, the man concluded that there were no toxicity problems at the plant, that he wanted to back that up with air testing, but that seems a little reversed.

One of the other papers that has been -- that I think will be cited later on in the course of this three-day session, is a paper that appeared in Lancet by, I believe, Ann McGuire. It was an investigation of another alleged mass psychogenic illness episode in which the particular symptoms were those of dermatitis.

Now, I'm not here to argue that this was or was not a case of mass psychogenic illness. I was intrigued, though, by one statement made by the author in the course of the paper, who had gone in and looked at the conditions and then said, and had concluded from a look at the conditions that the dermatitis was not occupationally-related and she says, "At this stage it was decided to patch test the women, despite the lack of evidence to support a diagnosis of contact dermatitis, primarily in order to allay doubts about the handled materials."

Now, the patch tests were negative, but that isn't the point. The point is that here is an obvious example of a preconception, of bias of someone who was patch testing not to investigate the cause of the disease but to allay doubts. That sort of thing is dangerous.

Let me give you one other example of what I think is the too-early acceptance of mass psychogenic illness as a phenomenon in a particular case, and this is from a paper that appeared in the Journal of Occupational Medicine back in June of 1978 by three NIOSH investigators, Mike Smith, Mike Colligan and Joseph Hurrell. It's entitled "Three Incidents of Industrial Mass Psychogenic illness".

Now again, I'm not here to critique this paper, but I am here to say that in the paper they report three cases of alleged mass psychogenic illness. The findings in one of the cases were reported as follows:

"Compared to the non-affected workers, those affected (1) tended to have less education; (2) tended to have fewer members of the household employed; (3) expressed a higher level of jobrelated stress; (4) reported that they were more frequently bothered by poor lightning and temperature variations; (5) reported taking more sick days; and (6) tended to score higher on the hysteria and depression scales of the MPI."

Now, it's possible those findings were absolutely valid, but it's my understanding that many of the particular--of these cases involved research in which questionnaires were administered to a large group of workers but where the response rate was in the neighborhood of 40 to 75 percent. That may not be the case in this study; we are not told in this study what the response rate was, nor are we told whether any of the findings were significant statistically, nor are we told any numbers.

Now again, it's possible that the findings were absolutely valid, absolutely statistically significant. I don't mean to criticize this paper. I do mean to criticize the Journal of Occupational Medicine. I can't imagine that journal accepting an article on, say, fine diameter fiberglass as a cause of respiratory illness in which the results were reported as "Those who developed respiratory illness tended to work around fiberglass, tended to have more time at the plant, and tended to be exposed to higher levels," with no tables, no numbers, no reported tests of significance and no indications as to what the percentage of the population studied was. I can't imagine that.

So I think that there is a danger that some of these things may be too early accepted.

Let me conclude with a few simple points. First, I'm not denying the existence of mass psychogenic illness or mass hysteria or another term that I have heard I think from Ann McGuire, which is psychic possession; that's the title of her paper, "Psychic Possession in the Work Place," which I think is quite a classic title. It certainly puts mass psychogenic illness to shame. And I have a vision of a new profession springing up of industrial exorcism.

But let me say that I'm not denying the existence of such things. I'm not denying the existence of mass hysteria or mass psychogenic illness or whatever you want to call it. On that point I'd like to remain agnostic until I know more, and I'm very happy the results of this proceeding are going to be published; I look forward to hearing the other speakers and reading those and I want to keep an open mind on that.

However, I do want to stress that a diagnosis of mass psychogenic illness should very much be a diagnosis of exclusion; I want to support what Mitch Singal says in the paper that he will present shortly: Chemical exposure should be rigorously ruled out. It's not adequate to do environmental sampling days after an incident and assume that exposures on the day of an outbreak of illness were similar, nor is it adequate to ignore low levels of new and complicated chemicals, tertiary amines, thioureas, other things which have been present in work places where some of these incidents have occurred, simply because there is no OSHA standard, no ACGIH TLV or no extensive literature.

It may be appropriate to also base a diagnosis of mass psychogenic illness on positive factors as well, but two of the factors usually proposed, that is transmission and respiratory alkalosis, can be mimicked by other factors. Transmission can be mimicked by a common exposure confined to a particular area of a plant on a particular day. Alkalosis can be caused by other conditions; I'm not a physician, so I took the liberty of consulting a recent medical text on the subject of hyperven-

tilation and subsequent alkalosis this morning and that text identified more than a dozen possible precipitating medical conditions, most of them were air, but including things like exposure to chemicals like dimentriphenol, anoxia caused by carbon monoxide, and by at least one common solvent, methylene chloride. So at its base, mass psychogenic illness has to be a diagnosis of exclusion; in fact, a diagnosis of last resort.

