

Appendix C

Health Risk Evaluation for Carbon Dioxide (CO₂)

General Information

Carbon dioxide (CO₂) is a colorless, odorless, non-flammable gas that is a product of cellular respiration and burning of fossil fuels. It has a molecular weight of 44.01g/mol (NIOSH 1976). Although it is typically present as a gas, carbon dioxide also can be a solid form as dry ice and liquefied, depending on temperature and pressure (Nelson 2000). This gas is utilized by many types of industry including breweries, mining ore, and manufacturing of carbonated drinks, drugs, disinfectants, pottery, and baking powder (NIOSH 1976). It also is a primary gas associated with volcanic eruptions (Farrar et al. 1999; IVHHN 2005). CO₂ acts to displace oxygen, making compressed CO₂ the main ingredient in fire extinguishers (MDPH 2005). Occupations that are most at risk from CO₂ exposure include miners, brewers, carbonated beverage workers, and grain elevator workers (CCOHS 2005; Nelson 2000).

CO₂ is present in the atmosphere at 0.035% (Aerias 2005; CCOHS 2005). In terms of worker safety, Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit (PEL) for CO₂ of 5,000 parts per million (ppm) over an 8-hour work day, which is equivalent to 0.5% by volume of air. Similarly, the American Conference of Governmental Industrial Hygienists (ACGIH) TLV (threshold limit value) is 5,000 ppm for an 8-hour workday, with a ceiling exposure limit of 30,000 ppm for a 10-minute period based on acute inhalation data (MDPH 2005; NIOSH 1976). A value of 40,000 ppm is considered immediately dangerous to life and health based on the fact that a 30-minute exposure to 50,000 ppm produces intoxication, and concentrations greater than that (7-10%) produce unconsciousness (NIOSH 1996; Tox. Review 2005). Additionally, acute toxicity data show the lethal concentration low (LC_{Lo}) for CO₂ is 90,000 ppm (9%) over 5 minutes (NIOSH 1996). See Table 1 for a listing of regulatory agency standards for acceptable CO₂ concentrations in the workplace. CO₂ is a good indicator of proper building ventilation and indoor air exchange rates. Consequently, it is measured in buildings to determine if the indoor air is adequate for humans to occupy the building (MDPH 2005).

Table 1 Agency Standards for CO₂ in the Workplace.

Agency	Low end CO₂ Concentration (ppm)¹	High-end CO₂ Concentration (ppm)²
OSHA PEL	5,000 TWA	30,000 STEL
ACGIH TLV	5,000 TWA	30,000 STEL
NIOSH REL	5,000 TWA	30,000 STEL

¹Applies to CO₂ concentration in the workplace considered safe for a 40-hour week.

² Based on a 10-minute period for NIOSH and a 15-minute period for OSHA and ACGIH.

PEL = Permissible Exposure Limit

TLV = Threshold Limit Value

REL= Recommended Exposure Limit

TWA= Time Weighted Average

STEL= Short Term Exposure Limit

Although normal levels of CO₂ are considered harmless, under the right conditions, CO₂ can cause adverse health effects. High concentrations of CO₂ in confined areas can be potentially

dangerous. CO₂ may act as an oxygen displacer in confined spaces and cause a number of reactions. These reactions include, but are not limited to, dizziness, disorientation, suffocation, and under certain circumstances, death. Death occurs when there is a depression of the central nervous system (CNS) with prolonged exposure to high levels of CO₂ and the body's compensatory mechanisms are overwhelmed or fail (Farrar et al. 1999; IVHHN 2005; Nelson 2000; NIOSH 1976; NIOSH 1996).

