



Air Pollution Impacts on Infants and Children

GRADE C

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INTRODUCTION

As noted in previous Report Cards (2001, 2003, and 2004), stringent regulations on industrial and vehicle emissions have allowed the South Coast Air Basin (SoCAB) to make tremendous improvements in air quality over the past 30 years. Despite these improvements, this region still does not meet federal standards for ozone and particulate matter and Los Angeles continues to hold the dubious honor of being the most polluted city in the nation [1].



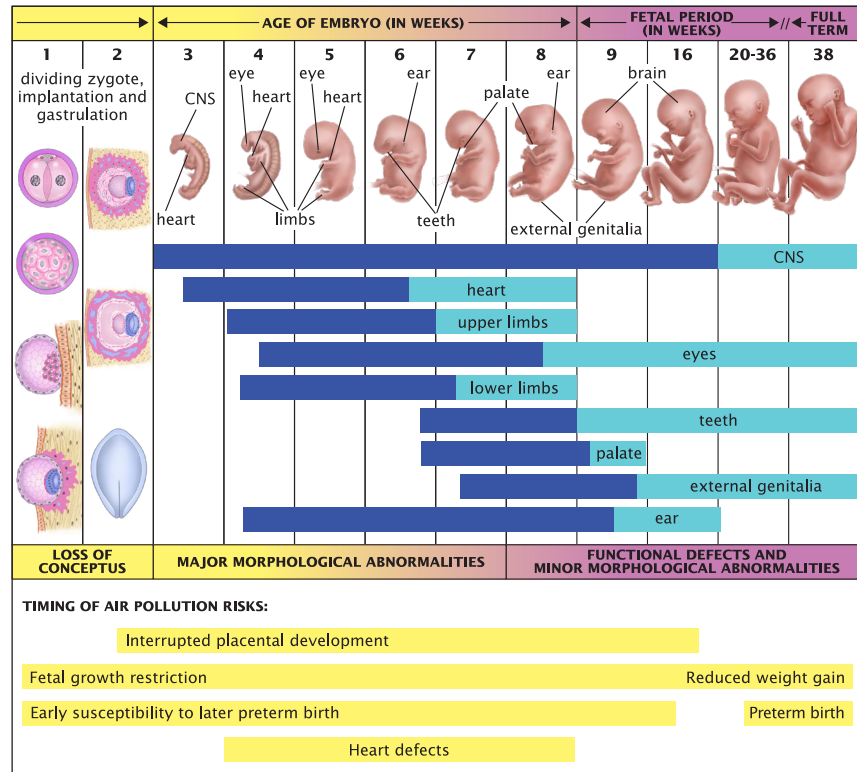
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This article focuses on health effects that ambient and traffic related air pollution has on pregnant women, their infants, and young children. Los Angeles County has a relatively young population due to births and migration. The County's population includes 2.7 million children under 18 years of age, and 27% of adults are in their peak reproductive years (ages 18-34). Approximately 150,000 births occur every year in Los Angeles County, accounting for over one-quarter of all births in California. Thus, any effects air pollution has on human development *in utero*, as well as on infant and children's health, is of great concern to those who live in this region.

The time between conception and birth is perhaps one of the most vulnerable life stages, during which the environment may have tremendous immediate and lasting effects on health. The fetus undergoes rapid growth and organ development and the maternal environment helps direct

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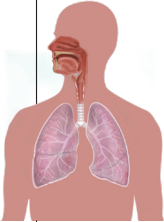
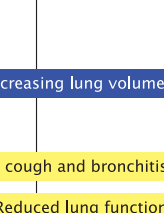
these processes, for better or for worse (Figure 1). Evidence is accumulating that environmental exposures can cause infants to be born premature (before 37 weeks of gestation) or low weight (less than 2500 grams, or 5.5 pounds), or to be born with certain birth defects. These babies are far more likely to die in infancy, and those who survive have high risks of brain, respiratory, and digestive problems in early life. The impact of environmental exposures on fetal development may be far-reaching, as data suggest growth and developmental delays *in utero* influence the risk for heart disease and diabetes in adulthood.



