Connections between air pollution, traffic and respiratory health

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ABSTRACT

Air pollution increases the likelihood of developing a variety of health issues, particularly in the field of respiration. Involuntary exposure to traffic-related air pollution affects humans from fetal development to death. Nitrogen dioxide, sulfur dioxide, carbon monoxide, benzene, ozone, particulate matter 10 (PM10), lead, arsenic, cadmium, nickel, benzopyrene, and particulate matter 2.5 (PM2.5) are among the lung-damaging substances found in air pollutants. Determining the causal relationship between various major road pollutants and the human respiratory system is a difficult task. In general, it can be concluded that traffic-related pollutants will induce an inflammatory response that will progress based on the variation of humans exposed. The concept of oxidative stress resulting from traffic-related pollutants has also begun to receive extensive research. This gives hope for assisting the human antioxidant system in combating the increasingly difficult-to-avoid threat of air pollution.

KEYWORDS

Air pollution, respiratory system, lung health, traffic

INTRODUCTION

Air pollution increases the likelihood of suffering from a variety of health problems, particularly in the respiratory system (World Health Organization, 2010). Recent studies reveal a sharp and very significant increase in air pollution levels throughout the world, particularly in countries with large populations. This increases the associated morbidity with air pollution. In 2010, air pollution caused more than two hundred thousand lung cancer deaths worldwide (World Health Organization, 2017). In 2012, the World Health Organization (WHO) reported that air pollution caused 3.7 million premature deaths (which should not have occurred) (Lelieveld et al., 2015; World Health Organization, 2017).

Similar to cigarette smoke, traffic-related air pollution causes respiratory health issues. In contrast to smoking, exposure to air pollution is unavoidable and causes health problems in humans from conception to death. Urban communities' primary source of air pollution is motor vehicles. The global increase in the number of motorized vehicles is becoming progressively unstoppable. According to the journal of the global automotive...
industry, there were 1.2 billion motor vehicles worldwide in 2014. Exhaust gases from motor vehicles contain numerous hazardous substances. Until recently, it was known that motor vehicles emit carbon dioxide gas, carbon monoxide, hydrocarbons, nitrogen oxides, particulate matter (PM), and other hazardous substances that are classified as mobile source toxic air pollutants (MSAT) (Environmental Protection Agency, 2008; World Health Organization, 2005).

Normal humans inhale between 10,000 and 15,000 liters of air per day. It is through this inhalation process that humans are exposed to airborne pollutants. Humans are unable to select the air that will enter their respiratory system. This makes the respiratory system the primary target for various airborne toxins. In addition to the main pollutants that have been determined to be harmful to respiratory health (particulate matter, ozone, and nitrogen dioxide), a number of new air pollutants, including polycyclic aromatic hydrocarbons (PAH) and volatile organic compounds (VOC), have been identified. Recent research has shown that a collection of multiple pollutants has a far greater negative impact on health than the sum of their individual effects (Lelieveld et al., 2015; World Health Organization, 2005).

Our awareness and vigilance regarding air pollution, particularly as it relates to traffic, are still severely lacking. Development and an expanding economy have resulted in densely populated cities with a growing number of automobiles. On the other hand, traffic-related air pollution is increasing, which poses a threat to respiratory health in particular. Following is an explanation of traffic-related air pollution and its effects on respiratory health, focusing on inflammation and oxidative stress experienced by the lungs.

**Air pollutants**

In 2008, the European Union Parliament passed an air pollution regulation known as the Environmental Protection Act (EPA). The European Union countries agreed in the regulation to conduct stringent monitoring of their air quality. The Environmental Protection Agency (EPA) identifies 12 airborne substances as health hazards. These include nitrogen dioxide, sulfur dioxide, carbon monoxide, benzene, ozone, particulate matter 10 (PM10), lead, arsenic, cadmium, nickel, benzopyrene, and particulate matter 2.5 (PM2.5) (Environmental Protection Agency, 2008).