Unfortunately, given what I think are NIOSH's currently skewed research priorities, it's very difficult to adequately make a diagnosis of exclusion.

One of the great unanswered questions in this whole area is the question of sensitization to low levels of certain agents. I don't think that NIOSH's dismissal of that possibility in the past has been very convincing. I don't want to get deeply into that area; Claudia Miller will discuss it at length tomorrow.

The problem is that--well, at least somebody is going into the research of mass psychogenic illness. I think some are saying not enough. The problem is that almost no money from NIOSH, at least, has gone into the interaction between low levels of complex, ill-characterized chemicals, into the problem of chemical sensitization and subsequent symptoms which may mimic mental disorders, or psychogenic reactions, and certainly very little money has gone into the interaction between toxic chemicals and physical stress.

I think that lack of funding can only be called irresponsible. That kind of research is difficult but that's not an excuse for failing to attempt it, and without more information on that area, it's impossible to exclude with confidence the possibility that any particular incident is in fact an example of low level chemical sensitization and thereby make a diagnosis of mass psychogenic illness.

But I think some--let me throw in a historical reference as well. I think some of the--the past has seen many things which were thought at one point to be psychological but turned out, after later research, to be chemical in origin. I can't go back a thousand years, but I can go back to 1713, to Remizini, who talked about the diseases in his book,Diseases in Workers in 1713, talked about a variety of work place conditions causing ill health.

At that time, many of these things were attributed to psychological conditions; they didn't call them psychological conditions, the term wasn't invented, and Remizini, in describing the diseases of painters, gave reference to that. In describing the diseases of painters he said "Their sedentary life and melancholy temperament may be partly to blame" for the diseases that he mentions, "for they are almost entirely cut off from intercourse with other men and constantly absorbed in the

creatures of their imagination," in other words, psychosocial factors. He goes on to say, "But for the liability to disease there is a more important cause; I mean the materials and the colors that they handle and smell constantly, such as red lead, cinnabar, white lead, varnish, linseed oil, etc."

In other words, at the time Ramazzini wrote, the diagnosis of these things was shifting from psychosocial factors to chemical factors; that may well happen in this case. We don't know without more research.

Third, I think we have to be very careful to consider our own subtle social and sexual biases, and perhaps not so subtle social and sexual biases in those who report such incidents. I have seen a number of cases in mills and other work places where a group of men have gotten--one or two have suffered what they term symptoms, a number of people have gotten scared, walked out of the work place; that's been called a wildcat strike by management.

Those incidents sound suspiciously like incidents which have been called mass psychogenic illness where women are involved. Now, I'm not saying that the earlier cases of men were not mass psychogenic illness. What I am saying is that we should be very careful not to too-easily accept some kind of sexual difference in susceptibility to any kind of disease.

Fourth, we have to be very careful with terms. Some of the reactions that are being called mass psychogenic illness seem to me to be reactions which have in the past been called stress. Stress, as you know, refers not only to stressful conditions but to a physiological reaction, and I think in those kinds of cases it might be more appropriate to call it stress than to call it mass psychogenic illness.

You may wonder why the term is important. I think the term is important because the term stress more easily leads a layman, a worker, a plant manager, to an understanding that it's work place conditions that are involved; physical conditions, in some cases social conditions of the work place rather than a pathology of the individual who reacts, which is unfortunately what the term psychogenic illness conjures up.

And finally, let me repeat a plea for rigorous objectivity and very great caution in this kind of thing. I think--and let me give you the reasons for that. The reasons are that the consequences of a mistaken diagnosis of mass psychogenic illness are very grave. They include first the possibility that very toxic chemicals or the interaction between toxic chemicals may go undiscovered with a possibility of severe future incidents and a substantial loss to worker health.

The other possibilities of a misdiagnosis include a general

loss of confidence in the government and a general loss of confidence in NIOSH in particular. I know of one incident where, in the very first or second NIOSH visit to a plant, a psychologist came on the visit. There was a little incident where everyone was sitting around and introducing themselves to a group of workers: the industrial hygienist introduced himself, the physician introduced himself, and then the psychologist introduced himself as a psychologist. Now, I'm not saying he shouldn't have been there. What I am saying, though, is at that point, cooperation with the workers became very, very difficult because the immediate conclusion to which they jumped is "They think we're crazy." And it was very difficult for the union to convince people to cooperate with them and NIOSH is not popular in that plant either, right now, so again, the consequences are grave.

