SUMMARY STATEMENT FROM THE EPA ADVISORY PANEL ON HEALTH EFFECTS OF PHOTOCHEMICAL OXIDANTS

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Prepared for the Environmental Protection Agency
under the supervision of the Institute for Environmental
Studies of the University of North Carolina at Chapel Hill.

I. Composition of the Panel

The Advisory Panel on Health Effects of Photochemical Oxidants (hereafter called the "Health Panel") was convened at the request of the Office of Air Quality Planning and Standards, U. S. Environmental Protection Agency. The Institute for Environmental Studies, University of North Carolina at Chapel Hill, served as advisor in the selection of panel members and as host for the panel meeting held in Chapel Hill, N. C. on June 7th and 8th, 1977. Panel members were:

Carl M. Shy, (Panel Chairman), Director, Institute for Environmental Studies, University of North Carolina at Chapel Hill

Stephen M. Ayres, Chairman, Department of Internal Medicine, St. Louis Univeristy School of Medicine

David V. Bates, Dean, Faculty of Medicine, University of British Columbia

T. Timothy Crocker, Chairman, Department of Community and Environmental Medicine, University of California College of Medicine, Irvine

Bernard D. Godlstein, Associate Professor, Department of Medicine and Department of Environmental Medicine, New York University School of Medicine

John R. Goldsmith, Environmental Epidemiology Unit, California State Department of Public Health

Staff and scientists of the U. S. Environmental Protection Agency participated in the discussions of the Health Panel; documents prepared by FPA staff were distributed and, where appropriate, commented on. The conclusions and recommended guidelines for protection of public health given in this summary statement represent a consensus of the panel members only.

II. Purpose and Scope of the Meeting

The purpose of the panel discussions was to interpret the current state of knowledge on health effects of ozone and other photochemical substances, with the objective of developing a guideline to the Environmental Protection Agency (EPA) for the protection of public health. These discussions were prompted by EPA's current program for review and re-assessment of the existing air quality standard for photochemical oxidants.

The panel reviewed studies relating to the following categories of health effects:

A. Human studies

1. Mechanical function of the lung (controlled human exposures)
2. Asthma and lung function in children
3. Athletic performance
4. Other effects: mortality, occupational hazards

B. Toxicological studies

1. Experimental infection of animals
2. Morphological abnormalities of the respiratory system
3. Biochemical effects
4. Mutagenic and teratogenic potential
5. Performance and behavioral effects

In its discussions, the Panel considered several issues bearing on the overall interpretation of reported studies. Among the major issues were: relative weights to be given to published and unpublished reports, concept of threshold concentrations for effects, human health significance of toxicological data, chemical specificity of the air quality standard ("ozone," "oxidants," "photo-chemical substances") margins of safety, and the health significance of exceeding a stated concentration one or more times.

III. Format for Discussion of Health Effects

The EPA staff requested the Panel to consider three characteristics of population exposure that bear upon protection of public health: the concentration at which human health risk would be increased, the duration of exposure which is related to this risk, and the temporal exposure pattern that would be associated with increased risk. In its deliberations on each category of health effects, the Panel addressed the probability that the health effects are induced by exposure to ozone or other photochemical substances, the severity of each health effect and the uncertainties of the evidence.

IV. Concept of Threshold Concentrations

A threshold was defined as a concentration between a no-effect level and the lowest concentration at which a health effect was demonstrated. Identification of a threshold relevant to protection of public health requires evidence for specific exposures that produce no effect as well as exposures that produce effects in susceptible segments of the population. The Panel emphasized that biological reactions to pollutants are not characterized by sharp discontinuities in dose-response relationships, and that demonstration of no-effect levels is dependent upon the sensitivity of the measurement of effects and exposure, as well as the selection of the most sensitive groups and reaction systems. Since "thresholds" will depend upon who is studied and what is measured, it is unlikely that scientific evidence for a specific effects threshold can be satisfactorily derived from existing data. Limited studies can be performed on groups of unusually sensitive persons. Most experimental studies of humans are

performed on small numbers of healthy subjects who do not adequately reflect the range of human sensitivity. Toxicological studies usually cannot utilize appropriate models of sensitive human populations. Thus, the Panel concurred in the statement that thresholds for sensitive persons are difficult or impossible to determine experimentally, while the threshold for healthy persons or animals is not likely to be predictive of the response of more sensitive groups.