Based on the process of their formation, air pollutants can be categorized as either primary air pollutants or secondary air pollutants. Primary air pollutants are various substances released directly into the atmosphere by their source. Primary air pollutants include smoke from factory exhaust pipes and certain components of automobile exhaust gases. These primary air pollutants’ emissions can be calculated directly from the source’s exhaust pipe end. Unlike secondary air pollutants, hazardous materials are produced in the atmosphere. These hazardous substances result from the chemical reactions of primary air pollutants. This chemical reaction also involves oxygen and water, which are natural components of the atmosphere. The most prevalent example of a secondary air pollutant is ozone, which is produced through a unique chemical reaction that is influenced by a variety of atmospheric natural factors. It is difficult to measure the emission levels of secondary air pollutants. Although it can be approximated using its constituent parts. Traffic-related pollutants can take the form of both primary and secondary air pollutants in relation to motor vehicle emissions (RCP, 2016; World Health Organization, 2005).
Additionally, the physical properties of air pollutants must be determined. On the basis of their physical properties, air pollutants can be classified as either gaseous or particulate. Gaseous air pollutants are minute molecules that exist in the form of gases or vapors and can pass through filters that do not react with them. Typically, this gaseous contaminant does not enter the lower respiratory tract or lung tissue. While air pollutants in the form of particles are solid or liquid substances that have become trapped in the atmosphere. These particles may be primary or secondary air contaminants of varying sizes (Hendriks et al., 2013).

**Mechanism of formation of various air pollutants**

**Sulphur dioxide**

The combustion of sulfur-containing fuel is the primary source of sulfur dioxide in the air. From the source, fossil fuels contain 1 to 5 percent sulfur (see Table 1). As a result of the combustion process, the sulfur in the fuel becomes sulfur dioxide. In various parts of the world, the refining of fuel has begun to reduce the sulfur content of crude oil. This relates to the fuel trade process and the extremely stringent emission standards established by western nations as fuel consumers (Héroux et al., 2015).

**Nitrogen oxide**

Similar to the formation of sulfur dioxide during the combustion of fuel, this process will convert the nitrogen component into nitrogen oxides (see Table 1). This process utilizes a substantial amount of coal as the source material, as oil contains significantly less nitrogen than coal. The majority of airborne nitrogen oxides are nitric oxide (NO\textsubscript{2}). More than ninety percent of this nitrogen oxide formation process occurs through atmospheric chemical reactions (Bernstein & Rice, 2013; Fuzzi et al., 2015).

**Carbon monoxide**

Carbon monoxide is a byproduct of the incomplete combustion of carbon-containing fuels (see Table 1). However, complete combustion will produce carbon dioxide as the exhaust gas. The vast majority of oxidation of motor vehicle fuel in the world produces only carbon monoxide. This is the reason why automobiles are the primary source of carbon monoxide pollution (Bernstein & Rice, 2013).

**Volatile organic compounds (VOC)**

Volatile organic compounds (VOCs) consist of various hydrocarbons, oxygenates, halogenates, and other carbonaceous materials that exist in the atmosphere as vapor. The primary sources of these volatile organic compounds are leaks from pressurized gas systems (such as methane and natural gas) and evaporation of liquid fuels from automobile fuel tanks (see Table 1). Due to the presence of unburned or partially-burned fuel components, the combustion of fossil fuels also produces these volatile organic compounds (Sierra-Vargas & Teran, 2012; World Health Organization, 2005).
Table 1. Types of pollutants in the air and the mechanism of their formation

| Types of Pollutants          | Forming mechanism                                                                 |
|------------------------------|-----------------------------------------------------------------------------------|
| Sulfur dioxide               | Residual combustion of fuel containing sulfur                                      |
| Nitrogen oxide               | The rest of the fuel combustion comes mainly from coal                             |
| Carbon monoxide              | Produced due to incomplete combustion of carbon-containing fuel                    |
| Volatile organic compounds   | Consists of various types of hydrocarbons, oxygenates, halogenates, and other carbonaceous materials sourced from leaks of pressurized gas systems (e.g. methane and natural gas) and evaporation of liquid fuel from motorized vehicle tanks. |
| Carbon particles             | Produced from the burning of fossil fuels and biomass consisting of carbon in elemental and organic forms which have low volatility |
| Non-carbon primary particles | Ash consisting of mineral material released during the combustion of fuel          |
| Nitrogen oxide/ozone system  | Nitric oxide (NO), which is 95% of the nitrogen oxides produced by combustion and reacts with ozone |

