Anne Cardwell - Fwd: More pdfs for Council packet on traffic and air quality

From: Charlie Knox
To: Anne Cardwell; Jayne York
Date: 9/30/2008 11:45 AM
Subject: Fwd: More pdfs for Council packet on traffic and air quality

>>> Marilyn Bardet <mjbardet@sbcglobal.net> 9/30/2008 11:38 AM >>>
Hi Charlie,
Here are two more fact sheets on tailpipe emissions and the impacts of idling, which is now a targeted campaign by Cal-EPA-ARB. The idling fact sheet is from Missoula City-County Air Quality Advisory Council. Interesting that a small city has such an advisory council on air quality.

--Marilyn
for Benicia First

VIII-B-428

Idling Fact Sheet

Compiled by the Missoula City-County Air Quality Advisory Council

Reducing idling reduces air pollution. Vehicle exhaust contains at least 21 air toxics which, by definition, are hazardous to human health. Major pollutants from automobiles include hydrocarbons, nitrogen oxides, carbon monoxide, and particulate matter, all of which have significant health and environmental impacts. Emissions from **idling** vehicles can be as much as 20 times greater than those from one traveling at 32 mph. Many communities in the United States and Canada have or are considering ordinances that restrict excessive vehicle **idling** in order to improve air quality and protect citizens' health.

Use the 30-second rule to save gas and reduce emissions. Contrary to the commonly held misconception, frequent restarting has little impact on engine components such as the battery and starter motor. Your savings in fuel will easily offset the cost of what little wear does result. Remember, when you are idling, you are getting zero miles per gallon. The U.S. EPA's website states, "You will save gas by turning the engine off and restarting it again if you expect to idle for more than 30 seconds. You will also prevent pollution by preventing long idles. Try parking your car and going into restaurants, banks, and the like instead of idling in drive-up lanes." When you must wait in a drive thru, turn your engine off.

The best way to warm up your vehicle is to drive it. It's a common misconception that **idling** for several minutes is the best way to warm up a vehicle. Not only is extended **idling** unnecessary, but many parts of the vehicle—including wheel bearings, tires, and the suspension system—only warm up once the vehicle is moving. You only need to idle long enough to get the oil circulating—about 30 seconds—before driving away, and it's a good idea to avoid high speeds and fast acceleration until the engine temperature rises. Modern diesel engines also need only a short engine warm up times. To keep windows from fogging up, clear snow from the

air intake on top of your hood (before you start the engine) and open a window slightly as soon as you get in the car. When temperatures are in the teens or colder, use an engine block heater for 2-4 hours to help your car start more easily, get your defroster working faster, improve your winter gas mileage as well as reduce air pollution (see Engine Block Heater Fact Sheet for more details).

Reducing idling reduces wear and tear on your engine and saves money. Idling creates wear and tear on your engine because fuel doesn't combust completely, and some fuel residue can condense on cylinder walls. Also, excessive idling can cause condensation to form in the exhaust, which may result in corrosion and reduced lifespan of the exhaust system. Idling for 10 minutes a day uses an average of 26 gallons of gas a year. At $2 a gallon, a driver could save over $50 a year in gasoline costs just by turning off the engine.

Unattended idling vehicles are unsafe, illegal, and vulnerable to theft. Not only does common sense tell us that leaving a running vehicle unattended can be dangerous, but it is also illegal to do so. Specifically, Missoula's Municipal Code on Unattended Motor Vehicles (10.14.050) states that, "No person driving or in charge of any motor vehicle except a licensed delivery truck or other delivery vehicle, shall permit it to stand unattended without first stopping the engine, locking the ignition and removing the key." Unattended **idling** vehicles are not only unsafe and illegal, they are an open invitation to easy theft!

Some other things you can do to improve air quality: (1) Plan ahead to combine errands or avoid the trip altogether; (2) bike, walk, ride the bus, or carpool; (3) maintain your car regularly; (4) drive smoothly and avoid sudden throttling.

For more information, visit the following websites or call the Health Department at 258-4755.