The Panel discussed alternatives to the "threshold" concept for arriving at recommended guidelines for protection of public health. An acceptable alternative considered by the Panel was

(1) to state at what level health effects have been demonstrated in population groups, in controlled human exposures, and in experimental animals,

(2) to evaluate the relationship between the responses of subjects or animals studied and sensitive segments of the population,

(3) to assess the severity of each category of effect,

(4) to recognize the uncertainty in this body of evidence,

(5) to propose guidelines for a margin of safety, given the strength of the evidence, the severity of the effects, and the magnitude of the uncertainties,

(6) to recognize that decisions on margins of safety involve more than scientific evidence.

V. Acceptabiliy of Cited Reports

In their assessment of evidence from the various sources of information on health effects, Panel members agreed that greater weight should be given to peer reviewed publications than to unpublished reports or reports published without this review. The Panel felt that the latter category of data should be considered, but could not serve as a major contribution to its recommendations concerning public health protection. Overall,

there was a sufficient body of peer reviewed publications to serve alone as the basis for the Panel's recommendations. In the one instance where a significant human effect was demonstrated at a low ozone exposure, the Panel took account of the data but made its conclusion primarily on the basis of other published evidence.

VI. Human Studies

A. Mechanical function of the Lung

The Panel focused its discussion on the more recently published human exposure data as reported by von Nieding, et al. (1977a, 1977b), Hackney et al. (1975a, 1975b, 1975c, 1977), Bates and Hazucha (Bates and Hazucha 1973, Hazucha 1973, Hazucha et al. 1973, Hazucha and Bates 1975). The Panel agreed that there was convincing evidence for pronounced effects on mechanical function of the lung, as variously measured by flow rates, airway resistance and other parameters of ventilatory function, at ozone exposures of 0.37 to 0.75 ppm for two hours under conditions of intermittent moderate exercise. The Panel also accepted the validity of findings showing an effect on lung function at 0.25 ppm, but recognized that these effects were less pronounced than at higher concentrations and that they occurred in Montreal subjects and not in Los Angeles subjects studied under identical protocols. The difference between the dose-response relationships for Los Angeles and Montreal subjects was felt to be explainable by one or more of the following factors: adaptation in Los Angeles subjects, admixture of submicronic aerosols in the Montreal chamber, and/or selective migration away from Los Angeles of persons who could not tolerate previous repeated ambient ozone exposures without undue reactions.

The Panel also identified the fact that a plateau in the dose-response relationship in Montreal subjects was not demonstrated, and therefore that effects may be produced at even lower concentrations than those employed. In support of this hypothesis were the data from recent publications of von Nieding et al. (1977a, 1977b). Differences from other study protocols in the measurement of airway resistance and of arterial partial pressure of oxygen were recognized. The small standard error about mean values of airway resistance was also noted. The Panel felt that these aspects of the von Nieding studies required replication of their results by other investigators but that the data was not thereby invalidated. The Panel concluded that results of the von Nieding studies served to reinforce the conclusion that changes in mechanical function of the lung may well occur in some subjects at ozone concentrations less than 0.25 ppm for two hours, and that there may be some risk of inducing functional changes at levels in the range of 0.15 to 0.25 ppm.

The Panel considered the issue of repetitive experimental exposures, and asked whether effects on mechanical function would be more severe with repetition of exposure or would possibly occur at lower concentrations with repetition of exposure. The Panel consensus was that the risk of effect was related to the total dose of ozone delivered to the respiratory tract within a day (but not over long periods), and that this dose increased with the frequency of exposures, with the concentrations of a single exposure, and with the intensity of exercise of exposed subjects.

