

HEALTH EFFECTS

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Summary

Automobile and truck exhausts contain substances that can adversely affect human health if encountered in sufficiently high concentrations. Included are carbon monoxide, oxides of nitrogen, oxides of sulfur, uncombusted and partially combusted hydrocarbons (including aliphatics, olefins, benzene and other simple aromatic compounds, aldehydes, ketones, and polynuclear aromatic hydrocarbons), and particulate matter of varying composition. In addition, exhaust product mixtures may react in the presence of sunlight to form other potentially toxic materials like ozone and peroxyacetyl nitrate (PAN). This article discusses the healthy effects of the substances listed above respectively.

1. Introduction

A substance must reach a target organ at levels of a certain concentration, called the threshold level, to be detrimental to health. The target organ may be non-specific (as when corrosives are involved) or may be specific (as when hydrocarbons affect the liver). Inhalation is the most common route of entry for highway-related pollutants, both gases and particulates.

The concentration of toxic material that reaches the target organs depends on factors such as concentration in the ambient environment, and the frequency and length of exposure. Usually, concentrations of highway-related pollutants remain well below hazardous levels. However, photochemical smog may occur in some high traffic areas. When this occurs, toxic substances may reach levels that can cause adverse health effects for some

individuals.

Understanding how highway pollutants affect human health requires knowledge about potential health effects of the individual components and synergistic effects that might occur from exposure to multiple toxins. Knowledge of how individual components may react to produce new toxic substances is also important. In most cases, little is known of the toxicological properties of pollutants at the low levels likely to be encountered. It is therefore necessary to extrapolate results from studies on animals or from exposure to humans at higher concentrations (occupational exposures).

Epidemiological studies may be inconclusive because the effects are often quite subtle, concentrations vary significantly, and the combination of multiple pollutants affects the interactions that arise from photochemical smog.

2. Carbon Monoxide

The effects of carbon monoxide exposure at high levels are well known. When carbon monoxide is inhaled, it enters the blood stream and binds to hemoglobin. The resultant compound is carboxyhemoglobin. Because the binding affinity of carbon monoxide to hemoglobin is so much stronger than that of oxygen (about 210 times stronger), it blocks the ability of hemoglobin to transport oxygen. The blood is then unable to supply sufficient oxygen to the cells of the body. The ultimate effect is similar to that seen in severe anemia.

Percent COH _b	Sign and symptoms for an average human
0-1	No signs or symptoms
10-20	Tightness across the forehead, possible slight headache, dilation of the cutaneous blood vessels
20-30	Headache and throbbing in the temples
30-40	Severe headache, weakness, dizziness, impaired vision, nausea, vomiting and collapse
40-50	Same as above, greater possibility of collapse, syncope and increased pulse and respiratory rates
50-60	Syncope, increased pulse rate, coma, intermittent convulsions, and Cheyne-Stokes respiration
60-70	Coma, intermittent convulsions, depressed heart action and respiratory rate, and possible death
70-80	Weak pulse, slow respirations, respiratory failure and death within a few hours
80-90	Death in less than an hour
90+	Death within a few minutes

Table 1: Signs and symptoms at various concentrations of carboxyhemoglobin

Carboxyhemoglobin formation effectively lowers the available hemoglobin. In normal individuals, no effect is noted until at least 5-10% of the hemoglobin is converted to carboxyhemoglobin. The reserve capacity of the blood prevents effects at lower

carboxyhemoglobin levels. As carboxyhemoglobin levels increase, impairment of judgment, nausea and vomiting, coma and convulsions occur. Death may occur if the carboxyhemoglobin levels reach about 70%. The toxic effects of carbon monoxide as measured by the percentage of carboxyhemoglobin in the blood are given in Table 1.

There have been a number of research studies about the effects of carbon monoxide. In some experiments, effects were seen in angina patients at carboxyhemoglobin levels of 2 to 4%, the same could be seen in certain groups of non-smokers exposed to high levels of carbon monoxide. In people who tend to have irregular heart rhythm, this was enhanced after carbon monoxide exposure (6% COHb). There are some important findings that suggest people with existing ischemic heart disease who have symptoms of *angina pectoris* may suffer these symptoms after mild exertion following exposure to carbon monoxide. The serious problems mentioned above are most likely to be experienced by road users or others, as a result of exposure to vehicle exhaust. Exposures to carbon monoxide from automobile exhaust are usually low except when vehicles are run in confined areas (garages, tunnels, enclosed parking lots and parking garages). In most cases, Carboxyhemoglobin levels remain a low level (below 5%).