**Carbon particles**

The particles produced by the combustion of fossil fuels and biomass are composed of carbon in elemental and organic forms that are relatively stable (see Table 1). Carbon atoms combined with fire produce carbon free radicals and extremely toxic polycyclic aromatic hydrocarbons (PAHs) (Huang, 2014; Marino et al., 2015).

**Non-carbon primary particles**

Airborne ash is the primary source of this pollutant. This ash is composed of mineral material that is released during the combustion of fuel and transported into the atmosphere, where it is then trapped (see Table 1). Ash and road dust are also sources of these non-carbonated particles, although not to the same extent as minerals from combustion residues (HEI Traffic Panel, 2010; Hendriks et al., 2013).

**Nitrogen oxide/ozone system**

The majority of nitrogen oxide waste is nitric oxide (NO), which accounts for 95 percent of the nitrogen oxides produced by combustion (see Table 1). Under these circumstances, NO is harmless to human health. But when NO chemically reacts with ozone in the atmosphere to form nitrogen dioxide, it is too late to anticipate the real problem. The chemical process that leads to the formation of nitrogen dioxide from NO is known as the ozone system. Sunlight availability is a requirement for this reaction (Costa et al., 2014; World Health Organization, 2005).

As a significant component of the ozone system, ozone is a secondary air pollutant that originates in the atmosphere. The local sources of ozone are the northern hemisphere of the Earth and the sun-mediated breakdown of oxygen, which typically surrounds the stratosphere. In both locations, the ozone layer shields us
from the harmful effects of ultraviolet rays. Additionally, the troposphere can generate ozone through the chemical reactions of reactive hydrocarbons. The result of this reaction is a peroxy radical. In polluted (NO-rich) air, peroxy radicals react with NO to produce nitrogen dioxide, which is extremely hazardous to human health and the environment (Perez et al., 2010).

**Air pollution related to traffic and respiratory health**

Traffic-related air pollution is a very complex topic to discuss. This type of air pollution is caused by exhaust gas emissions from motorized vehicles, road surface deterioration, and brake, clutch, and wheel friction. Traffic-related air pollution does not exhibit the same characteristics in different countries, according to the findings of a variety of studies. It is extremely difficult to quantify the impact of each source of pollution on health disorders. Some toxicological studies have demonstrated the effects of individual pollutants on the human body, but this is not the case for the combined global processes underlying traffic-related air pollution. It is still difficult to establish a causal relationship between exposure and particular health problems. Figure 1 depicts an outline of the effects of air pollution on breathing (World Health Organization, 2005).

Recent studies on traffic-related air pollution have focused on ozone, sulfur dioxide, nitrogen oxides, and particulate matter (both PM2.5 and PM 10). These pollutants' effects are observed both individually and within an air unit. In the inflammatory process that occurs along the respiratory tract, the impact of these pollutants on respiratory health occurs earliest. Experimentally measured ozone exposure has been shown to increase levels of inflammatory mediators (IL-8) in animals (Vawda et al., 2014). In ozone-exposed human respiratory tracts, the release of tumor necrosis factor (TNF-) and interleukins (interleukins 8 and 6) was also significantly increased (Minelli et al., 2011).