The Panel concluded that the evidence for a relationship between ozone exposure and effects on mechanical properties was conclusive. In discussing the severity of this effect, the Panel agreed that one exposure

ay not portend risk of serious consequences in healthy individuals. However, a single exposure of a sensitive individual such as an asthmatic, or other persons with airway disease, may induce a serious health effect, and repeated exposures even of healthy individuals may lead to increased risk of respiratory impairment in the form of irreversible effects or susceptibility to chronic respiratory disease. However, the Panel recognized that judgments concerning repetition of exposures fall in the area of greatest uncertainty because experimental human studies have not been conducted to evaluate repetitions of exposure. The Panel also cited recent experimental evidence that the maximum stimulus to histamine release in the lung occurred 24 hours after ozone exposure, suggesting that exposed persons with sensitive airways may experience untold delayed effects. These findings are not fully understood and generally have not been incorporated into the assessment of ozone induced health effects.

B. Asthma and Lung Function in Children

The Panel reviewed the air quality data available for interpreting the findings of the Schoettlin and Landau (1961) asthma study. Although there was some confusion in the early reviews of this study, it is now clear that oxidant measurements for each day were made by the Los Angeles Air Pollution Control District, that these data were obtained by the potassium iodide method, and that there were significantly more asthma epidodes in subjects on days when peak oxidant concentrations exceeded 0.25 ppm, and finally that these peak concentrations were associated with average maximum hourly oxidant concentration of about 0.20 ppm. From these data, the Panel agreed that the evidence supported the statement that a proportion of asthmatics will be affected by maximum

hourly oxidant concentrations of 0.20 ppm, and that the effect is likely to occur at concentrations in the range of 0.15 to 0.25 ppm in some asthmatics or other persons with sensitive airways.

The Panel also considered the recent reports of Kagawa and Toyama (1975) and Kagawa et al. (1976) on the association of changes in lung function of school children with oxidant exposure. These published reports show a decrease in ventilatory function of school children associated with increasing ambient ozone concentrations, from 0.1 to 0.30 ppm. The Panel noted that the authors stratified the data into low and high temperature seasons, but could not isolate the effect of ozone from other measured pollutants, since population exposures only occur in the presence of pollutant combinations, not for single pollutants in isolation. The Panel concluded that these studies further supported the evidence for an increased health risk from ozone exposures over the range of 0.15 to 0.25 ppm, and for the likelihood of a lesser but real health risk at even lower concentrations.

The Panel expressed additional concern for ozone exposures of young children, in view of the findings of Bartlett et al. (1974) which demonstrated a reduction in lung elasticity and overdistention of the lungs of rats exposed at 3-4 weeks of age for 30 days to 0.2 ppm ozone. The Panel felt that these data were particularly significant since there is continuous growth of lung capacity, in humans, both in terms of number of alveoli and ventilatory function from birth to age 8 or 9 years, and since ozone exposures which compromise lung development at these ages might have serious implications for risk of impairment later in life.

The evidence that ozone effects may be enhanced by other concurrent pollutants (Bates and Hazacha 1973) was interpreted by the Panel as indicating the desirability of providing a margin of safety between these observed effects and the primary standard for ozone and other photochemical substances.

C. Respiratory and Other Symptoms in Human Populations

Experimental exposures of humans and epidemiological observations support the conclusion that ozone exposures in the range of 0.15 to 0.25 ppm are associated with increased risk of cough, chest discomfort, susternal soreness, headache and eye irritation. Respiratory symptoms are enhanced by more intense exercise. The Panel judged that the attempts to obtain threshold estimates for these effects (Hammer et al., 1974) violated biological evidence for nonlinear dose-response relationships, and that in general, segmental regression analysis ("Hockey-stick" function) is inappropriate for determining the onset of health risk.

In reviewing the several Japanese reports on acute respiratory and other symptoms in school children during "photochemical smog" episodes, the Panel noted the occurrence of acute effects at ozone levels of 0.15 ppm and above. The Panel attributed the high rate of reporting of symptoms in the Japanese episode to a combination of several factors.