The effects of chronic exposure to low levels of carbon monoxide are not well understood and are subject to some controversy. It has been hypothesized that low-level exposures to carbon monoxide contribute to cardiovascular disease. Conflicting results have been obtained from animal studies. Weir and Fabiano reviewed the evidence and concluded that chronic exposure to low levels of carbon monoxide neither contributed to the atheromatosis nor adversely affected cardiac rhythm in humans.

L H Watkins holds another viewpoint. Although it seems a clear indication that carbon monoxide is not likely to produce any permanent effects or cause acute physical discomfort to most people, its effects cannot be entirely discounted. Apart from relatively minor physical discomfort in particularly susceptible people, quite small amounts of COHb in the blood have been shown to temporarily impair mental functions (Beard, 1967) and Schulte. (Schulte, 1963) claims that perception is reduced at levels of less than 5 percent, which can affect driving ability. Also, impaired oxygen transport in the human body has serious implications for persons with pre-existing heart or lung pathologies (WHO, 1979), and for fetuses. Chronic exposure to low levels of carbon monoxide can affect brain structure. Cardiac effects of carbon monoxide include effects on the electro-physiological properties of the heart at quite low levels of carboxyhemoglobin (5.5%), and may reduce the threshold at which cardiac arrhythmias or arrest can occur. High levels of carbon monoxide during pregnancy can reduce fetal growth and survival in animals, as carbon monoxide has been shown to preferentially bind to fetal hemoglobin.

3. Nitrogen Oxides

Nitric oxide has no direct effect on health because of its low toxicity and relatively rapid removal from the atmosphere. However, it is converted to nitrogen dioxide, playing a role in photochemical smog generation. Nitrogen dioxide is much more toxic than nitric oxide. Nitrogen dioxide is an oxidizing agent and can damage lung tissue via its oxidizing properties. It acts on the pulmonary alveoli and impairs their structure (in extreme cases causing emphysema), and has a cytotoxic effect on the alveolar macrophages. It affects

macrophage activity in low doses, weakening the natural defense mechanisms of the body, thus aggravating other complications (infection, etc.). Nitrogen dioxide can cause the death of specific cells in the lungs and impair the regulation of the pulmonary function. At very high doses it acts as a potent initiator of inflammation within the lung, preferentially affecting the small airways, close to the site of gas exchange in the lungs.

Some studies that have been conducted indicate that exposure to nitrogen dioxide may be linked to an increased susceptibility to respiratory infection, increased airway resistance in asthmatics, and decreased pulmonary function (Walsh, 1988). Short-term exposures to nitrogen dioxide have resulted in a wide-ranging group of respiratory problems in school children, such as coughs and sore throats. Some well-designed studies have shown an association between nitrogen dioxide and respiratory symptoms in healthy individuals and people with asthma; the latter studies from Arizona also demonstrating a significant independent effect on lung function in asthmatic individuals. One study from the United Kingdom found an association between nitrogen dioxide levels and impaired lung function in patients with asthma:

There has been a marked increase in the incidence of respiratory problems in recent years. Between 1976 and 1987, acute attacks of asthma more than doubled in England and Wales (from 10.7 to 27.1 per 100,000 patients a week). The greatest increase was in children (from 13.5 to 74.4 per 100,000 in the 0-4 age group and from 17.4 to 58.9 in the 5-14 age group). It seems unlikely that these were changes in reporting or diagnosis. There is some evidence that hay fever symptoms are exacerbated when concentrations of pollutants are high. Vehicle emissions may enhance sensitivity to pollen allergens. Some recent epidemiological studies have indicated that individuals suffering from respiratory disorders, including asthma, may experience a worsening of their symptoms when there are elevated ambient levels of nitrogen dioxide and associated pollutants, especially particulates. The factors which lead to an increase in the number of attacks of asthma in already susceptible individuals are not, however, necessarily the same as factors which cause individuals to have respiratory problems in the first place.

The above-mentioned studies have major flaws. They occurred at concentrations typically higher than normal air quality standards. The possibility of effects from continuous exposure to very low levels of pollutants remains a worry, and there is also the possibility of adverse effects resulting from the absorption of nitrogen oxides on the carbon particles produced from vehicle exhaust. Therefore, no effect as yet can be definitively ascribed to nitrogen dioxide at ambient levels.