Missoula County Environmental Health Division—http://www.co.missoula.mt.us/EnvHealth

Missoula Municipal Code—http://www.ci.missoula.mt.us/cityclerk/city_code.htm

"**Idling** and climate change go hand in hand," Natural Resources Canada, Office of Energy Efficiency— http://oee.nrcan.gc.ca/transportation/**idling**/issues/why-**idling**-problem.cfm?attr=8

US EPA—http://www.epa.gov

"Your car and clean air: What YOU can do to reduce pollution" (**idling** info on p.3)—http://www.epa.gov/otaq/consumer/18-youdo.pdf

Mobile source emissions: Past, present, and future (Pollutants): http://www.epa.gov/otaq/invntory/overview/pollutants/index.htm

Mobile source air toxics: http://www.epa.gov/otaq/toxics.htm

National Safety Council, Environmental Health Center, "Environmentally friendly maintenance and repair"—http://www.nsc.org/ehc/mobile/mainrepa.htm

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Automotive Exhaust Chemicals: disease causing effects

A short list of the likely pathogens in car exhaust:

- Carbon Monoxide
- Nitrogen dioxide
- Sulphur dioxide
- Suspended particles including PM-10, particles less than 10 microns in size.
- Benzene
- Formaldehyde
- Polycyclic hydrocarbons

Background Information - Adverse Health Effects of Chronic Exposure to Petroleum Combustion Products. On November 18, 1994, the first-ever conference on "Air Pollution: Impacts on Body Organs and Systems" was held in Washington, D.C. by the National Association of Physicians for the Environment. An abridged version follows. The relevance of this perspective increases with increasing air pollution and climate change. 2008 Update.

Jaffe and Badman at the same conference summarized the effects of polluted air :

"Blood perfuses all of the body's organs and can carry toxic substances as well as beneficial substances, such as oxygen, to them. Air pollution is the source of many materials that may enter the human bloodstream through the nose, mouth, skin, and the digestive tract. Chemicals known to be harmful, such as benzene, lead and other heavy metals, carbon monoxide, volatile nitrites, pesticides, and herbicides. These substances have been shown to produce harmful effects on the blood, bone marrow, spleen, and lymph nodes. Blood cells are constantly undergoing turnover, with new blood cells entering the circulation as mature cells are lost, making the

blood system especially vulnerable to environmental poisoning. For example, lead interferes with normal red blood cell formation by inhibiting important enzymes. In addition, lead damages red blood cell membranes and interferes with cell metabolism in a way that shortens the survival of each individual cell. Each of these harmful effects can result in clinical anemia.

Benzene and other less known hydrocarbons are produced in petroleum refining, and are widely used as solvents and as materials in the production of various industrial products and pesticides. Benzene also is found in gasoline and in cigarette smoke. It has been shown that exposure to benzene is related to the development of leukemia and lymphoma. Benzene has a suppressive effect on bone marrow and it impairs blood cell maturation and amplification. Benzene exposure may result in a diminished number of blood cells or total bone marrow loss. A number of metabolites appear to be involved in this process, and there may be several targets of toxicity, including stem, progenitor, and some stromal cells.

Common air pollutants also have an affect on blood and thus on organs of the body. For example, carbon monoxide binds to hemoglobin two hundred times more avidly than oxygen and distorts the release to the tissues of any remaining oxygen. Thus, CO poisoning is a form of suffocation. Carbon monoxide can exacerbate cardiovascular disease in humans. Some airborne chemicals stimulate the immune system to activate leukocytes and macrophages that can produce tissue damage, especially to the cells that line human blood vessels. The combined effect of these events is to accelerate the changes that eventually lead to hypertension and ischemic heart disease.

Cory-Slechta and Lundberg discussed the adverse effects of pollution on the central nervous system: "The central nervous system (CNS) is the primary target for many serious air pollutants, such as lead, which is a major environmental hazard. Research provided evidence that levels of lead exposure associated with central nervous system effects, particularly as manifest in behavioral changes was lower than previously realized. Blood lead concentrations in children were not considered problematic until they exceeded 30 to 40 micrograms per deciliter ('g/dL); however, studies demonstrated

changes in cognitive function at blood concentrations as low as 10 to 15 'g/dL. While children are more susceptible to lead's CNS effects, adults exhibit similar deficits in learning and memory. Advanced aging increases vulnerability to the toxic effects of lead. In Germany, a large study documented an age-related decline in bone lead concentrations with advancing age. This effect was more pronounced in women than in men, reflecting post-menopausal processes in women which contribute to bone resorption and the release of lead back into the bloodstream. These results mean that brain lead exposure is actually increased during a period of already heightened susceptibility due to concurrent degeneration of other physiological functions, including both CNS and renal functions.

Although lead is the most studied of hundreds of known or suspected neurotoxic air pollutants, other heavy metals, pesticides, and organic solvents also cause neurobehavioral dysfunction. Expanded research in behavioral neurotoxicology is urgently needed. Changes in mood, cognition, and behavior are endpoints that need to be evaluated in addition to cancer rates or mortality data and may be more common. In various studies, increased levels of air pollutants are accompanied by increased psychiatric emergency calls and hospital admissions, behavior changes, and a lessened sense of well-being. Irritating odors and cigarette smoke have been found to increase aggressive behavior, and to decrease helping behavior and altruism, leading to a degradation of social interaction."