(1) biological reactions producing manifest symptoms, particularly in actively exercising school children,

(2) Probable combination of ozone with other pollutants which interact in their biological effects,

(3) sociological factors which in one culture may result in repression of perceived symptoms and in another articulation of perceived symptoms, and

(4) psychological factors which may alter the individual's judgment concerning the severity of symptoms.

One or more of these factors may account for the apparently high incidence of chest discomfort and eye irritation in affected juvenile groups and for the associated extrapulmonary manifestations such as numbness, fainting and necessity for hospitalization. Thus the Panel concluded that the Japanese reports of symptoms at 0.15 ppm should be given due weight in arriving at a guideline for public health protection. The repetition of these episodes with associated symptoms adds to their significance.

D. Athletic Performance

The Panel's previous comments concerning the inappropriateness of segmental regression analysis was stated to apply to Wayne et al.'s (1967) study of athletic performance. The data as presented in the original study do not suggest a plateau in the dose-response function. Nevertheless, some members of the Panel were unconvinced of an association between impaired performance and oxidant concentrations less than 0.15 ppm. However, these members stated that their judgment was based on a visual truncation of data points rather than on quantitative statistical analysis. The Panel noted that a recent paper (Folinsbee et al. 1977) has documented a decline in maximal oxygen uptake in healthy young subjects exercising under controlled experimental exposure to 0.75 ppm ozone, thus suggesting a mechanism for the effect on athletic performance.

E. Other Effects: Mortality, Occupational Hazards

Review of existing studies fails to show any evidence for increased risk of mortality in association with daily oxidant concentrations measured in the Los Angeles basin. The Panel stated that there was no new evidence to alter this conclusion.

The Panel expressed concern with the absence of studies of different groups occupationally exposed to ozone. These groups include airline pilots and crews, workers exposed to coronal discharges in the electric

utility industry, and other persons working in the vicinity of ultraviolet lights, e.g., in cold storage rooms. The Panel was quite convinced that some adverse reactions would be produced in these occupational settings, and expressed concern that the absence of systematic studies will lead to continued exposures without identification of the induced health risks.

F. Conclusions from Human Studies

The Panel reached consensus on the conclusion that short term exposures to ozone in the range of 0.15 ppm to 0.25 ppm may impair mechanical function of the lung, and may induce respiratory and related symptoms in sensitive segments of the population. These symptoms and effects will be more readily induced in exercising subjects, particularly in a complex urban atmospheric environment in which ozone can interact with other pollutants.

Short term exposures only 3 or 4 times this level, that is, exposures to 0.75 ppm for two hours, can induce severe symptoms in exercising subjects. Such experiments are ethically unacceptable.

The Panel judged that the occurrence of respiratory symptoms and alteration of mechanical function of the lung have important public health implications, particularly for the developing lungs of young children. Although such effects appear to be reversible in exposed young adults, they represent a potentially serious risk for asthmatics and other individuals with airway disease. In the population of individuals with varying states of biological adaptability, exposures which produce the above described effects may at times overwhelm the biological defense of some persons. Thus reversibility of effects in experimentally exposed healthy subjects should not be generalized to the entire population.

The Panel related many of the experimental human effects to a two-hour averaging time. However, the Panel noted that more intense exercise was

likely to bring about respiratory symptoms and ventilatory function effects within a period of one hour. Panel members concurred that it was not possible to perform a fine tuning on the averaging time associated with this category of effects, and agreed that a one-hour averaging time represented a satisfactory estimate of the exposure duration which should be considered in a primary air quality standard aimed at protection of public health. To some extent, a one-hour averaging time may provide a desirable addition to the margin of safety, in that it may better protect actively exercising persons.

