Ozone

Updated: December 2015

Ozone (O_3) is triatomic form of oxygen.

It is normally present in the atmosphere in minute quantities without any harmful effects but it occurs in increased concentrations in high altitudes [1].

Ozone is a colorless, odorless gas formed by the reaction of volatile organic compounds (VOC) and nitrogen oxides (NOx) with sunlight. Ground-level ozone should not be confused with the stratospheric "ozone" layer 15 to 40 km above the earth's surface, which provides a shield from the sun's ultraviolet rays. Motor vehicles account for more than half of all ozone precursors in industrialized locations and in sites with heavy vehicular traffic.

The conversion of VOC or NOx to ozone occurs over a period of 6 to 12 hours [2]. The meteorological conditions that tend to foster the generation of ozone are typically present from late spring to early fall. Peak concentrations of O_3 typically occur in mid-afternoon, after both the morning rush hours and several hours of bright sunlight [3].

Ozone occurs in stratosphere (up to 10 ppm from UV effect on oxygen) and in troposphere (photochemical smog, electrical storms) [4].

Indoor sources of ozone include office equipment with electric motors or ultraviolet light such electrostatic devices such as air purifiers and ion generators. Certain electrical equipment generates significant levels of ozone. This is especially true of devices using high-voltages such as electric arc welding, photocopying machines, laser printers.

Electric motors that use brushes can generate ozone from repeating sparking inside the unit.

Ozone can be produced from electroplating, mercury vapour lamps, X-ray generators, photoengraving, electrostatic air cleaners.

Usage

Ozone is generated on-site, at its point of use, because of the difficulty, hazards and high cost of transporting ozone and because it is not easily stored.

Ozone can be generated by dissociation of molecular oxygen electrically (silent discharge) or photochemically (ultraviolet irradiation). Gaseous ozone can be adsorbed by porous solid substrates, such as silica gel, and is often used in this form in organic synthesis.

Ozone is used:

- for the purification and disinfection of drinking water;
- for high purity water systems (e.g. bottling and canning plants); disinfection and odor control of industrial and municipal wastewater and sewage, swimming pools and spas, and industrial processes;
- as a bleaching agent; food preservative;
- in cold storage rooms, brewery cellars, hotel and hospital air ducts and air conditioning systems;
- in organic synthesis; medical applications; aquatic oxidant;
- in high purity silver production.

Ozone has becoming increasingly important as alternative to chlorine in pulp paper bleaching.

Tens of millions of persons in the United States are exposed to levels of O_3 above the current NAAQS (National Ambient Air Quality Standards) [5].

Route of exposure

As a reactive oxidizing agent that is slightly soluble in water, ozone is a potent respiratory tract irritant. Exposure occurs most commonly by breathing.

Absorption and metabolism

Ozone is a strong irritant, and its relatively low solubility facilitates delivery to the lower respiratory tract. Dosimetric studies indicate that much of the inhaled O_3 is deposited in the upper and proximal lower airways, however, because of its relative water insolubility, a considerable fraction does penetrate to the distal airways and alveoli. Still, ozone is absorbed throughout the respiratory tract. Approximately 40-50% of inspired ozone is taken up in the nasopharynx, while about 90% of the ozone reaching the lower respiratory tract is removed. A small fraction of inhaled ozone is absorbed into the blood.

Increased respiratory flow, such as with exercise, may overcome the upper airway "scrubbing mechanism" and cause greater deposition of O_3 in the distal lung.

Ozone is a potent oxidant and is capable of reacting with a variety of extracellular and intracellular molecules. Ozone's toxicity has been attributed to oxidation of amino acids and sulfhydryl groups in enzymes and other proteins, and polyunsaturated fatty acids to fatty acid peroxides, resulting in free radical formation. Cellular membranes contain both protein and lipid, and are thought to be the major site of action of ozone toxicity.

The study in Mexico investigated exposure to a group of air pollutants including ozone and severity of DNA damage. The DNA damage magnitude was positively correlated with ozone exposure [6].

There is interesting fact that endogenous ozone is generated in the atherosclerotic vessel as a by-product of the inflammatory response [7].

Target organs: upper and lower expiratory tract

Health effects

Acute exposure

Acute illness following heavy exposure (in excess of 2 ppm) is rapid in onset consisting of severe headache, substernal pain and dyspnea or develops more slowly with irritation of the nose and eyes which may last for days, with severe cough, bloodstained sputum, dyspnea and fever. The symptoms, physical signs and radiographic appearance are those of pulmonary edema. Exposure of from 5 to 20 ppm from 1 hour or more may be fatal [Parkes].

Ozone inhalation by healthy subjects causes mean decrements in forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) that correlate with concentration, exposure duration, and minute ventilation. The mechanism of the decreased inspiratory capacity appears to be neurally-mediated involuntary inhibition of respiratory effort involving stimulation of C-fibers in the lungs. The acute decrements in pulmonary function induced by ozone usually resolve within 24 hours.

Another adverse effect of short-term exposure to ozone is enhanced airway responsiveness to nonspecific stimuli such as methacholine and histamine.

Ozone produces local irritation of the eyes and mucous membranes.

Nasal inflammatory changes, type I alveolar and ciliated airway epithelial cell injury, infiltration of the airway mucosa by neutrophils, and increased bronchoalveolar lavage (BAL) fluid neutrophils and inflammatory mediators have also been observed after exposure.

Exposure to ozone gases was studied in pulp mills. The study shows that acute exposure to high levels of ozone may increase the risk of noninfectious rhinitis [8].

The effects of chronic exposure in humans have not been adequately defined.

A growing body of evidence indicates that maternal exposure to air pollutants, including ozone, is associated with adverse pregnancy outcomes. A major research

focus has been to investigate the effects of air pollution on birth weight, low birth weight (LBW; <2,500 g), and intrauterine growth retardation (IUGR). O3 levels in the second and the third trimesters were independently associated with lower birth weight and IUGR in term infants through the mechanism of reduced fetal growth [9].

The study in London tests the effects of air pollution on the heart. Ozone is implicated as a cause of adverse cardiovascular effect [10].

Because O_3 inhalation can induce both airway inflammation and enhanced airway responsiveness, it is reasonable to expect persons with asthma to have greater susceptibility to ozone. Ozone exposure in the paper industry in Sweden has been shown to be a risk factor for asthma.

Evidence is accumulating that exposure to ambient levels of O_3 is associated with increased risk of mortality [La Dou].

References

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