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## Health impacts of traffic related air pollution

Amit U. Raysoni

*The University of Texas Rio Grande Valley*

Wen-Wahi Li

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Review Paper (NS-1)**HEALTH IMPACTS OF TRAFFIC RELATED  
AIR POLLUTION****Amit U. Raysoni<sup>\*1</sup> and Wen-Whai Li<sup>1,2</sup>**

1. Department of Environmental Science and Engineering Ph.D. Program, The University of Texas at El Paso, El Paso, Texas 79968, (USA)
2. Department of Civil Engineering, The University of Texas at El Paso, El Paso, Texas 79968, (USA)

*Received July 20, 2009**Accepted December 04, 2009***ABSTRACT**

Ambient air pollution can be a serious cause of concern for any community. Anthropogenic ambient air pollutants can emanate from industries, traffic, geological sources and domestic heating and cooking. However, studies have shown that traffic related air pollution can have far more detrimental health effects than non-combustion sources. These adverse health effects are most profound in sensitive populations like the elderly and young children. The World Health Organization (WHO) attributes more than 300 million deaths every year to ambient air pollution. The WHO's Air Quality Guidelines (AQG) and the United States Environment Protection Agency's (USEPA) National Ambient Air Quality Standards (NAAQS) provide specific ambient air quality standards for several air pollutants. This review paper describes some of the criteria air pollutants (as designated by USEPA) like particulate matter, nitrogen dioxide, and carbon monoxide. Short and long term exposures to these pollutants can lead to cardiovascular, respiratory morbidity and premature death in some cases. In the elderly population, exposure to particulate matter has shown an increase in cases of atherosclerosis, irregular heartbeats, emphysema and onset of myocardial infarction. Young children are particularly vulnerable to damaging effects of traffic air pollutants. Children's lungs are in the process of development and their airway exposure per unit time is more than adults. Also, their defense mechanisms are evolving, thereby, raising their susceptibility to air pollution. In young asthmatic children, exposure to high levels of particulate matter has led to the exacerbation of their asthma. High levels of nitrogen dioxide have shown a decrement in the lung function of young children. This leads to increased rates of school absenteeism, greater use of asthma medications, emergency room visits and hospital admissions. Elucidating the various health effects of traffic pollutants on these sensitive populations is another focal point of this paper.

**Key Words :** Traffic, Air Pollution, Health, Particulate Matter, Nitrogen Dioxide, Carbon Monoxide, Asthma, Children

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\* Author for correspondence

## INTRODUCTION

A plethora of air quality and epidemiological studies have shown an acute association between ambient air pollutants like particulate matter (PM), carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>) etc. and adverse health effects<sup>1</sup>. These adverse health effects are most profound in sensitive populations like the elderly with cardiovascular problems and asthmatic children.

Anthropogenic ambient air pollutants can emanate from traffic, heavy industries, geological sources and domestic heating and cooking. However, of all the above mentioned sources, traffic related pollution is known to have far more detrimental health effects than non-combustion sources<sup>2</sup>. Road traffic is one of the major sources for air pollutants such as NO<sub>2</sub>, CO, PM and volatile organic compounds (VOCs).<sup>3</sup> Many researchers e.g. Briggs et.al, (1997); Wrobel et.al, (2000)<sup>4, 5</sup> have attributed more than 50% of PM emissions to traffic. The United Kingdom Department of Transportation (2002)<sup>6</sup>, in one of their investigatory reports, found that 80% of all PM pollution in London was from road traffic. As such, traffic related air pollution can be a major cause of concern for any community.

### Objectives

The objective of this research paper is to succinctly describe the various traffic related air pollutants and elucidate their detrimental health impacts on sensitive populations. This research paper also describes a few international case studies which have