So what I'm closing with is a plea that you consider this problem carefully, you consider what psychogenic illness is and you consider perhaps even more carefully what it is not, and that you take into consideration the effect of your actions on worker health because that, after all, is the bottom line; that's what NIOSH is for and that's what the AIHA is for.

Thank you.

MS. BERTINUSON: I can limit my remarks very greatly since Michael has spoken some of my points already.

I, too, would like to start out with a little bit of historical perspective, again from Alice Hamilton's book, Exploring the Dangerous Trades. And I will quote you from that.

"I was reminded of an interesting incident in a rubber factory during the Tsar's days which was related by a Russian doctor in 1914 in a German medical journal, since he was forbidden to publish it in Russia. He tells of a mysterious outbreak of illness among women working with a rubber solution in a large factory in St. Petersburg. A new solvent had been introduced and soon after, many of the women began to complain of headache, dizziness, nervousness, some were greatly excited, some fainted, others had convulsions. The foreman evidently did nothing sensible about it and by the end of four days, 231 had been affected. The tales spread and with it the rumor that the employers were starting a wholesale murder of the workers, so the sickness spread to the women in chocolate and tobacco factories. The employers lost their tempers and declared a lockout, whereupon the Duma voted an emergency fund for the thousands turned out in the death of winter."

"Naturally, the doctors were much interested and eager to discover whether this really was an incidence of intoxication by a volatile solvent or only mass hysteria, but they were not allowed to find out for the Chief of Police stepped in, declared it was all the work of radical agitators, forbade any discussion of the subject by a group larger than three, and refused to let the Duma distribute any relief."

"The factories were reopened after the workers had been brought to a proper state of meakness and the incident was closed."

"About the same time, a similar situation developed in a rubberworks in Moscow where a quiet investigation revealed the illness to be acute benzol intoxication, for the new cement had been made with almost pure benzol. The illness in the chocolate and tobacco factories was undoubtedly hysterical."

I started off with the quote because I think it brings out two points that will be brought out in the course of this symposium. The first is, if we look at the chocolate and tobacco factories, those women were not exposed, from what Hamilton says, although she did not investigate the situation herself, to the benzol, to the solvent in the high concentrations that caused the problem in the other factory. In the other factory we indeed had a situation, again from her understanding, where there was acute benzol intoxication causing the symptoms which are very much associated with that kind of exposure.

So I would like to start out as Mike finished, by saying that

in more recent studies of incidents that have been labeled mass hysteria, assembly line hysteria, mass psychogenic illness, there have been trigger mechanisms that have varied from place to place--in this situation in Russia,that I described the trigger mechanism was this rumor, actually the workers were becoming sick and the rumor spread that the employers were trying to do away with all the workers, and in these cases that have been reported in the literature, most of them by NIOSH, it seems tenable that when they have explored to some degree the exposures to toxic substances in those work places, there seems to be a trigger response that's reported throughout all of these different studies.

It seems that we should indeed look at the possibilities of psychological problems contributing to an incident of mass illness, and again, with Michael, would like to state from the beginning that I do not disbelieve that there is such a thing as mass illness that can have psychological factors contributing to it, or such things as stressful working conditions, as was pointed out in many of the studies done by NIOSH, but I think as Michael pointed out, there are several questions that have to be raised and I think this is the time to raise them.

I think as we are talking about developing a methodology to evaluate incidents of mass illness that may be due to psychological or partially due to psychological factors, we have to look at some of the other possibilities.

The first one certainly is whether or not, when we talk about low level exposures to substances, even getting away from the whole idea of a hypersusceptibility reaction, whether by saying a substance is below the permissible exposure limit or the federal standard, we automatically then say that this level is safe. I think if we look at permissible exposure levels, TLV's or whatever, we look at as a standard of exposure, and ask ourselves do we consider this a safe level of exposure, we all have to answer, I would hope, no. They are considered guidelines; that was one of the first things that we learned about TLV's, that they are guidelines, and indeed, in terms of long-term exposure to low levels of substance, we know very little in general about the majority of substances used in the work places.

The descriptions that are used in primarily NIOSH Health Hazard Evaluations, which are the main reports I have read, to describe the levels of exposure, are such things as "non-toxic accumulation of odors, potentially irritating, below harmful levels, within current standards, safe levels of exposure, significant levels not present," and I would suggest the first place we have to start is looking at the significance of those statements. What indeed are we talking about in terms of safe levels of exposure.

Following that, I think one point that Mike raised is also very important and that is the interaction of chemicals in a given work environment, because at this point in time, we know so little about the interaction not only of chemicals in the work place environment, but also in the bodies of workers. To exclude the possibility of interaction I think is untenable, and again in a majority of the reports that I have read there has been no discussion with one exception, of the interaction of chemicals in the report itself. Now, I do not know if the possible interaction was considered.