Goldstein and Albright discussed immune system effects: "The effects of airborne pollutants on the immune system have been most widely studied in the respiratory tract. An airborne pollutant may enter the respiratory tract as a volatile gas (e.g., ozone, benzene), as liquid droplets (e.g., sulfuric acid, nitrogen dioxide), or as particulate matter (e.g., components of diesel exhaust, aromatic hydrocarbons). These pollutants interact with the immune system and may cause local and systemic responses ranging from overactive immune responses to immunosuppression. Most airborne pollutants are small molecular weight chemicals that must be coupled with other substances (e.g., proteins or conjugates) before they can be recognized by the immune system and cause an effect. Some disorders which may occur because of pollutants in the

respiratory system are the following:

Immunosuppression can be demonstrated following exposure to polycyclic aromatic hydrocarbons (e.g.,tetrachlordibenzo-p-dioxin).

Hypersensitivity reactions (e.g., occupational asthma) can occur following exposure to toluene diisocyanate and other volatile chemicals.

There is clearly an underlying genetic basis for susceptibility to immunologic disease resulting from exposure to pollutants, but knowledge in this area is rudimentary at this time. For example, there is little understanding of genetically-determined susceptibility or resistance to pollutant-induced immune disorders. There is a lack of appropriate in vitro models, and it is difficult to identify specific, biologically-active substances that may be linked to immune disorders."

Workplace Exposure to Vehicle Exhaust and Chronic Illness

Here is a summary of a report on one patient with a known chemical exposure, as an example: Mr. A had been exposed to diesel and gasoline engine exhaust in the workplace from 1982 onward and had become too ill to work by Oct. 1993. Improvements to his work environment were not made until 1992, but apparently these were not adequate and he continued to be ill through 1993. He developed a chronic illness in the mid-80's which was slowly progressive and he beleived that chemical exposure at work had made him ill.

Mr. A manned a parts counter in a vehicle repair shop. The ventilation of this indoor work-space was judged to be inadequate and Mr A was exposed to significant concentrations of exhaust products which include a number of pathogenic possibilities.

His chief complaints were: chronic fatigue, recurrent flu-like illness, limited exertional tolerance, vertigo, dyspepsia with epigastric pain, chest pain with episodes of shortness of breath, cognitive dysfunction, anxiety. He described progressive dysfunction over several years and was concerned that exhaust exposure at work was making him ill. He described a progressive loss of tolerance for physical exertion - attempts at vigorous exertion left him exhausted, weak, and dizzy with cognitive

dysfunction; recovery from, for example, attempts to play ice-hockey would often take 48 hour or longer. He had trouble concentrating and suffered recent memory loss. He often felt severe fatigue with difficulty concentrating and memory drop outs at work. He withdrew from work Oct. 1993. He was more attentive to food choices and good nutrition and attempted to follow a graded exercise program. He reported benefit by staying away from the work environment and controlling his diet.

Prolonged exposure to exhaust gases seem to induce allergy symptoms or hypersensitivity. Activated immune systems tend to start reacting to airborne and food antigens - exposed patients begin reacting to food materials following a typical pattern of chronic illness, and routinely report intolerance to even short exposures of low concentrations of airborne chemicals. Unfortunately there is very little research to help us understand this problem. Since chronic fatigue syndrome has become a common term, studies have been reported showing a scatter of immune and endocrine abnormalities but no simple diagnostic test has emerged. Tests of indoor air quality were conducted but were inadequate to access his exposure.

INDEX CHEMICALS BENZENE AND THE POLYCYCLIC HYDROCARBONS ARE INDEX CHEMICALS - EXAMPLES OF THE MANY CHEMICALS PRESENT IN COMBUSTION PRODUCTS AND WHILE THE DESCRIPTIONS OF THE CHEMICALS ARE VERY GENERAL, THE READER SHOULD HAVE NO DOUBT THAT CHRONIC EXPOSURE CAN PRODUCE PROFOUND AND LONG-LASTING CHANGES IN BIOLOGICAL FUNCTION. THIS INFORMATION IS TAKEN DIRECTLY FROM INFORMATION BULLETINS ISSUED BY THE U.S. AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY DIVISION OF TOXICOLOGY 1600 CLIFTON ROAD, E-29ATLANTA, GEORGIA 30333 USA.