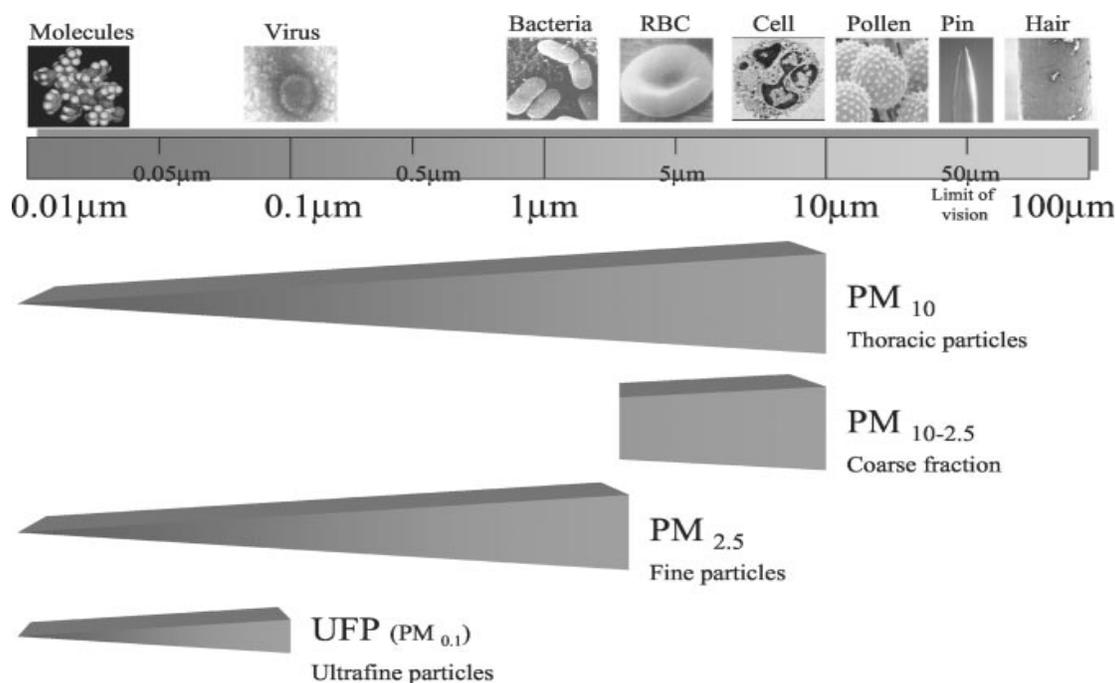
attributed high levels of traffic pollutants to the health of the community in question.

### Particulate Matter

Particulate matter or PM is a complex and heterogeneous mixture of solid and liquid particles suspended in air. Particulate pollutants are made up of a number of components such as acids (nitrates and sulfates), organic chemicals, and soil and dust particles.

Particulate matter comes in various sizes and varies chemically in space and time<sup>7</sup>. The size distribution of PM in ambient air is trimodal, including coarse particles, fine particles and ultrafine particles. In air quality and epidemiological studies, size-selective sampling of PM is conducted to collect particles below, above or within a specified aerodynamic range. Particle size is normally defined to a 50% cut point at a specific aerodynamic diameter. This size selective sampling relates to inhalation and deposition of these particles in the lungs, sources and levels of toxicity<sup>8</sup>.

Coarse particles (PM<sub>10-2.5</sub>) are larger than 2.5 μm but smaller than 10 μm in diameter. They are also generally defined as the difference between PM<sub>10</sub> and PM<sub>2.5</sub> mass concentrations<sup>9</sup>. They generally arise from natural sources like wind-blown soil, pollen, non-exhaust vehicle emissions or from construction and quarrying activities. Fine particles are particles 2.5 μm or smaller in diameter. The primary sources of fine particulate pollution are forest fires, burning of biomass, the reaction of gases in air when emitted from power plants, industries and automobiles<sup>10</sup>.



**Fig 1 :** Particulate matter air pollution size distribution (Source: Brook et.al, 2004).

**Fig. 1** shown above characterizes PM based on size.  $PM < 10 \mu m$  ( $PM_{10}$ ) can enter the human extra thoracic and upper tracheobronchial region, whereas  $PM_{2.5}$  is the size fraction that can reach the small airways and alveoli.

#### Traffic Air Pollution and Lung Function in Children

Children are especially susceptible to the harmful effects of traffic air pollution. High levels of PM and  $NO_2$  have shown to impact their lung function. Lung growth in children is a complex process guided by precisely timed sequence of chemical messages. Many of these pollutants have the potential to interfere with the signaling pathways<sup>11</sup>. There are 24 million alveoli at birth. This increases to 267 million at 4 years and 600 million by adulthood. The air epithelium of growing children is more susceptible to these pollutants<sup>11</sup>.

#### Health Implications of $PM_{2.5}$

Fine particles are considered more toxic because they include nitrates, sulfates, acids, metals adsorbed onto their surfaces.  $PM_{2.5}$  can also be breathed more deeply into the lungs (as mentioned above), penetrate more readily into indoor environments and are transported over great distances<sup>12</sup>.