VIII. Animal Toxicology Studies

A. Experimental Infection of Animals

Increased susceptibility to bacterial infection following ozone exposure at 0.1 ppm is described by several investigators (Coffin et al. 1968, Ehrlich et al. 1976, Gardner et al. 1974) in reports that do not fulfill the desideratum of peer reviewed publications. These reports are, however, consistent with a larger body of published evidence (Coffin and Gardner 1972, Goldstein et al. 1971a, 1971b, Goldstein and Hoeprich 1972, Goldstein et al. 1974 and others) that establishes indices of infection or of mortaility from bacterial infection as sensitive measures of the effect of ozone in rodent lung. Additional stress such as heat, exercise or a combination with other pollutants, according to some reports, may enhance the effect of ozone on susceptibility to infection, and may thereby lower the ozone dose at which the organism will be adversely affected.

The Panel agreed that these finding have definite human health implications although an exposure level associated with such effects in humans may be different. These reactions in mice represent basic biological responses to infectious agents, and there is no reason to believe that pollutant induced alterations of basic defense mechanisms in experimental mice would not

occur in similarly exposed and challenged humans. However, the Panel is not aware of epidemiological evidence that susceptibility to infection increases in persons exposed to ozone and other photochemical materials. However, the biochemical and cellular alterations described below for rodents suggest that multiple epithelial and biochemical targets are perturbed by ozone exposure. Since similar epithelial perturbations occur in humans when viral infection precedes the onset of bacterial pneumonia, it is reasonable to expect that chemical injury to respiratory epithelium will predispose to infection. Ozone induced irritation of the major bronchi in man does occur at ozone concentrations in the range of 0.25 ppm. Hence it is possible that ozone damage at the level of respiratory bronchioles and alveoli occurs in man as well as rodents, leading to promotion of susceptibility to bacterial infection of the lower respiratory tract. The Panel observed that it may be particularly difficult to demonstrate these relationships because bacterial pneumonia is a disease of low incidence, occurring more in the winter season when oxidant concentrations are typically low. It is possible that concentrations of ozone high enough to injure macrophages do not reach the alveoli in man but do so in rodents because of anatomic differences between human and rodent lungs. In this case, the significance of the animal data on ozone-induced susceptibility to infection lies in the demonstration that a measurable effect on a biologically important system (defense against infection) occurs in rodents at concentrations that also produce measurable responses in man (altered mechanical function of the lung).

B. Morphological Abnormalities of the Respiratory System

A range of morphological effects was noted in association with experimental ozone exposures of 0.2 to 1.0 ppm. These effects varied from replacement of Type I with Type II alveolar cells, which are not known to

have associated harmful consequences, to emphysematous changes and terminal bronchiole and alveolar damage. These effects occurring after long-term low concentrations, raise the level of suspicion that repeated or chronic exposures have the potential for inducing similar effects in humans.

C. Biochemical Effects

The Panel observed that there is an impressive variety of biochemical alterations associated with ozone exposures over the range of 0.1 to 1.0 ppm. The Panel judged that effects induced by 0.1 to 0.2 ppm exposures are probably largely adaptive while effects caused by levels of 0.5 ppm and greater have definite toxic potential. The Panel judged that these biochemical changes are significant in demonstrating that there are ozone-induced effects at cellular sites and organ systems distant from the lung. While many of the biological reactions were in the nature of a protective response and could possibly be prevented or reversed with increased vitamin E levels in the lung or increased antioxidants at other tissue sites, nevertheless they represented the organism's response to stress. The Panel agreed that such perturbations in biological systems may pose a health risk to the population of impaired or susceptible individuals. One of the panel members noted that, in experimental studies, Canadian subjects who manifested greater change in mechanical function of the lung than in Los Angeles subjects also showed greater hemolysis of red blood cells. These observations suggest that ozone levels which produce lung function changes can also lead to biochemical changes and other reactions of definite concern to health, and vice versa. Overall, the Panel felt that there is not a sharp dividing line between protective responses and potential for pathological consequences.