Note: Blue bars indicate time periods when major morphological abnormalities can occur, while light blue bars correspond to periods at risk for minor abnormalities and functional defects.

Figure 1. Fetal development and timing of air pollution risks.

Early childhood is also a critical period for the continued development and maturation of several biological systems such as the brain, lung, and immune system and air toxics can impair lung function and neurodevelopment, or exacerbate existing conditions, such as asthma (Figure 2). Infants who were born premature or growth-retarded may be particularly vulnerable to additional environmental insults, for example, due to immaturity of the lungs at birth.

Stage: Age:	Newborn 0-2 mos	Infant/Toddler 2 mos-2 yrs	Young Child 2-6 yrs	School-Age Child 6-12 yrs	Adolescent 12-18 yrs
					
Lung development:	Alveolar development				
	High respiratory rate		Increasing lung volume		
Air pollution risks:	Respiratory death		Chronic cough and bronchitis		
			Reduced lung function		
			Wheezing and asthma attacks		
	Respiratory symptoms and illnesses*		Respiratory-related school absences		

*Air pollution exposure has also been more recently linked to respiratory symptoms and illnesses in early life including cough, bronchitis, wheeze and ear infections

Figure 2. Air pollution effects on the developing respiratory system.

Exactly what compounds in the ambient air most affect reproductive and children's health, and how these exposures result in restricted fetal growth, early parturition, and development of respiratory diseases remains largely unknown. The study of air pollution's impact on reproductive outcomes is still a developing area of science with many important questions unanswered, but more evidence is emerging that air pollution exposures in pregnancy and early childhood put children at higher risk of adverse health outcomes. Despite the long history of research linking smoking to poorer birth outcomes and the known similarities in components of cigarette smoke and air pollution, the bulk of all air pollution research targeting reproductive health has been conducted only in the past decade. Recently this research has begun to focus on one specific source of modern-day air pollution -- traffic exhaust.

In this article, we discuss the epidemiologic research that illuminates whether and which types of air pollution cause fetal growth restriction, premature labor, infant death, and childhood respiratory diseases.

AIR POLLUTION AND INFANT MORTALITY

Mothers exposed to high levels of CO and particles during pregnancy are at higher risk of adverse birth outcomes, including preterm delivery, low birth weight, and congenital heart defects.



Only in the 1990s, did studies begin to systematically investigate links between air pollution and infant mortality. These studies largely focused on potential mortality impacts of airborne particulate matter small enough to penetrate into the human respiratory tract, referred to as PM₁₀ (particulate matter less than 10 microns in aerodynamic diameter) and more recently have examined PM_{2.5}, even smaller size particles which can penetrate deep into the lung. Most findings from this research indicated infants living in areas with high levels of these types of particulate matter had a greater risk of mortality during the first year of life, particularly from respiratory causes. In our own study of the SoCAB between 1989 and 2000, we found higher risks of infant mortality for very young infants (1-3 months of age) breathing high levels of carbon monoxide (CO), and that infants 4-12 months of age exposed to high levels of particulate matter (PM₁₀) were at higher risk of death from respiratory illnesses. Furthermore, infants exposed to high concentrations of the gaseous pollutant nitrogen dioxide were at increased risk of dying from Sudden Infant Death Syndrome (SIDS). However, studies of air pollution's effect on SIDS to date are equivocal[2], while results for particulate matter and respiratory infant mortality are fairly consistent and further supported by the strong and well established link between particles and adult mortality. These studies nevertheless have not been able to identify the specific components of particulate matter, nor elucidate the mechanism by which these pollutants affect health in children and infants, which may be different from adults.

AIR POLLUTION AND BIRTH OUTCOMES

In Los Angeles, we found that air pollution caused by traffic disproportionately affected women in low-income and disadvantaged neighborhoods.