Particulate matter (PM), which contributes significantly to air pollution, is detrimental to respiratory health. Multiple studies have demonstrated an increase in inflammatory mediators and immune cells following PM exposure in the laboratory as well as from diesel and gasoline-powered vehicle exhaust gases (Yang et al., 2008). In animal studies involving exposure to traffic-related air pollution, a reduction in lung function has also been demonstrated as a result of inflammation-related reductions in lung elasticity and structure. Inflammation of the entire body has also been shown to result from prolonged exposure to PM. In experimental animals, this systemic inflammation causes cardiovascular and autonomic nervous disorders (Vawda et al., 2014).

Exposure to nitrogen dioxide is associated with damage to the airway and lung epithelial cells. The destruction and death of epithelial cells in the respiratory systems of experimental animals are not yet conclusively linked to a particular inflammatory process or to exposure to other air pollutants. Several studies compiled by the American HEI study group did not establish a causal link between nitrogen dioxide exposure and its consequences (HEI Traffic Panel, 2010; Sierra-Vargas & Teran, 2012). Exposure to sulfur dioxide in the human airways can result in bronchoconstriction and lung tissue inflammation. The reactions that activate cytokines and chemokines in lung tissue are not fully understood (Janeway & Medzhitov, 2002; Yang et al., 2008).

In its most recent publication, the HEI study group describes the effects of exposure to traffic-related
pollutants on the cardiopulmonary system. These exposures result in an increase in respiratory complaints, a decrease in peak expiratory flow, and an increase in the inflammatory response of adult airways. Exposure to traffic-related pollutants for at least 20 minutes can affect the respiratory tract of healthy volunteers, manifesting as an increase in the neutrophil count, exhaled nitric oxide, a slight decrease in peak expiratory flow, and airway resistance (HEI Traffic Panel, 2010). The results of research by Costa et al. (2014) could not find a causal relationship when a sample of healthy volunteers was exposed to pollutants individually in the laboratory (Costa et al., 2014). This inconsistency makes it difficult to draw conclusions about the effects of exposure to traffic-related pollutants on the human respiratory system (Costa et al., 2014; Perez et al., 2010).

Recent studies have investigated the role of oxidative stress in the relationship between traffic-related air pollution and human respiratory health. This is based on numerous studies on particulate matter (PM), the primary pollutant associated with vehicular traffic. The majority of studies have discovered that PM has the potential to generate oxidants that are harmful to the human respiratory system.

Under normal conditions, the human lungs inhale 10 liters of air per minute, which is evenly distributed over 100-140 m² of lung surface area. The area is susceptible to multiple types of air pollution and infections. The human respiratory system is already equipped with a very sophisticated and comprehensive defense system to combat these threats. In the upper respiratory tract, harmful substances-laden air will be humidified and filtered mechanically. In the human respiratory system, epithelial cells serve as a barrier to protect our respiratory health. Epithelial cells initiate cell movement and the immune system’s defense against microorganisms, allergens, and chemicals such as those found in traffic-related air pollution (Barker-Collo et al., 2013; Vawda et al., 2014).

Through various chemical reactions, certain traffic-related pollutants continuously generate free radicals in the atmosphere. These free radicals constantly expose humans to danger. As a result of chemical reactions associated with respiration and cell phagocytosis, humans are also exposed to endogenous oxides. In healthy individuals, genes play a crucial role in maintaining a balance between antioxidant and xenobiotic responses to air pollution (Minelli et al., 2011). This balance prevents harmful effects of free radicals on the human respiratory system (Kelly, 2003).

**CONCLUSION**

Globally, traffic-related air pollution is a significant risk factor for respiratory disease. Involuntary exposure to traffic-related air pollution affects humans from fetal development until death. Determining the causal relationship between various major road pollutants and the human respiratory system is a difficult task. In general, it can be concluded that traffic-related pollutants will induce an inflammatory response that will progress based on the variation of the exposed human. The concept of oxidative stress resulting from traffic-related pollutants has also begun to receive extensive research. This provides hope for assisting the human anti-oxidant system in combating the increasingly difficult-to-avoid threat of air pollution.
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