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Short Term Respiratory Effects of Sulphur Dioxide Exposure

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Commentary

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DESCRITPION

Sulfur dioxide is a common air pollutant emitted from both anthropogenic and natural sources, and has long been known to cause adverse respiratory health effects. SO_2 has been established as the chemical indicator for the National Ambient Air Quality Standards for gaseous sulfur oxides under the US Clean Air Act. Current SO_2 standards have been promulgated for 24-hr and annual average concentrations; however, there is a strong body of evidence demonstrating adverse respiratory effects at much shorter exposure durations. Studies of controlled human exposures have consistently observed increases in respiratory symptoms and decreases in lung function among exercising asthmatics following 5 to 10 min exposures to SO_2.

These findings are supported by epidemiologic studies that have demonstrated associations between ambient SO_2 levels and both respiratory symptoms and emergency department visits and hospitalizations for respiratory causes. The immediate effect of acute exposure to SO_2 in animals and humans is bronchoconstriction. In animals, SO_2 exposure activates irritant gas-sensitive receptors such as the rapidly activating receptors (RARs) and sensory C-fiber receptors in the tracheobronchial tree. These receptors can stimulate the central nervous system reflexes that lead to bronchoconstriction, mucus secretion, mucosal vasodilation, cough, apnea followed by shallow breathing, and cardiovascular system effects such as bradycardia and hyper- or hypotension. Both cholinergic and noncholinergic mechanisms have been demonstrated to play a role in these events. The cholinergic parasympathetic pathways involving the vagus nerve are involved in initial ventilatory responses, but not in the prolonged bronchoconstrictor response. Neurogenic inflammation, smooth muscle cell contraction, and histamine

may be involved; however, differences in respiratory tract innervation between rodents and humans suggest that C-fiber-mediated neurogenic inflammation may be unimportant in humans.

SO_2-induced bronchoconstriction can be attenuated by anticholinergic treatment in nonasthmatic humans, suggesting the cholinergic pathways as a main contributor to this pathology. However, in asthmatics, bronchoconstriction is only partially blocked by anticholinergic agents, beta-2-adrenergic agonists, or leukotriene receptor antagonists. This implies involvement of both parasympathetic pathways and inflammatory mediators. Inflammation may enhance the sensitivity to SO_2 in asthmatics by altering autonomic responses, enhancing mediator release, or sensitizing C-fibers and RARs.

Exposure to sulfur dioxide may cause irritation to the eyes, nose, and throat. Symptoms include: nasal mucus, choking, cough, and reflex bronchi constriction, and when liquid: frostbite Workers may be harmed from exposure to sulfur dioxide. The level of exposure depends upon the dose, duration, and work being done.

Sulfur dioxide is used in many industries. It's used to manufacture sulfuric acid, paper, and food preservatives. Some examples of workers at risk of being exposed to sulfur dioxide include the Factory workers in industries where it occurs as a by-product, such as copper smelting or power plants, Industry workers that manufacture sulfuric acid, Workers in plants that produce paper, Food processing to preserve foods, such as dry fruits, Workers who manufacture fertilizers, etc. Synonyms include sulfur oxide, sulfurous acid anhydride, sulfurous anhydride, and sulfurous oxide. Persons exposed only to sulfur dioxide gas pose no risk of secondary contamination. Persons whose skin or clothing is contaminated with liquid sulfur dioxide can secondarily contaminate rescuers by direct contact or through off-gassing of vapor. At room temperature, sulfur dioxide is a nonflammable, colorless gas that is heavier than air. Its strong, pungent odor and irritating properties usually provide adequate warning of its presence.

Sulfur dioxide is readily absorbed through the upper respiratory tract; no data were located regarding dermal absorption. Sulfur dioxide is present in some foods; therefore, oral ingestion, although insignificant, is possible.