Benzene The greatest possibility for high-level exposures is in the workplace... most people are exposed to benzene in tobacco smoke and automobile exhaust. Benzene has been found in at least 337 of 1177 National Priorities List hazardous waste sites. Other environmental sources of benzene include gasoline (filling) stations, vehicle exhaust fumes, tobacco smoke, underground storage tanks that leak, wastewater from industries that use benzene, chemical spills, groundwater next to landfills containing benzene, and possibly some food products that contain benzene naturally. Brief Exposure at High

Levels--Death may occur in humans and animals after brief oral or inhalation exposures to high levels of benzene; however, the main effects of these types of exposures are drowsiness, dizziness, and headaches. These symptoms disappear after exposure stops.

Long-Term Exposures at Various Levels--From overwhelming human evidence and supporting animal studies, the U.S. Department of Health and Human Services has determined that benzene is carcinogenic. Leukemia (cancer of the tissues that form the white blood cells) and subsequent death from cancer have occurred in some workers exposed to benzene for periods of less than 5 and up to 30 years. Long-term exposures to benzene may affect normal blood production, possibly resulting in severe anemia and internal bleeding. In addition, human and animal studies indicate that benzene is harmful to the immune system, increasing the chance for infections and perhaps lowering the body's defense against tumors. Exposure to benzene has also been linked with genetic changes in humans and animals.

Animal studies indicate that benzene has adverse effects on unborn animals. These effects include low birth weight, delayed bone formation, and bone marrow damage. Some of these effects occur at benzene levels as low as 10 parts of benzene per million parts of air (ppm). Although benzene has been reported to have harmful effects on animal reproduction, the evidence for human reproductive effects, such as spontaneous abortion or miscarriage, is too limited to form a clear link with benzene.

Benzene can be measured in the blood and the breath. The body changes benzene to phenol, which can be measured in the urine. Amounts of benzene in blood samples and phenol in urine samples cannot be used as yet to predict what degree of harmful health effects may occur. The meaning of benzene and phenol measurements in blood and urine should be viewed carefully for several reasons: 1) phenol occurs naturally in urine, and amounts of urinary phenol would have to be much higher than usual before any measurement was meaningful; 2) present test methods are limited and raise doubts about the blood level values found in some laboratories; 3) because smoking can raise the background level of benzene in the blood, smoking habits must be considered when evaluating exposure to benzene; 4)

benzene disappears rapidly from the blood and measurements may be accurate only for recent exposures; 5) average amounts of benzene found in the body have not been determined for the general population.

The Environmental Protection Agency (EPA) set the maximum permissible level in drinking water at 5 parts of benzene per billion parts of water (ppb). Because benzene can cause leukemia, EPA established an ultimate goal of 0 ppb for benzene in drinking water and in ambient water such as rivers and lakes. EPA realizes that this goal may be unattainable and has estimated how much benzene in ambient water would be associated with one additional cancer case for every 100,000 persons (6.6 ppb benzene), one case for every 1 million persons (0.66 ppb benzene), and one case for every 10 million persons (0.066 ppb benzene). The National Institute for Occupational Safety and Health (NIOSH) has recommended an occupational exposure limit in air of 0.1 part of benzene per million parts of air (ppm). The Occupational Safety and Health Administration's (OSHA) legally enforceable limit is an average of 1.0 ppm over the standard 8-hour workday, 40-hour workweek.

Polycyclic aromatic hydrocarbons (PAHs)

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances. PAHs can be man-made or occur naturally. There is no known use for most of these chemicals except for research purposes. A few of the PAHs are used in medicines and to make dyes, plastics, and pesticides. They are found throughout the environment in the air, water and soil. There are more than 100 different PAH compounds. Although the health effects of the individual PAHs vary, the following 15 PAHs are considered as a group with similar toxicity: acenaphthene, acenaphthylene, anthracene, benzanthracene, benzopyrene, benzofluoranthene, benzoperylene, benzofluoranthene, chrysene dibenzanthracene, fluoranthene, fluorene, indenopyrene, phenanthrene, pyrene.

Several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route

by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health. As pure chemicals, PAHs generally exist as colorless, white, or pale yellow-green solids. Most PAHs are found as mixtures of two or more PAHs. They can occur in the air either attached to dust particles, or in soil or sediment as solids. They can also be found in substances such as crude oil, coal, coal tar pitch, creosote, road and roofing tar. Most PAHs do not dissolve easily in water, but some PAHs evaporate into the air. PAHs generally do not burn easily and they will last in the environment for months to years.