A seminal paper published in 1977 by researchers in Los Angeles was the first to describe a possible association between air pollution at atmospheric levels and reduced birth weight. But only our more recent research conducted in the SoCAB prompted scientists worldwide to conduct similar studies starting in the late 1990s and early 2000s. Most used

data from birth certificates to assess specific birth outcomes, and air pollution measurements from government monitoring stations to determine a woman's air pollution exposures during specific periods of pregnancy. These studies have linked a number of air pollutants to adverse birth outcomes, including low birth weight (LBW) and small for gestational age (SGA), prematurity, and heart defects at birth. More recently, researchers have also begun to investigate effects on pre-eclampsia (pregnancy-induced hypertension) and spontaneous abortion, the latter possibly triggered by air pollution's damage to DNA in sperm.

In our studies of births during 1990-2003 to women residing in the SoCAB, we consistently found that mothers exposed to high levels of CO and particles during pregnancy are at higher risk of adverse birth outcomes, including preterm delivery, low birth weight, and congenital heart defects. Studies conducted in other urban locations around the world reported similar adverse effects of air pollution (especially for carbon monoxide, sulfur dioxide, and particulate matter) on fetal development, but preterm birth and low birth weight (LBW) have recently also been linked to ozone and nitrogen dioxide exposures. In the SoCAB, the most important source of the air pollutants we identified as relevant is vehicle exhaust, which is a complex mix of many gases and particles. Thus, it is possible other components of exhaust are agents that harm fetal development, while the pollutants that are routinely measured at government monitoring stations serve as indicators of such compounds and the level of traffic exhaust pollution in these areas. In fact, in one study, we linked residential traffic density to preterm and LBW which provides further evidence that traffic exhaust may be particularly important.

Although the majority of these studies were conducted in large urban centers, pregnant women living in less polluted regions seemed to be affected by air toxins as well. Researchers studying

Vancouver and New England, where air pollution levels are far lower than in Los Angeles, also observed lower birth weights and more preterm births to mothers exposed to more air pollution.



The timing of exposure and the specific components of air pollution that possibly impact fetal development and birth outcomes are critical to our understanding of these processes. In our SoCAB studies, we associated preterm birth and low birth weight mostly with high levels of carbon monoxide and particulate matter during the first trimester and the final months before birth. Air pollution exposure during early pregnancy may interfere with placental development and subsequent oxygen and nutrient delivery to the fetus throughout pregnancy, while

the last trimester is important for fetal weight gain. Exposure to air pollution during specific pregnancy periods may also trigger inflammation and lead to preterm birth. Increased risks of certain heart defects were found to be correlated to high levels of carbon monoxide and possibly ozone in the second month of pregnancy, presumably because this is the most important time period for fetal heart development.

To date, scientists have not identified how a mother's exposure to air pollution translates into poorer health of the fetus. Some studies have found that polycyclic aromatic hydrocarbons (PAHs), chemicals formed by the combustion of fossil fuels such as gasoline and diesel, may cause some of the damage. Vehicle exhaust also contains very small or ultra fine particles (UFP), which have a high surface area. PAHs stick to these small particles which are easily inhaled into the human lungs. These UFP can penetrate through the lung barriers into the bloodstream and via the blood, they can enter other organs including the brain and placenta. Studies in mice have shown that particles of the same size as UFP in exhaust can travel to the placenta, chorion, and amniotic fluid, thereby exposing the fetus to these potentially harmful chemicals. These particles can cause inflammation, which may trigger premature labor. PAHs may also interfere with placental development and fetal growth early in pregnancy, but more studies are needed to understand the mechanisms that can cause this to happen.

From a public health perspective, we are also interested in determining which groups of pregnant women or young children are most affected by these environmental toxins. Those with already compromised health may be more susceptible to air pollution's effects. Additionally, people with socioeconomic disadvantages may also be exposed to more toxins at work or in their neighborhoods, have poorer diets and health behaviors, and lack adequate access to health care. In Los Angeles, we found that air pollution caused by traffic disproportionately affected women in low-income and disadvantaged neighborhoods during the winter, when meteorological conditions cause vehicle emitted pollutant levels to peak. Such conditions resulted in the highest risks of preterm birth for African Americans and Hispanics, and also for teenage mothers and mothers giving birth at age 35 or older. In a region where a large proportion of the most polluted neighborhoods are those that are home to the poorest women and children with the least resources, these women and their offspring are clearly the groups that carry the greatest burden of our collective air pollution problem.

