OZONE

CAS: 10028-15-6

O_3

CEILING LIMIT, 0.1 ppm (≈ 0.2 mg/m³)

Ozone is a liquid or gas, depending on temperature, each appearing bluish in color. The gas possesses a characteristic odor in concentrations of less than 2 ppm.[1] Physicochemical properties include:

Molecular weight: 48.00
Density of gas: 1.6 (air = 1)
Melting point: −193°C
Boiling point: −111.9°C
Critical temperature: −12.1°C

Ozone is a powerful oxidizing agent. The liquid and concentrated solutions explode on warming.

Ozone is used as a disinfectant for air and water, for bleaching textiles, oils and waxes, and in organic syntheses.[1]. It is also produced in welding arcs, corona discharges, and by ultraviolet radiation.

Acutely, ozone is injurious or lethal at relatively low concentrations and at short exposure periods (white rat, LD_{50} in 4 hrs = 4.8 ppm).[2] The primary site of acute injury is the lung, an injury characterized by pulmonary congestion, edema, and hemorrhage. There are indications in man that there are secondary sites of reaction to ozone characterized by a defect in the dissociation of oxygen from oxyhemoglobin.[3] Chronic exposure to ozone has been reported to result in bronchiolitis and bronchitis in animals exposed daily for six hours over a period of one year, at concentrations slightly in excess of 1.0 ppm.[4] Jaffe[5], in a review paper, noted that exposure at 0.1 and 0.2 ppm seven hours a day for three weeks resulted in increased neonatal mortality in mice.

On the basis of a report of Griswold *et al*,[6] the susceptibility of man to ozone appears to be at least equal to that of the most susceptible animal species (mouse and rat). Human exposure for two hours at an average concentration of 1.5 ppm ozone resulted in a 20% reduction in timed vital capacity of the lung and other effects. Kleinfeld and Giel[7] reported pulmonary congestion in welders using the inert-gas shielded-arc process in which the ozone concentration reached a maximum of 9 ppm. Challen and co-workers[8] found similar effects in welders from exposure somewhat under 2 ppm, which disappeared when ozone levels were reduced to around 0.2 ppm.

In addition to these serious effects of ozone, air concentrations of ozone in excess of a few tenths ppm cause occasional discomfort to exposed individuals in the form of headache, and dryness of throat and mucous membranes of the nose and eyes following exposures of short duration.[9,10]

The important points to consider in the setting of a TLV for exposure to ozone are: 1) whether pulmonary effects are a function of dose and whether exercise potentiates the effects of exposure, 2) whether effects of chronic exposure occur at concentrations below those causing acute effects, and 3) whether workers with chronic obstructive pulmonary disease are at greater risk than workers without pulmonary disease.

Dose response characteristics of human exposure to ozone at steady state exercise levels are addressed by the work of McDonnell *et al*.[11] Six groups of young men, ranging in number from 20 to 29, were exposed for 2.5 hours to one of six concentrations of ozone while undergoing intermittent exercise at a level that increased pulmonary minute ventilation to approximately 35 lpm/m² of body surface area. (This corresponds to a moderately heavy work load.) The six ozone concentrations were 0, 0.12, 0.18, 0.24, 0.30, and 0.40 ppm. Significant decreases in forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were seen at 0.12 ppm and were successively greater at the higher ozone concentrations. Coughing was increased at 0.12 ppm as well as at the higher concentrations. At ozone levels of 0.3 ppm, Adams *et al*[12] and Folinsbee *et al*[13] demonstrated that concentration is more effective in reducing pulmonary function values than is increased minute ventilation or duration of exposure.

The effect of exercise was demonstrated at ozone levels of 0.3 ppm in an animal model by Mautz *et al*.[14] Rats were exposed to 0.2 ppm under conditions of rest and treadmill exercise up to 30 lpm at 20% grade. The abundance and severity of focal lung parenchymal lesions increased as exercise and duration of exposure were increased. At concentrations above 0.3 ppm, it was found that the concentration of ozone was the primary determinant of pulmonary effect, rather than the effective dose rate (estimated as concentration X minute-volume). At lower levels, the pulmonary effect was dependent on both exercise rate and concentration.