Toxicology of CO₂

CO₂ is considered to be a potential inhalation toxicant and a simple asphyxiate (Aerias 2005; NIOSH 1976; Priestly 2003). It enters the body from the atmosphere through the lungs, is distributed to the blood, and may cause an acid-base imbalance, or acidosis, with subsequent CNS depression (Nelson 2000; Priestly 2003). Acidosis is caused by an overabundance of CO₂ in the blood. Under normal physiological circumstances, there is a higher concentration of CO₂ in the blood than in the lungs, forming a concentration gradient, where blood CO₂ diffuses into the lungs and then is exhaled. An increase in inhaled CO₂ and subsequent reaction with water in the blood forms carbonic acid (H₂CO₃), which then dissociates into hydrogen ions [H⁺] and bicarbonate [HCO₃⁻]. The excess CO₂ shifts the equilibrium toward the creation of more hydrogen ions, thus creating an acidic environment (see equation below). During respiratory acidosis, the pH of the blood becomes less than 7.35 (Priestly 2003).



Electrolyte imbalance occurs due to decreased blood plasma chloride, potassium, and calcium and increased blood plasma sodium. Furthermore, the oxygen depleted environment does not allow for cells in the body to obtain the oxygen they need to survive. Fortunately, the body compensates for the excess in H⁺ ions by binding of the protons to hemoglobin. In addition, the lungs attempt to compensate by removing the excess CO₂, which is the reason rapid breathing is apparent during acute CO₂ exposure. After prolonged exposure, the kidney begins to balance blood pH by retaining bicarbonate and excreting hydrogen ions to correct acidosis (Priestly 2003).

Symptoms related to acute CO₂ exposure are shown in Table 2 (Aerias 2005; IVHHN 2005). Treatment to high exposures of this compound involves removing the victim from the confined space or oxygen inadequate environment, and increasing the oxygen supply to the exposed individual (MSDS for CO₂ 2003; Nelson 2000; Priestly 2003). The condition of acidosis is reversible upon removal from a high CO₂ environment.

Table 2 Symptoms from Low to High Concentrations of CO₂

%CO ₂	Symptoms
2 to 3	Shortness of breath, deep breathing
5	Breathing becomes heavy, sweating, pulse quickens
7.5	Headaches, dizziness, restlessness, breathlessness, increased heart rate and blood pressure, visual distortion
10	Impaired hearing, nausea, vomiting, loss of consciousness
30	Coma, convulsions, death

Animal and Epidemiological Studies

Numerous animal studies have been performed to study CO₂ toxicity. Monkeys with implanted electrodes in areas of the brain were exposed to concentrations from 0-30% CO₂, and electrical activity was recorded. This study showed that as CO₂ concentrations increased, the time to obtain electrical stimulation also increased, indicating CNS depression (NIOSH 1976). Rat and guinea pig exposure to higher concentrations of CO₂ indirectly caused a decrease in lung stability. Studies have indicated further that respiratory acidosis was followed by fluid build up and a decrease in lung surface tension in the male guinea pig. Most animal studies concluded adverse effects on the lungs. However, cardiac, kidney, and reproductive effects also have been shown in animals as a result of exposure to high levels of CO₂ (NIOSH 1976).

Epidemiological studies have been performed to observe human toxicity to CO₂, as well. Normal blood concentrations of CO₂ act physiologically to stimulate the CNS, while extremely high concentrations exert CNS depression (CCOHS 2005; NIOSH 1976). Within 1 minute of exposure to 20-30% CO₂, unconsciousness and convulsions occur in humans. Neurologic symptoms including eye and extremity twitching, and convulsions have been observed in humans after CO₂ exposure (CCOHS 2005; NIOSH 1976).

Occupational Exposure

For centuries, miners have been aware of the occupational hazard of “black damp,” a condition of low oxygen levels in mine shafts (Cable 2004; NIOSH 1976). It was common for miners to send a candle or mouse into the mine prior to entering and watch for the candle to extinguish or the mouse to lose consciousness, indicating a lack of oxygen, hence, a poor working environment (NIOSH 1976). Brewers also are confronted with the potential of CO₂ poisoning. Yeast releases CO₂ as a byproduct in the process of fermenting alcohol (Nelson 2000; Tox. Review 2005).