PAHs that are attached to dust and other particles in the air and originate from vehicle exhausts, asphalt roads, coal, coal tar, wildfires, agricultural burning and hazardous waste sites. Background levels of PAHs in the air are reported to be 0.02-1.2 milligrams per cublic meter (mg/m3) in rural areas and 0.15-19.3 mg/m3 in urban areas. You may be exposed to PAHs in soil near areas where coal, wood, gasoline, or other products have been burned or from the soil on or near hazardous waste sites, such as former manufactured-gas sites and wood-preserving facilities. PAHs have been found in some drinking water supplies in the United States. The background level of PAHs in drinking water ranges from 4 to 24 nanograms per liter . For most people, the greatest exposure to PAHs occurs in the workplace.

PAHs can enter the body through the lungs. PAHs enter the body quickly and easily by all routes of exposure. The rate at which PAHs enter your body is increased when they are present in oily mixtures and tend to be stored in the kidneys, liver, and fat, with smaller amounts in the spleen, adrenal glands and ovaries. Results from animal studies show that PAHs do not tend to be stored in for a long time and are excreted within a few days in the feces and urine.

The U.S. Department of Health and Human Services has determined that PAHs may be carcinogens. Several of the PAHs, including benzanthracene, benzopyrene, benzofluoranthene, benzofluoranthene, chrysene, dibenzanthracene, indenopyrene have caused tumors in laboratory animals when they ate them, when they were applied to their skin and when they breathed them in the air for long periods of time.

Reports in humans show that individuals exposed by breathing or skin contact for long periods of time to mixtures of other compounds and PAHs can also develop cancer. Mice fed high levels of benzopyrene during pregnancy had difficulty reproducing and so did their offspring. The offspring from pregnant mice fed benzopyrene also showed other harmful effects, such as birth defects and decreased body weight. Similar effects could occur in humans, but we have no information to show that these effects do occur.

Studies in animals have also shown that PAHs can cause harmful effects on skin, body fluids, and the body's system for fighting disease after both short- and long-term exposure. These effects have not been reported in humans. PAHs are changed into chemicals that can attach to substances within the body. The presence of PAHs attached to these substances can then be measured in body tissues or blood after exposure to PAHs. However, this test is still being developed and it is not known yet how well it works. PAHs or their breakdown products can also be measured in urine. Although these tests can tell that you have been exposed to PAHs, it is not yet possible to use these tests to predict the severity of any health effects that might occur or to determine the extent of your exposure to the PAHs. These tests are not routinely available at a doctor's office because they require special equipment for sampling and detecting these chemicals.

Anne Cardwell - Fwd: USC summary of Southern California Children's Health Study--for council packet

From: Charlie Knox
To: Anne Cardwell; Jayne York
Date: 9/30/2008 12:30 PM
Subject: Fwd: USC summary of Southern California Children's Health Study--for council packet

more for the packet

>>> Marilyn Bardet <mjbardet@sbcglobal.net> 9/30/2008 12:00 PM >>>
Charlie,
This article posted by USC refers to the Southern California Children's Health Study, a long-term study undertaken between 1993 and 2001, which deals with lung function development of children chronically exposed to ozone. Dr. Tager discussed this study at Benicia First's forum. He said that 1500 children were studied for lung function over 8 years. For the So-Cal study, he described the "zone of influence" that would characterize effects of traffic pollution as the distance of 500 meters from a freeway.

Dr. Tager cited residential distance to freeways and traffic exposure as highly significant impacts on lung function. "Lung function is a better indicator of mortality--better than blood pressure." (Quote from my notes taken at the forum.) He also said that lung sacs develop fully in the first 20 weeks of life. Lung function is diminished as measured between ages 10 and 18 years.

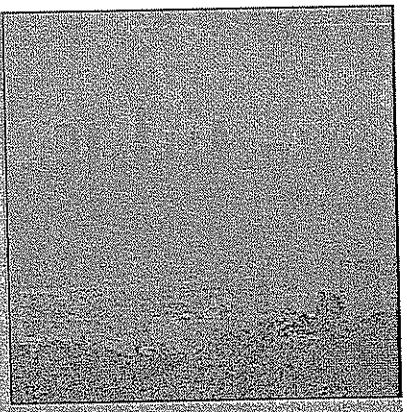
USC News

Smog May Cause Lifelong Lung Deficits

09/08/04

A long-term USC study following the pulmonary health of children in polluted L.A. areas signals likely health problems in adulthood.

By Alicia Di Rado



By age 18, the lungs of many children who grow up in smoggy areas are underdeveloped and will likely never recover, according to a study in this week's issue of the New England Journal of Medicine.

The research is part of the Children's Health Study, the longest investigation ever into air pollution and kids' health.

Between 1993 and 2001, study scientists from the Keck School of Medicine of USC tracked levels of major pollutants in 12 Southern California communities while following the pulmonary health of 1,759 children as they progressed from 4th grade to 12th grade.