The question of whether effects from chronic exposure occur at concentrations of ozone below the threshold for acute effects probably cannot be answered directly, but is closely related to the issue of whether a TWA exposure is applicable to ozone. Since chronic effects from exposure to ozone at levels below 1.0 ppm have not been reported, inferences from animal studies and human short-term exposure studies may be useful. Freeman *et al*[15] showed that dogs exposed to 1.0 ppm ozone for 16 hours had less than half as much macrophage response in their airways as dogs exposed for only 8 hours to 2 ppm, even though the time-weighted average exposure was the same in both experiments. Similarly, dogs exposed to 1.0 ppm for 24 hours had about half of the response of dogs exposed for 8 hours to 3 ppm. Dungworth *et al*[16] studied effects of ozone concentrations ranging from 0.2 to 0.8 ppm on rhesus and bonnet monkeys exposed 8 hours per day for 7 days. Histological examination of conducting airways of the bonnet monkeys revealed the luminal surfaces coated with numerous macrophages, other cellular elements, and small quantities of debris. There were both hyperplasia and hypertrophy of bronchiolar epithelium which resulted in replacement of the usual nonciliated cuboidal and squamous cells by low columnar cells rich in organelles. The authors state that the threshold for detectable morphological effect in bonnet monkeys is below 0.2 ppm and may be close to 0.1 ppm. However, changes evoked during the first three to four days bring about a state of adaptation whereby continued exposure results in little or no additional damage.

With regard to populations with obstructive pulmonary disease, Linn *et al*[17] measured pulmonary function and biochemical parameters in 22 asthmatic volunteers exposed for two hours to ozone concentration approximating 0.2 ppm under conditions of heat stress and intermittant exercise. There were no significant changes in FEV_1, FVC, lung volume, or single breath N_2 indices. Statistically significant, but very small changes in a number of biochemical measures, such as glucose-6-phosphate dehydrogenase and acetylcholinesterase, were reported.

Based upon the information from man and animals, it appears that exposures to ozone in the order of 0.2 ppm produce mild acute, but not cumulative effects. Thus, control of exposure should not be

based on the concept of cumulative dose as measured by the eight-hour TWA. It also appears that exposures in the order of 0.1 ppm will be well tolerated by most workers including asthmatics.

Based on all of the above information, a ceiling limit of 0.1 ppm is recommended. The intent of the Committee being that this ceiling value has the effect of establishing a 15-minute time-weighted average of 0.1 ppm.

References

1. *The Merck Index*, 10th ed., p. 1002, Merck & Co., Inc., Rahway, New Jersey (1983).
2. **Stokinger, H.E.:** *Arch. Ind. Health 15*:181 (1957).
3. **Brinkman, R. and H.B. Lamberts:** *Nature 181*:1202 (1958).
4. **Stokinger, H.E., W.D. Wagner and O.J. Dobrogorski:** *Arch. Ind. Health 16*:514 (1957).
5. **Jaffe, L.S.:** *Am. Ind. Hyg. Assoc. J. 28*:267 (1967).
6. **Griswold, S., L.A. Chambers and H.L. Motley:** *Arch. Ind. Health 15*:108 (1957).
7. **Kleinfeld, M. and C.P. Giel:** *Am. J. Med. Sci. 231*:638 (1956).
8. **Challen, P.J.R., D.E. Hickish and J. Bedford:** *Br. J. Ind. Med. 15*:276 (1958).
9. **Wilska, S.:** *Acta. Chem. Scand. 5*:359 (1951).
10. **Truche, M.R.:** *Arch. Mal. Profess. 12*:55 (1951).
11. **McDonnell, W.F., D.H. Horstman, M.J. Hazucha et al:** *J. Appl. Physiol. 54*:1345 (1983).
12. **Adams, W.C., W.M. Savin and A.E. Christo:** *J. Appl. Physiol. Respirat Environ. Exercise Physiol. 51*:415 (1981).
13. **Folinsbee, L.J., B.L. Drinkwater, J.F. Bedi and S.M. Horvach:** *Environmental Stress: Individual Human Adaptations*, pp. 111-124. Academic, New York (1978).
14. **Mautz, W.J., T.R. McClure, P. Reischl, R.F. Phalen and T.T. Crocker:** *J. Toxicol. Environ. Health 16*:841-854 (1985).
15. **Freeman, G., R.J. Stephens, D.L. Coffin and J. Stara:** *Arch. Environ Health 26*:209 (1973).
16. **Dungworth, D.L., W.L. Castleman, C.K. Chow et al:** *Fed. Proc. 34*:1670 (1975).
17. **Linn, W.S., R.D. Buckley, C.E. Spier et al:** *Am. Rev. Respir. Disease 117*:835 (1978).

MAC, 1 ppm, 1946-1947
TLV-TWA, 1 ppm, 1948-1953
TLV-TWA, 0.1 ppm, 1954-1986
TLV-STEL, 0.3 ppm, 1976-1986
TLV-CEILING, 0.1 ppm, proposed 1987
Documentation reviewed/revised, 1987