Brewers entering enclosed areas, such as cleaning out tanks subsequent to fermentation, could be overcome by high levels of CO₂. A study on brewery workers determined that they are exposed to 1.08% over an 8-hour workday on average (Nelson 2000; NIOSH 1976; Tox. Review 2005). CO₂ is also a byproduct of metabolic activity of organic grains. Therefore, employees working in grain elevators and silos, where stored grain produces 37% CO₂ during oxidation of carbohydrates, are at risk for high levels of CO₂ exposure (Nelson 2000; NIOSH 1976).

From long-term exposure to 3% CO₂, submarine workers have shown symptoms such as flushing of the skin, a fall in blood pressure, and decreased oxygen consumption (CCOHS 2005). However, long-term exposure to low concentrations of CO₂ has not resulted in asphyxiation; adaptive physiological mechanisms to long-term exposure have been reported (CCOHS 2005).

Other Exposures

At CO₂ levels greater than 0.5%, adverse health affects are present in humans, animals, and plants. Plants utilize CO₂ as a primary ingredient in photosynthesis and depend on the gas for survival. However, under concentrated conditions, plant roots can actually be suffocated, which inhibits the uptake of nutrients, and subsequently kills the plants (Farrar et al. 1999; NIOSH 1976). This phenomenon was noted in Mammoth, California, recognized for infrequent, yet recent volcanic activity. Researchers investigating this phenomenon discovered concentrations as high as 95% CO₂ by volume from magmatic emissions (Farrar et al. 1999). These elevated

concentrations were measured in pits in the snow and soil, buildings with poor ventilation, and in belowground valve boxes in the vicinity of Mammoth Mountain. Accumulation in pits and wells occurs due to the fact that CO₂ is denser than air and may slowly accumulate (IVHHN 2005). Specifically, soil gas levels of CO₂ in a snow well in Mammoth were measured at 70% after the death of a skier in the vicinity of the well (Farrar et al. 1999; IVHHN 2005).

Several erupting volcanoes have claimed the lives of people for centuries due to CO₂ exposure. The gas is more dense than that of ambient air (1.8 g/L at 25°C and 1 atm), and therefore, the excess CO₂ flows down the side of the mountain and is trapped near the ground surface (IVHHN 2005). Dieng Plateau, Indonesia released a CO₂ cloud with concentrations of 98-99%, killing approximately 142 villagers (IVHHN 2005). Mount Vesuvius, a volcano in Italy, has claimed the lives of many people due to measured concentrations of up to 100% CO₂.

Several other accounts of excess CO₂ exposure have been recorded. A couple in West Virginia experienced symptoms of CO₂ exposure, including mild confusion, headaches, and blurred vision, from CO₂ levels of 9.5% in their basement crawl space. The West Virginia department of Environmental Protection revealed that their home was receiving high concentrations of CO₂ because it was built above a reclaimed surface and an abandoned deep coal mine (Cable 2004; PGS no date)). Another episode of CO₂ poisoning occurred in a poorly ventilated walk-in refrigerator, where a 50-year-old man was found dead among 15 blocks of dry ice. The off-gassing of the dry ice and non-functional ventilation system was blamed for his death (Nelson 2000).

Conclusions

CO₂ is a naturally occurring atmospheric gas that is considered safe at levels below 0.5% according to OSHA standards (CCOHS 2005). However, occupational hazards related to CO₂ exposure may occur under certain conditions. The American Society of Heating, Refrigerating, and Air Conditioning Engineer, Inc., recommends that indoor air CO₂ levels be less than 700 ppm above the outdoor air concentration of CO₂ (Aerias 2005).

In addition to potential indoor exposure, high concentrations of CO₂ can collect outdoors. Outdoor exposure can occur where CO₂ is venting from below ground sources such as mining operations, natural gas production, and magmatic emissions. Aboveground sources of exposure can occur during volcanic eruptions. External air factors are mostly related to the fact that CO₂ is denser than ambient air and therefore, tends to accumulate near the ground surface (IVHHN 2005).

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