Another possibility again that Mike mentioned was the hypersusceptibility which I will not even deal with at this point.

Finally, many of the reports point out that various stressors in the work place environment, such as poor labor-management relations, the stress of having to work on boring, repetitive assembly line jobs--most of these cases were in those kind of work places--mentioned that these stressors are possibly or probably the causative agents or contributory to many outbreaks of mass illnesses that were studied. I think, then, that we have to look at the contribution that stressors of a person in an environment where they feel overly-stressed because of conditions in the work place, how their body reacts to low levels of chemicals and the fact that the low levels can indeed make them more susceptible.

We know that when someone is under stress, when they are experiencing a stress reaction, that their body cannot deal with other insults as well as it normally could, and I think that we have to look at the fact that in this kind of situation, that insults in the form of chemical insults are probably not as well dealt with by the exposed person.

Okay, I believe that those questions are basic ones that must be answered and must be looked at because we are seeing increasing reports of outbreaks of mass illness, and the descriptive terms such as mass hysteria--particularly mass hysteria--and mass psychogenic illness are being tossed around fairly freely by a lot of different types of people and they are--the term is being grabbed onto, as Mike mentioned, as a way of explaining away the situation in a work place without in some cases adequately evaluating the situation in a work place, and I think you can all see the danger of that.

The term is also being picked up by the media. It's a very sexy term at this point in time, and I'd like to point out that there are other people who are picking up the term. There was an example just recently, this was reported in the San Antonio Saturday Express News, May 5, 1979, and it was an incident where 45 women were rushed to a hospital after an odor infiltrated their plant; they manufacture curtains.

The newspaper account doesn't give very much information about the whole situation, what the final resolution of it was at all, but I think it's interesting that in the paper, the person who made the diagnosis and who is quoted is the fire inspector. He said "Apparently there was some seepage of butal mercaptan into the fabric. Most people do not get ill from the smell", said Remetaria, who is the fire inspector, who speculated "some of the women were victims of mass hysteria," and I think that that kind of thing points out one of the dangers that we find ourselves in when we pick up a description of a mass illness as mass hysteria, pick up a term that's easy to be coined without looking at some of these other possibilities.

I'd like to give one example of a situation that was never labeled as mass hysteria. It was never labeled as mass hysteria principally because in this particular work place, there was a very strong union and they saw to it that the situation was very thoroughly investigated and evaluated, but there were certain points in time when the company refused to let the workers stay out of work, refused to pay sick pay, because they could not find the cause, and I think that had the incident occurred a few years later, this occurred four and a half years ago, that the term mass hysteria might very well have been used.

This was a warehouse employing people, primarily women, in packaging pharmaceuticals. It's a fairly large warehouse; most of the women who were initially affected were working on a packaging line next to a loading dock. There was an occurrence, the first thing that happened was the sprinkler system was drained and there was an odor, some kind of noxious odor in the warehouse. Six women in the Shipping Department, one of them five months pregnant, became extremely ill and they exhibitied some or all of the following symptoms: headache, nausea, vomiting, dizziness, trembling, muscular weakness, severe drowsiness, and some of them fainted.

They were sent to an industrial clinic where they were seen by a physician who did very cursory examinations and sent them all home. The company shut the plant down and sent everybody home. Most of the women reported that they slept a great deal over the weekend.

A sample was taken from the sprinkler system--there was an assumption that there must be something in the sprinkler system. Nothing was found that could be related to these kinds of symptoms.

The women returned to work at varying times, some leaving again because they were still sick.

Our program came into the situation, did a walkthrough and discussed some of the possible sources of the odor and the illness

with the company, and the company was at that point very cooperative. However, following the initial week when the company did keep the plant shut down, more workers--the workers eventually came back to work and again people in the Shipping Department were becoming ill. At this point the company no longer was as cooperative, would not shut the plant down,and would not let the people go off work with sick pay.

Again, there was another mass incident and approximately nine people became ill. They were then sent to the industrial clinic where someone had the bright idea to do carboxyhemoglobin levels and indeed, the carboxyhemoglobin levels in these particular people were quite elevated, significantly elevated.