IMPACT OF AIR POLLUTION ON CHILDREN'S RESPIRATORY HEALTH



There are several biological reasons why young children may be more susceptible to air pollution's effects. Children's lungs, immune system, and brain are immature at birth and continue to rapidly develop until approximately age 6, and the cell layer lining the inside of the respiratory tract is particularly permeable during this age period. Compared to adults, children also have a larger lung surface area in relation to their body weight, and breathe 50% more air per kilogram of body weight. The process of early growth and development is important for the health

Asthmatic children with higher levels of traffic within approximately 500-1000 feet of their home were more likely to report emergency room visits and/or hospitalizations due to asthma compared to those living in lower traffic areas.

of the child in general, and therefore may also be a critical time when air pollution exposures can have lasting effects on future health. Additionally, children tend to spend more time outdoors doing strenuous activities, such as playing sports, so they are breathing more outdoor air compared to adults, who spend on average about 90% of their time indoors.

One of the most comprehensive, long-term studies to date examining the impact of air pollution exposure on children's respiratory health is the Children's Health Study (CHS). Beginning in 1992, University of Southern California researchers collected data from over 6,000 children attending public schools in 12 selected Southern California communities with varying levels of air pollution for a period of 8 or more years. This study has reported on several important findings, including short-term effects of air pollution, such as acute respiratory illnesses and asthma attacks, and longer-term health effects, such as chronic respiratory diseases and development of asthma. For example, researchers found that short-term increases in ozone concentrations were associated with increased school absences due to respiratory illnesses. There is now ample evidence from this and many other studies showing that ozone and particles exacerbate symptoms in asthmatic children. CHS researchers also reported reduced lung function and increased chronic cough and bronchitis in young children and teens chronically exposed to high levels of air pollution, especially those living in areas with high particle concentrations. Southern California children playing many outdoor sports in high-ozone areas were three times more likely to develop asthma. Generally researchers agree that air pollution can trigger asthma attacks and worsen asthma symptoms, but studies have not yet provided unequivocal evidence that it can cause asthma to develop.

Most health effect studies have focused on "criteria" air pollutants regulated under the Clean Air Act, such as CO, ozone, PM10, PM2.5 and NO2, but there is also an increasing awareness that pollutants not routinely measured may be responsible for the detrimental respiratory effects of air pollution, especially UFP from combustion of gasoline and diesel. Animal studies have shown that UFP cause inflammation in respiratory systems and greater allergic reactions, and diesel particles can carry allergens into the body, resulting in a magnified allergic sensitivity and response. Given the evidence these particles may have important effects on the lung, and the fact that children's lungs may be more susceptible to damage, additional research on the effects of ultra fine particles in the context of children's respiratory health are needed.

Many studies in Southern California and around the world have linked exposure to traffic with various childhood respiratory health

outcomes. Based on the CHS, children living near freeways and heavy traffic have markedly reduced lung function by the time they become adults, and are more likely to have asthma. In our own work in LA County, we studied the relationship between living near heavy traffic roadways and the risks of asthma symptoms and attacks using California Health Interview Survey (CHIS) data. We found that asthmatic children with higher levels of traffic within approximately 500-1000 feet of their home were more likely to report emergency room visits and/or hospitalizations due to asthma compared to those living in lower traffic areas. In another study that focused on low socioeconomic status neighborhoods, we found that asthmatic children living in areas with higher carbon monoxide levels were more likely to report asthma attacks, compared to those living in neighborhoods with lower CO levels. CO is directly emitted by vehicle tailpipes and concentrations of this pollutant are elevated closer to heavy traffic roadways. But CO is not known to have any inflammatory effects in the lung, thus, we hypothesize that CO could be acting as a marker for other toxins in vehicles exhaust such as UFP and PAHs.