The 12 communities included some of the most polluted areas in the greater Los Angeles basin, as well as several low-pollution sites outside the area.

Keck School researchers previously found that children who were exposed to more air pollution scored more poorly on respiratory tests. In this latest study, researchers analyzed the same children's respiratory health at age 18, when lungs are almost completely mature.

"Teenagers in smoggy communities were nearly five times as likely to have clinically low lung function, compared to teens living in low-pollution communities," said W. James Gauderman, associate professor of preventive medicine at the Keck School and lead author of the study.

People with clinically low lung function have less than 80 percent of the lung function expected for their age – a significant deficit that would raise concerns during a doctor's exam.

"When we began the study 10 years ago, we had no idea we would find effects on the lung this serious," said John Peters, Hastings Professor of Preventive Medicine in the Keck School, director of the Southern California Environmental Health Sciences Center and senior author of the study.

Study technicians traveled to participating schools every year and tested children's lung function, a measure of how well their lungs work. As an example, someone with sub par lung function cannot exhale and blow up a balloon as quickly or as big as someone with good lung function.

Researchers correlated the students' lung health measurements with levels of air pollutants monitored in the communities during the same time period.

They found greater deficits in lung development in teenagers who lived in communities with higher average levels of nitrogen dioxide, acid vapor, particulate matter with a diameter of less than 2.5 micrometers (about a tenth the diameter of a human hair) and elemental carbon.

"These are pollutants that all derive from vehicle emissions and the combustion of fossil fuels," Gauderman said.

Deficits in lung function have both short- and long-term effects.

"If a child or young adult with low lung function were to have a cold, they might have more severe lung symptoms, or wheezing," Gauderman said. "They may have a longer disease course, while a child with better lung function may weather it much better."

Potential long-term effects are more alarming. "Low lung function has been shown to be second only to smoking as a risk factor for all-cause mortality," Gauderman said.

Lung function grows steadily as children grow up, peaking at about age 18 in women and sometime in the early 20s in men. Lung function stays steady for a short time and then declines by 1 percent a year throughout adulthood.

As lung function decreases to low levels in later adulthood, the risk of respiratory diseases and heart attacks increases.

Researchers are unsure how air pollution may retard lung development.

Gauderman believes chronic inflammation may play a role, with air pollutants irritating small airways on a daily basis. Scientists also suspect that pollutants might dampen the growth of alveoli – tiny air sacs in the lungs.

The research team will continue to follow the study participants into their early 20s, when their lungs will mature and stop developing entirely. The team seeks to find out if the participants begin to experience respiratory symptoms and if those who moved away from a polluted environment show benefits.

The California Air Resources Board, National Institute of Environmental Health Sciences and Hastings Foundation supported the research.

From: Marilyn Bardet <mjbardet@sbcglobal.net>
To: Charlie Knox <Charlie.Knox@ci.benicia.ca.us>
Date: 9/30/2008 11:06:10 AM

Charlie,
Here are (2) different, additional materials for the Council packet in pdf form. I'll be sending more...
–Marilyn

CC: Jerry Page <Jkjerome@aol.com>, Steve Goetz <sgoet@sbcglobal.net>, Anne Cardwell <Anne.Cardwell@ci.benicia.ca.us>

Air Pollution and Children's Health

A fact sheet by
Cal/EPA's Office of Environmental Health Hazard Assessment and
The American Lung Association of California.

In the past 30 years, state and federal air-quality programs have made great progress in reducing air pollution. However, there has been an increasing awareness in recent years that children may be more susceptible than adults to the harmful effects of air pollutants.

The California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) assesses health risks from environmental contaminants. OEHHA currently is reviewing whether the state's ambient air quality standards are adequate to protect the health of infants and children and is working to identify toxic air contaminants that may cause infants and children to be especially susceptible to illnesses. The American Lung Association of California (ALAC) and its 15 local associations work to prevent lung disease and promote lung health. Since 1904, the American Lung Association has been fighting lung disease through education, community service, advocacy and research.

This fact sheet by OEHHA and ALAC provides information on air pollution and children's health.

Why may children be at greater risk than adults from air pollution?

In many cases, children may have greater exposure than adults to airborne pollutants. Infants and children generally breathe more rapidly than adults, which increases their exposure to any pollutants in the air. Infants and children often breathe through their mouths, bypassing the filtering effect of the nose and allowing more pollutants to be inhaled. Children generally spend significantly more time outdoors than adults, especially during summer months when smog levels are highest.