D. Genetic and Teratogenic Potential

The Panel focused its attention on the reports of chromosomal abnormalities in peripheral leukocytes first observed upon exposure of intact hamsters to 0.2 ppm ozone for five hours (Zelac, 1971). One experimental study of humans (Merz et al. 1975) found similar abnormalities in individuals exposed to 0.5 ppm ozone. The significance of chromosomal aberrations in peripheral blood cells has not been determined. Furthermore these studies have not,been replicated, and the Panel agreed that there was insufficient evidence to evaluate the long term implications of these reports. Although confirmation of the results may suggest another category of ozone-induced effects, the preliminary evidence does not suggest these effects occur at drastically lower levels of exposure. It was further noted that genetic consequences of population exposures to human mutagens are very difficult to identify, even for such well recognized mutagens as ionizing radiation.

Veninga's observation (1967) of increased neonatal deaths and congenital abnormalities in newborn mice was cited by the Panel as grounds for raising the index of concern over the potential teratogenic effects of ozone. The evidence for the mutagenic effect of ozone in bacterial systems and lung tumorigenesis was also noted. The Panel observed that, were similar findings demonstrated for food additives or pesticides, these substances would undoubtedly be banned from use.

E. Performance and Behavioral Effects

Few experimental studies of performance and behavioral effects of ozone have been reported and the Panel considered this category of effects to be a neglected area of research both in animal and human ozone studies. The effects of ozone and other oxidant exposure on athletic performance (Wayne et al. 1967) and on motor vehicle accidents (Ury 1968) provide epidemiological evidence for impact on performance in humans.

F. Implications of Animal Toxicology Data

The Panel observed that the ozone dose which caused chronic tissue damage in the lungs of experimental animals was much closer to ambient concentrations than are the doses of carcinogens employed to produce tumors in laboratory animals. The chronic respiratory diseases related to similar tissue changes in humans were felt to be at least as important, from a public health perspective, as respiratory cancer. Thus, it was noted that the margin of difference between ozone concentrations that produce serious toxicological effects in animals (as well as symptomatic and lung function changes in humans) and ambient levels of ozone is much smaller than for any other atmospheric pollutant. The Panel also emphasized that there was a remarkable clustering and convergence of various toxicological experimental human, and epidemiologically observed effects within a narrow ozone concentration range, that is 0.2 to 0.6 ppm. These findings further reinforce the judgment that there is not a sharp cut-off for ozone dose-response relationship. The Panel hypothesized that exposures above maximum observed background levels of 0.05-0.06 ppm may well be associated with some increased health risk; this perception is based on the convergence of various effects at concentrations minimally above background (less than one order of magnitude).

The Panel pointed out that chronic disease effects of long term ozone exposure in man cannot be quantitatively related to specific ozone concentrations of short (hourly or daily) averaging times due to the long period of disease induction and the varied exposures of individuals during the period of induction. Epidemiological studies may establish relationships between long-term ozone exposure and human chronic disease risk, but we must rely on toxicological studies to guide us in quantifying the ozone level that may induce chronic effects.

VIII. "Ozone" vs. "Oxidant" Standard

The question was raised by the EPA staff whether the Health Panel felt that an ozone standard or a standard for ozone and other photochemical substances was required for protection of public health. The Panel agreed that two characteristics of ozone exposure should be cited with reference to public health protection: (1) ozone itself is a primary cause of most of the health effects reported in toxicological and experimental human studies and the evidence for attributing many health effects to this substance alone is very compelling, and (2) ozone measurements should be considered as an index for the complex of atmospheric photochemical substances some of which are known to produce health effects which either are not attributable to pure ozone, e.g., eye irritation, or which may possibly augment the effects of pure ozone. The Panel judged that it was not appropriate at this time to consider a primary air quality standard for specific photochemical compounds other than ozone.

The Panel agreed that when ozone is measured specifically, in contrast to measurements of total oxidants, it should be considered both as a causal agent of adverse health effects in its own right and as an index for the photochemical mixture which is associated with a broader range of health consequences. Beyond this, the Panel did not reach consensus as to whether the primary air quality standard should be expressed solely as an ozone standard or as a standard for ozone and other photochemical substances. Some Panel members were concerned that a standard for ozone alone would lead some to dismiss epidemiological evidence relating health effects to "oxidant" concentrations and further to discount the occurrence of symptoms and effects not attributable to pure ozone. Other Panel members believed that citing ozone as an index as well as primary agent in its own right would obviate these problems.