Pope and Dockery (2006) have summarized the detrimental impacts of particulate pollution on human health, especially for a young vulnerable population like children<sup>13</sup>. Children's exposure to particulate pollution is very different from adults. Children's lungs are in the process of development and the higher metabolic rate of children results in a higher breathing rate<sup>14</sup>. Children are also more active than adults which results in a higher intake of air into their lungs as compared to adults<sup>15</sup>. A higher

intake of air means that more ambient air pollutants enter the lungs. During exercise, the deposition of fine particles in the lungs of children increases five-fold than during rest time.

This increases the exposure of the airway per unit time<sup>16</sup>. This results in more particulate pollutants entering and depositing in their lungs. Activities like PE can increase their breathing rates, thereby increasing their particulate pollution exposure as compared to adults. Children's defense mechanisms are still evolving thereby raising their susceptibility to particulate air pollution<sup>17</sup>. Also, infants and young children have a higher resting metabolic rate of oxygen consumption per unit body weight than adults. This is because they have a larger surface per unit body weight. Children have a smaller lung surface area/kg as compared to adults. Thus ambient air pollutants can impact the lung tissues in children much more than adults<sup>18</sup>. Also, on the basis of body weight, the volume of air passing through the lungs of a resting infant child is twice that of a resting adult. Hence, the infant child's lungs are exposed to twice the amount of ambient air pollutants than adults. Air pollutants may cause massive irritation in the narrower airways of young children.

Inhalation of fine particulate air pollution creates and exacerbates both pulmonary and systemic inflammation and oxidative stress, leading to direct vascular injury, atherosclerosis and autonomic dysfunction<sup>19</sup>.

Buildup of atherosclerotic plaque, measured by the carotid intima-media thickness, is higher in communities with higher mean PM<sub>2.5</sub> concentrations<sup>20</sup>. A study from Germany published in 2004 in the *New England Journal of Medicine* found an

association between exposure to traffic and the onset of myocardial infarction within one hour afterward<sup>21</sup>. An increase in the risk of myocardial infarction and the time spent in cars, on public transportation, on motorcycles was observed. The researchers studied 691 subjects of nonfatal myocardial infarction. These subjects were the hospitalized survivors of non-fatal myocardial infarction between 25-74 years of age. The time of onset of myocardial infarction was defined as the time of the onset of chest pain (angina pectoris) that lasted at least 20 minutes and was not relieved by the administration of nitrates, or the generation of Q waves on the electrocardiogram or elevated levels of certain enzymes.

The results showed that exposure to traffic were more frequent on the day of the onset of the myocardial infarction than during the previous three days (469 person hours with exposure to traffic). Also, one hour before the onset of the myocardial infarction, exposure to traffic was twice as frequent as any other time. The research did not pinpoint on any one specific pollutant that would have aggravated the situation that led to myocardial infarction.

## NO<sub>2</sub>

NO<sub>2</sub> is a gaseous air pollutant. It is one of the nitrogen oxides (NO<sub>x</sub>), a group of air pollutants produced from combustion sources. The presence of NO<sub>2</sub> is mainly due to traffic<sup>22</sup>. The primary emission of nitrogen oxides from vehicles is largely nitrogen monoxide (NO). Formation of NO<sub>2</sub> from NO occurs after emissions and depends on the level of ozone. NO<sub>2</sub> is normally used as an indicator of motor vehicle exhaust because it is easy to measure and environmental air

quality standards for  $\text{NO}_x$  are based on  $\text{NO}_2$ <sup>23</sup>.

### Health effects of $\text{NO}_2$

Exposure to  $\text{NO}_2$  may decrease lung function and increase the risk of respiratory problems, particularly in asthmatic children. Short-term exposure to peak levels can increase respiratory allergic reactions<sup>15</sup>. Populations living close to busy roads are particularly susceptible to  $\text{NO}_2$  pollution<sup>3</sup>.

### Carbon monoxide

CO is a colorless, odorless gas that is formed due to the incomplete combustion of carbon. It is a component of motor vehicle exhaust. Areas with high traffic congestion normally see higher levels of CO. As per the USEPA, in most urban areas, 85 to 95 percent of all CO emissions emanate from motor vehicles<sup>24</sup>.

### Health Impacts of CO

Short term and long term exposure to carbon monoxide can have cardiovascular and central nervous system effects in humans. Lower levels of CO are known to have caused heart disease like angina and congestive heart failure. Carbon monoxide can reduce oxygen delivery to the various organs of the body like heart, brain and other tissues. High levels of CO can lead to vision and cognitive problems, reduced manual dexterity and a decrement in performing complex tasks. At very high levels, CO can cause death. CO also contributes to the formation of smog which, in turn, can trigger serious respiratory problems<sup>24</sup>.