Children are often more susceptible to the health effects of air pollution because their immune systems and developing organs are still immature. For example, lead that is inhaled is more easily deposited in the fast-growing bones of children. Irritation or inflammation caused by air pollution is more likely to obstruct their narrower airways. It may also take less exposure to a pollutant to trigger an asthma attack or other breathing ailment due to the sensitivity of a child's developing respiratory system. Exposure to toxic air contaminants during infancy or childhood could affect the development of the respiratory, nervous, endocrine and immune systems, and could increase the risk of cancer later in life.

What are the major kinds of air pollutants and their impacts on children?

"Criteria" Air Pollutants

Several common air pollutants are regulated under the state and federal Clean Air Acts and are known as "criteria" air pollutants. Two of the most widespread criteria pollutants are particulate matter (PM) and ozone. PM consists of microscopic particles less than one-seventh the width of a human hair. These particles come from a variety of both manmade and natural sources, such as diesel engines, smoke from fireplaces as well as forest and agricultural fires, and dust from tilled farmland. PM can bypass the body's natural defenses and penetrate deep into the lungs. The elderly, children and people with existing respiratory or cardiac diseases are considered to be especially sensitive to the harmful effects of PM. Recent studies suggest that PM may exacerbate asthma and cause coughs and other respiratory symptoms in children. Recent studies also suggest that prolonged exposure to PM may also affect the growth and functioning of children's lungs. Researchers found that as children grow up in smoggier areas, there is a notable lag in lung function growth.

Ozone is the major component of urban smog. It is formed by chemical reactions in the atmosphere involving sunlight and various gases in motor vehicle exhaust and industrial emissions. Ozone is a powerful respiratory irritant that can cause lung inflammation, transient decreases in lung function, shortness of breath, chest pain, wheezing, coughing and exacerbation of respiratory illnesses such as asthma. Long-term and repeated ozone exposures may lead to chronically reduced lung function.

OEHHA provides detailed analyses of health information on PM, ozone and other common pollutants to the California Air Resources Board (ARB), which sets ambient air quality standards for those pollutants. These air quality standards have been established at levels that are intended to protect the health of all Californians. Unfortunately, PM and ozone levels in most urban areas of California frequently exceed the ambient air quality standards. ARB and local air districts operate regulatory programs under state and federal requirements to reduce airborne levels of these pollutants to the ambient air quality standards.

Other "criteria pollutants" include nitrogen dioxide, carbon monoxide, lead, sulfur dioxide, sulfates and hydrogen sulfide.

ARB is sponsoring a major 10-year study of the effects of air pollution on children's health. Information on this study is available at ARB's web site at http://www.arb.ca.gov/research/abstracts/98-320.htm.

Toxic Air Contaminants

California also regulates the emissions of other pollutants, known as toxic air contaminants, which may pose a present or potential hazard to human health or contribute to an increase in deaths or serious illnesses. OEHHA provides assessments of the health risks from various toxic air contaminants to ARB, which can enact control measures designed to reduce the exposure of Californians to these contaminants. More than 200 chemicals are currently listed as toxic air contaminants in California.

Many toxic air contaminants are present in motor vehicle exhaust and industrial emissions, and are formed from the combustion of other chemicals. Among these byproducts of combustion are dioxins and polycyclic organic matter (POM), which may

affect the development of the fetus and increase cancer risks later in life. Particles found in diesel exhaust may make children more susceptible to allergies and asthma. Other toxic contaminants have numerous sources. Acrolein, which may exacerbate asthma, is found in motor vehicle exhaust, tobacco and wood smoke, some industrial emissions, and is also formed in the atmosphere from chemical reactions involving other pollutants. Some toxic air contaminants, such as lead, are naturally occurring in the environment. Lead has been banned as an additive in gasoline and household paint but is present in some industrial emissions. It can cause developmental problems and harm the central nervous system.

What is California doing to improve health protection for children?

The Legislature in 1999 approved the Children's Environmental Health Protection Act (Senate Bill 25), authored by Senator Martha Escutia, which seeks to ensure that California's air quality programs protect the health of infants and children. The Act requires ARB, in consultation with OEHHA, to review all ambient air quality standards to determine whether they adequately protect the health of the public, including children. The Act also requires OEHHA to identify toxic air contaminants that may cause infants and children to be especially susceptible to illness, and it requires ARB to determine the adequacy of existing control measures for toxic air contaminants or the need for new control measures to protect the health of the public, particularly infants and children.

The initial stage of the ambient air quality standards review was completed in December 2000. ARB and OEHHA concluded that PM and ozone may cause health effects in children even at levels meeting the state's ambient air quality standards. The amount of time children play outdoors and their higher breathing rates are some of the reasons why children may be more sensitive to these pollutants than adults. The review also found evidence that levels of nitrogen dioxide (a pollutant in motor vehicle exhaust and many kinds of industrial emissions) that meet the ambient air quality standard may harm asthmatic children.