CONCLUSION AND GRADE

Compared to the inhalation of cigarette smoke during active or passive smoking, the gases and particles in ambient air pollution are relatively diluted, resulting in relatively small risk increases for reproductive and children's health outcomes. For the most part, the increased risks for pregnancy outcomes in more polluted versus less polluted areas range from 10 to 30 percent for preterm birth and low birth weight, and between 5 and 20 percent for infant mortality. Yet when we consider the number of infants, children, and pregnant women affected by air pollution in Southern California, and that these outcomes are not rare, even small relative increases in risk translate into large numbers making the total impact due to air pollution a major problem. Certain vulnerable population segments -- the poorest and most exposed -- may experience much higher risks.

The question then arises what we can do to make the air cleaner and safer in terms of reproductive and children's health. Currently, air pollution impacts on pregnant women and infants are not taken into consideration in setting environmental standards on a state or federal level. Since these populations are perhaps the most vulnerable to air pollution, taking these impacts into consideration would help strengthen air quality controls.

The recently-released South Coast Air Quality Management Plan states that we will not be able to meet federal air quality standards without even stricter controls on motor vehicle exhaust and industrial sources. To achieve air clean enough to have only negligible effects on pregnancy, infant and children's health will likely require drastic changes to motor vehicles and transportation systems, as well as industrial processes, all of which take many years or decades. As with the previous Report Card articles, we strongly commend the air quality improvements over the past 30 years in Southern California. However, until policies are implemented to achieve the air quality standards, vulnerable populations will continue to suffer from higher morbidity and mortality. Our overall grade for Southern California's air pollution in relation to the health of pregnant women, infants and children is therefore a C.

Acknowledgment

We would like to acknowledge our doctoral research assistant, Ms. Jo Kay Ghosh for her contributions and help with editing.

Additional Reading

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Beate Ritz, MD, Ph.D., is Professor and Vice Chair the Department of Epidemiology with co-appointments in the department of Environmental Health at the UCLA School of Public Health and the department of Neurology UCLA School of Medicine; she is a member of the Center for Occupational and Environmental Health, the NIEHS-UCLA-USC Environmental Health Science Center, and co-director of the NIEHS-funded UCLA Center for Gene-Environment Studies of Parkinson's disease. Her primary research interests are the effects of occupational and environmental toxins such as pesticides, ionizing radiation, and air pollution on chronic diseases including neurodegenerative disorders (Parkinson's disease), cancers, and adverse birth outcomes including birth defects and asthma. She currently studies the effects of air pollution on adverse birth outcomes and asthma in children in Southern California and investigates the long-term effects of pesticide exposures on Parkinson's disease and cancers. Her research involves extensive geographic information system (GIS) modeling of environmental exposures including pesticide use and traffic related air pollution in California and the application of hierarchical modeling procedure of longitudinal data in cohort studies. She is directing or collaborating in a large number of federally (NIEHS), state (California Air Resources Board), and foundation (Michael J Fox Foundation) funded research projects.



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Michelle Wilhelm, Ph.D., is an Assistant Professor In Residence in the Department of Epidemiology and member of the Center for Occupational and Environmental Health, School of Public Health, UCLA. Dr. Wilhelm's research focuses on the impact of environmental exposures, particularly air pollution, on perinatal and early childhood health. She published the first study examining associations between residential proximity to traffic during pregnancy and adverse birth outcomes and is particularly interested in how Geographic Information Systems and other novel approaches such as biomarkers can be used to estimate exposure to traffic generated air pollution in large epidemiologic studies. In addition to her work on birth outcomes, she is also currently involved in several studies concerning air pollution and exacerbation of asthma in Californian children. Dr. Wilhelm holds a bachelor's degree in Geography, a M.S. in Environmental Health Sciences and a Ph.D. in Epidemiology, all from UCLA. She currently teaches courses in Environmental, Occupational and Reproductive Epidemiology in the School of Public Health, in addition to working on her research.

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Website and Print Production: Scott Gruber

Medical Illustrations: James Dowdalls

Graph Production: Jane Teis Graphic Services

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