IX. Recommended Guidelines for the Protection of Public Health

In considering the margin of safety between concentrations that represent a public health risk and the recommended standard, the Health Panel called attention to several conslusions that were previously stated:

1. There appears to be a finite probability of health risk associated with population exposure to ozone concentrations above observed maximum background levels, i.e., 0.05 to 0.06 ppm. Specifically, there is evidence from studies of ozone and other photochemical oxidants for alterations of mechanical function of lung in humans, for exacerbation of asthma in humans, for alteration of ventilatory function in school children, for induction of acute respiratory symptoms in exercising children and young adults, for impaired athletic performance in young adults. The Panel judged, as discussed in the previous sections, that these effects may be induced by short term exposures to ozone in the range of 0.15 to 0.25 ppm. An unreplicated study of airway resistance in humans and repeated studies of susceptibility to experimental respiratory infection in animals further suggest the possibility of adverse effects in some persons at short term ozone exposure of 0.10 ppm.

2. In contrast to most other atmospheric pollutants, there is a convergence of experimental animal, experimental human, and epidemiological studies demonstrating effects at relatively low ozone concentrations, 0.15 to 0.35 ppm. These concentrations represent a relatively small margin of difference between observed minimum background levels and concentrations causing increased health risks.

3. The implications of these demonstrated health risks are significant to the overall health of human populations.

4. There is new evidence for biological reactions to combinations of ozone with other commonly occurring atmospheric pollutants.

In reviewing the body of evidence on health effects, the Health Panel concluded that there is no compelling reason to suggest a change from the concentration defined by the existing primary air quality standard, namely, 0.08 ppm. This conclusion was based upon the previously cited Panel consensus that a variety of adverse effects are likely to occur in some segments of the population from short-term ozone exposures of 0.15 to 0.25 ppm, and upon other evidence that suggests, though less conclusively, the possibility of effects at concentrations as low as 0.10

ppm. The Panel recognized that this standard provides very little margin of safety, for the reasons cited immediately above.

In considering the appropriate averaging time to be associated with this recommended concentration limit, the Panel judged that a one-hour exposure limit would provide some slight margin of safety for exercising individuals, whereas two or three hour exposures at the same level would tend to increase the delivered dose and thus raise the level of risk. Thus the Panel concluded that a primary standard of 0.08 ppm for one hour represents a level of exposure which would be consistent with protection of public health.

The issue of how many times the 0.08 ppm one-hour level could be exceeded without increased health risk was addressed. The Panel agreed that the level of health risk increased (1) in proportion to the hourly concentration above 0.08 ppm, (2) in proportion to the number of hours in one day above 0.08 ppm, and (3) in proportion to the frequency of days in which hourly averages exceed 0.08 ppm, though the latter conclusion was recognized to be quite judgmental and generally lacking in confirmatory studies. Nevertheless, the Panel could cite no medical reason to suggest that any exceedances of the standard were without health risk.

In offering its recommendation of a 0.08 ppm one-hour exposure limit as a guideline for the protection of public health, the Panel proposed that this guideline be considered as a public health objective for developing control strategies, rather than a signal for injunctive legal actions. Excursions above the 0.08 ppm hourly limit should call for evaluation of implementation strategies and not for crisis reactions

that are disruptive of normal activities. As previously stated, the level of health risk is judged to be proportional to the concentration, duration and frequency of exposures above the standard. The Panel is not suggesting that small health risks are acceptable, but that occasional excursions should be evaluated in the context of long term goals and the difficulty of reducing the primary emissions necessary to achieve the standards. Overall, however, the Panel judges that there is now more experimental and epidemiological evidence to be concerned with ozone exposures above the standard than when the standard was originally established, and that this concern should reinforce the determination to implement the control strategies required to achieve the primary standard.

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