OEHHA completed a detailed review of the PM standards in May 2002. Based on this review, OEHHA recommended, and ARB adopted, stricter new PM standards in June 2002. (The report containing the results of the PM review and the new standards can be accessed at www.oehha.ca.gov/air/toxic_contaminants/PM10notice.html#may). OEHHA is conducting a detailed review of the ozone standard and is developing recommended revisions to the standard for ARB's consideration. After this is completed, OEHHA will review the nitrogen dioxide standard.

ARB and local air districts may have to develop new regulations to reduce emissions and ultimately reduce airborne levels of these pollutants to comply with the new PM standards and any future new standards for ozone and nitrogen dioxide.

The Act also requires OEHHA to identify toxic air contaminants that may cause infants and children to be especially susceptible to illnesses. ARB will review and, if necessary, revise or adopt any control measures needed to reduce the public's exposure to these contaminants. The Act required OEHHA to identify up to five contaminants in 2001. These contaminants - dioxin, lead, POM, diesel exhaust particles, and acrolein - are briefly discussed in the previous section. More detailed information is available at www.oehha.ca.gov/air/toxic_contaminants/SB25finalreport.htm.

OEHHA is developing guidelines for use in evaluating the adequacy of existing air toxics regulations. Beginning in 2004, OEHHA will annually evaluate selected toxic air contaminants and identify unhealthful levels of exposure to these contaminants. OEHHA will use these evaluations beginning in 2005 to annually update the list of toxic air contaminants that impact infants and children.

The Act also requires ARB to evaluate the adequacy of its current air-quality monitoring program to determine children's exposure to air pollutants, and it created the Children's Environmental Health Center within the California Environmental Protection Agency to coordinate Cal/EPA's activities and provide advice to the Governor and Cal/EPA Secretary on matters of children's environmental health.

In addition to complying with the requirements of the Children's Environmental Health Protection Act, OEHHA is taking two other steps in the area of children's health protection. OEHHA is developing improvements to scientific methods used to gauge cancer risks that children may face from exposure to contaminants in the environment. Also, OEHHA is refining methods to assess health risks from contaminants that may conceivably exist at proposed and existing school sites. These two projects are intended to improve the ability of scientists and regulators to make decisions that protect children from contaminants in the air, water, soil and food as well as elsewhere in the environment.

These efforts represent a commitment by the State of California to ensure that children have the opportunity to grow up in a healthy environment.

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Fact Sheet
April 2002

California Environmental Protection Agency
Air Resources Board

Fresno Asthmatic Children's Environment Study (F.A.C.E.S.)

The Fresno Asthmatic Children's Environment Study, which began in 2000, is a large epidemiological study of the effects of air pollution on children with asthma. About 300 asthmatic children who reside in the Fresno area of the Central Valley of California will be enrolled in the study. The overall goal of this study is to determine the effects of different components of particulate matter (PM), in combination with other ambient air pollutants, on the natural history of asthma in young children. The study is sponsored by the California Air Resources Board and conducted by investigators at the University of California, Berkeley.

Importance of the Fresno Asthmatic Children's Environment Study

- The information provided by the study will help the Air Resources Board (ARB) protect public health. The ARB sets California's ambient air quality standards to protect people who are most sensitive to air pollution.
- Children may be more strongly affected by air pollution because their lungs and bodies are still developing. Understanding the effects of air pollution on children with asthma is essential for setting health standards protective of sensitive populations.

The Pollutants Studied

- Particulate matter
- Criteria air pollutants: NO_x, SO_2, CO, ozone
- Polycyclic Aromatic Hydrocarbons (PAHs)
- Volatile Organic Compounds (VOCs)
- Environmental Tobacco Smoke
- Allergens

The Information Gathered by the Study

The study consists of a variety of measurements taken over the course of five years. Measurements taken include skin testing for allergies, lung function testing, and extensive questions about the child's health and home environment. Research staff will also visit the child's home to collect indoor air and dust samples. Children will keep a journal of activities and time spent in different locations throughout the day, as well as symptoms and medication use. Portable spirometers will be used to measure lung function at home.

For More Information

Please contact the ARB's Public Information Office at (916) 322-2990, or visit our web site at http://www.arb.ca.gov/research/faces/faces.htm. You may obtain this document in an alternative format by contacting our ADA coordinator at (916) 322-4505 (voice); (916) 324-9531 (TDD, Sacramento area only); or (800) 700-8326 (TDD, outside Sacramento).