There was subsequently a CAL-OSHA inspection and the CAL-OSHA inspector found the fork lift used in that area, which was kind of wandering around that area for all the time the women were working there,there was also a loading dock where trucks were coming in and out every day; found that the fork lift, the levels in the vicinity of the fork lift were between 500 and 600 parts per million of carbon monoxide. Now, certainly there was some dispersion of carbon monoxide in the air so it would not be that high around the women, but I use this incident to point out that there was a good possibility in that particular case that the situation would have been labeled a mass hysterical illness or assembly line hysteria, whatever you want, and it was not because the union took steps to make sure the situation was fully evaluated.

The last thing that I want to point out--and I have nowhere near enough time to look at a particular study that I think is rather important--is the importance again as we are beginning to look at something we are calling mass hysteria or mass psychogenic illness, the importance of developing good methodology and good epidemiology looking at these particular incidents is extremely important.

There was one incident at the Perry Norvell Shoe Company, which is, I guess, a subsidiary of Kinney Shoes in West Virginia, where there was an incident that was labeled mass hyperventilation syndrome or mass psychogenic illness and it occurred in 1977. Again, I don't have time to go through the whole investigation. What I do want to point out, though, is in the course of this investigation a number of women, four women became ill initially in working around a glue table operation. They asked to have the glue changed. It was changed; more people became ill.

Eventually, some workers called the local sheriff and fire department and there were a number of emergency vehicles that came in and a lot of people then started fainting, people were taken out of the work place, and subsequently a large number of people were taken to the hospital. That's condensing a

very long, drawn-out thing into just like a minute or so.

Okay. So NIOSH went in eventually and did a study of the particular incident. The problem again was they did not get in there right away. They came in, as a matter of fact, a number of days after the initial incident. There were several things that were done as part of that investigation; standard environmental monitoring, a walkthrough survey, looking at the ventilation and so on, medical evaluation, and a psychiatric questionnaire or psychological questionnaire was administered.

There are a lot of problems with this particular study that I'd like to point out as things that have to be corrected if we're going to look at the results of other such studies and make a conclusion, a valid conclusion about what is the causative agent in these particular situations.

Number one, when the sampling was done, there is no indication in the investigation report as to why certain substances--in this particular case, toluene, carbon monoxide, carbon dioxide and nitrogen oxide--were sampled for when there were numerous glues and other substances being used. I can't critique this fully because I don't know what the substances were; the report does not say.

The sampling was done after the plant was evacuated the first time, the second time when the plant was not operating, and the third time on December 8th and 9th of 1977 when there was not as much activity on the glue table as at the time of the outbreak, so the first problem is there was a failure to reproduce the original conditions, working conditions.

In terms of the investigation, the medical evaluation, seven persons were interviewed. These were seven persons who went back to work out of --120 of 400 went back to work and they were either persons who were in the plant or on an informational picket line outside the plant. They were not identified.

There is an obvious bias in that selection of those people for a medical evaluation. They did not, after talking to these people they decided not to look at the medical records. The tests that were done at the hospital, although for one person it says that the lab findings suggest no exposure to toxic substances or significant exposure to toxic substances, the lab tests were not laid out, nor were the results described.

The response to the questionnaire, "approximately" 120 workers were given the questionnaire when they reported for work, "approximately". An additional 80 "or so" at a union meeting. Now, it seems to me that if you're going to talk about a population that you are studying, you should know the number of people who have received questionnaires.

The response rate of the questionnaire was 18 percent. I would say with that kind of a response, I don't even see why there was any bother to report on the results of that questionnaire.

There was also--a lot of the conclusions were based on, according to the report, anecdotal data, talking to union people and other people in the area, and it seems to me that that kind of information used in making a decision, that creates real problems.

A final problem is that they never investigated the four index cases, or if they did, they were not indicated in the report. And again, you can't really talk about a representative survey if you don't know the number of people that you're sampling.

Now, the reason I bring this particular Health Hazard Evaluation out is because there were some real problems that I found; I'm not an epidemiologist but I could go through and pick out these things that I consider to be poor reporting and just poor epidemiology. The reason I bring it up again goes back to what Mike was saying and I was saying: the care that must be taken in looking at any kind of a mass illness affecting a large number of workers when you're dealing with terms that workers feel you are in effect saying to them, "you're crazy".

And I think there is a feeling about that. The women from--one of the women from this particular plant, it's a retail clerks local, basically believes the fumes did make the people ill and she has not been convinced otherwise and she basically calls the NIOSH theories a bunch of bunk.

Now, I think the real problem--I don't think that's funny. I think the real problem is that that lowers the credibility of our agencies that are doing good research and I think that creates a problem, again one that Mike brought out, about workers having a problem with NIOSH coming in at further points, so again, I would stress, as Mike did, the necessity to be extremely cautious at this point in time and take a very good look at the methodology that we are using to evaluate these incidents.