### Case Studies on Health Effects due to Traffic Related Air Pollution

Epidemiological studies have shown that children's respiratory health gets adversely affected when they live near major roads<sup>25, 26, 27</sup>. Gauderman et. al (2005) found that the closer the child lived to a freeway, the higher the child's asthma prevalence. Children who lived 400 meters from the freeway had an 89 percent higher risk of asthma than children living 1,600 meters away from the freeway<sup>28</sup>.

In another study, Van Roorbroeck et.al (2007) measured personal exposure in children to  $\text{PM}_{2.5}$ , soot and  $\text{NO}_x$ <sup>29</sup>. This study was done to validate exposure classifications based on school locations. 54 children attending four different schools in the city of Utrecht, Netherlands were recruited in this study. Two of the schools were located within 100 m of a major road (one ring road and one freeway). The other two schools were at a background location. Investigations revealed that personal exposure to soot was 30% higher at the freeway school compared to its matched background school. The outdoor concentration was 52% higher at the freeway school compared to the background school.

Janssen et. al (2001) measured  $\text{PM}_{2.5}$  mass and filter reflectance and nitrogen dioxide inside and outside twenty four schools in the Netherlands. These schools were within 400 m of major roadways. The researchers found  $\text{PM}_{2.5}$  and soot increased significantly with truck traffic and roadway proximity. Indoor  $\text{NO}_2$  was positively correlated with car traffic<sup>2</sup>.

**RESULTS AND DISCUSSION**

The following table lists the ambient air quality standards for several traffic air

pollutants like PM, NO<sub>2</sub> and CO. These standards are set by the WHO and the USEPA<sup>30,31</sup>.

**Table 1**

Traffic Air Pollutant	WHO Air Quality Guidelines	EPA NAAQS Guidelines
<b>PM<sub>2.5</sub></b>	10 µg/m <sup>3</sup> - annual mean	15.0 µg/m <sup>3</sup> - annual mean
	25 µg/m <sup>3</sup> - 24 hour mean	35 µg/m <sup>3</sup> - 24 hour mean
<b>PM<sub>10</sub></b>	20 µg/m <sup>3</sup> - annual mean	N/A
	50 µg/m <sup>3</sup> - 24 hour mean	150 µg/m <sup>3</sup> - 24 hour mean
<b>NO<sub>2</sub></b>	40 µg/m <sup>3</sup> - annual mean	100 µg/m <sup>3</sup> - annual mean (0.053 PPM) – annual mean
	200 µg/m <sup>3</sup> - 1 hour mean	N/A
<b>CO</b>	N/A	9 ppm (10 mg/m <sup>3</sup> ) – 8 hour
	N/A	35 ppm (40 mg/m <sup>3</sup> ) – 1 hour

As is evident from the table, the ambient air quality standards by WHO are much stricter than the USEPA for all the traffic air pollutants.

As per the WHO, there exists a signification inequality in traffic air pollution exposure and related health risk<sup>32</sup>. High levels of traffic air pollution can lead to a disproportionate disease burden in communities from developing countries, especially when they cumulate with social, physical and economical factors. The mean urban PM<sub>10</sub> in India is 84 µg/m<sup>3</sup>. As per the WHO, the Environment Burden of Disease for outdoor air pollution in India is 120, 600 deaths per year<sup>33</sup>. This is approximately three times higher in magnitude than USA where 41, 200 deaths per year are attributed to outdoor air pollution<sup>34</sup>. The mean urban PM<sub>10</sub> in USA is 24 µg/m<sup>3</sup>.

Developing countries face problems of poverty and social deprivations. The health

effects emanating from high levels of traffic related air pollution are, many a times, neglected by policy makers.

**CONCLUSION**

The human exposure to air pollutants can be limited by collective action at the local, state, national and international levels. Implementation of long-term policies to reduce the risks of traffic related air pollution to health would be the step in the right direction.

A report titled ‘Trends in Asthma Morbidity and Mortality’ published by the American Lung Association in 2006 shows that the economic cost of asthma in the United States for 2004 was \$ 16,100 million. This involved the medication costs, hospital costs, school days lost and loss of work. These figures get more complicated if even a small fraction of these costs are attributed to increase in traffic air pollution.

Results obtained from the above case studies and hundreds of other epidemiological and air pollution case studies should help the policy makers arrive at a logical decision in regards to the imposition of stricter standards for ambient air pollution.

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