4. Sulfur Dioxide

Sulfur dioxide is a moderate irritant, strongly hydrophilic and hence easily dissolved in the nasal mucous membranes, with little risk of affecting the lung. In the tissues, it is transformed into bisulfite and after enzymatic oxidation, and is eliminated by the kidneys in sulfate form. However, it impairs the body's defense mechanisms and thus contributes to effects caused by other pollutants (such as NO₂, PAH, and metals). Exposure to high levels of sulfur dioxide over a long period of time produces structural changes in the lung, with thickening of the lung lining, increase in glandular tissue, thickening of the protective mucus layer and reduction in mucus transport (which clears both mucus and

other debris from the lung).

Because levels of sulfur dioxide and particulates are closely related, it has been difficult to demonstrate effects of sulfur dioxide that are independent from those of particulates. In most recent studies, independent effects on mortality have been found for particulates but not sulfur dioxide. Ambient levels of sulfur dioxide were closely associated with hospital admissions for respiratory conditions and asthma during summers in Canada. A careful study of the relationship between particulates and sulfur dioxide exposures on emergency room visits for chronic obstructive pulmonary disease (COPD) showed significant associations below current European guideline levels in Barcelona, Spain. An association was also found between sulfur dioxide levels and hospital admissions for asthma in Birmingham, UK (during summer) having no correlation to particulate levels. Although it is clear that ambient levels of sulfur dioxide may still be associated with health effects below current air quality standards, not all studies confirm this effect.

5. Volatile Organic Compounds

Volatile organic compounds (VOCs) are complex mixtures consisting of aliphatics, olefins, aldehydes, ketones and aromatics. Many of these compounds are recognized as being potentially hazardous to human health. From a toxicological standpoint, benzene is probably the most important of the VOCs. Prolonged elevated exposures to benzene, either through the respiratory tract or cutaneous contact, can result in aplastic anemia or acute myelogenous leukaemia. Bone marrow is the target organ for benzene. As the marrow becomes increasingly affected, there is a decrease in circulating erythrocytes, platelets and leukocytes. In the most severe stages of benzene poisoning, all three of these cell types are depressed. This condition is called pancytopenia. When there is no longer any functional marrow, the condition is called aplastic anemia. Benzene interferes with both DNA and RNA synthesis in bone marrow cells.

Vehicle emissions are the most important source of exposure to benzene for people in urban areas who do not smoke. Benzene, which has a direct effect on genetic material, causes pancytopenia and aplastic anemia. In addition, chromosomal aberrations and leukemia have been noted as a result of benzene exposure. A major fraction of VOCs consists of aliphatic hydrocarbons. Inhalation of these vapors may be harmful because, at high concentrations, they depress the central nervous system causing dizziness and impaired coordination. Lower molecular weight compounds are very important in VOCs because of their higher vapor pressure and hence higher ambient concentrations. These compounds have very low acute toxicity levels. Prolonged exposure may result in peripheral neuropathies characterized by sensory loss and motor weakness in the hands, arms, legs and feet.

However, little is known of the toxicological consequences of exposure to these hydrocarbons at the very low levels expected as a result of vehicular traffic. Most individual hydrocarbons would be below measurable levels in the ambient atmosphere except in special circumstances. It is generally accepted that exposure to hydrocarbons at these low levels has little or no effect on human health; but because of the major role they play in photochemical smog, they are important in overall health effects.

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Biographical Sketches

Kebin is a Professor of Environmental Science & Engineering, Director of the Office of International Cooperation and Exchange, Tsinghua University. Since receiving his Ph. D. in environmental engineering in 1990, he has been conducting research on air pollution, including coal-fired air pollution and vehicular emission, for over ten years. Up to now, as a principal investigator, he has finished more than 20 research projects and published more than 90 academic papers. Dr. He has been a senior visiting scholar at the Technical University of Denmark, Leeds University in the UK, and Harvard University in the US. Dr. He also serves as a Member of the Council for the China Energy Research Society, as the Senior Member of China Society of Environmental Science, a Member of the Pollution Control Working Group in China Council for International Cooperation on Environment and Development, a Member of the Air & Waste Management Association in the USA, and a Member of the Society of Automotive Engineering in the USA.

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