Cooking Up Problems for Babies

Wood Smoke and Low Birth Weight

Low birth weight, defined as less than 2,500 grams (5.5 pounds), places newborns at increased risk of illness and death during infancy. Low birth weight is particularly critical in the developing world, where approximately 18% of newborns are affected. This month, Erick Boy, a senior program specialist at the Micronutrient Initiative in Ottawa, Canada, and colleagues investigate whether maternal exposure to smoke from cook fires, a common indoor environmental pollutant in the developing world, might reduce infants' birth weight [***EHP* 110:109–114**]. Of environmental factors already associated with low birth weight, environmental tobacco smoke (ETS) has been well scrutinized, and the researchers suggest that this model could explain some of the birth weight reduction observed in their study.

Hearth and health. Exposure to the smoke from wood-fueled cook fires may affect birth weight through the same mechanism as environmental tobacco smoke.

The study was conducted in Quetzaltenango, a western Guatemalan province, and included 1,717 women and their infants. Infants were weighed within 72 hours of birth, and the mothers were surveyed on physical, demographic, and socioeconomic characteristics. A key interview question focused on the fuel used for cooking—wood, coal, electricity, or gas—and, if wood or coal, whether it was burned in an open fire or a stove with a chimney. There were so few coal users that they were grouped in with wood users in the paper's subsequent analyses.

Among the 1,717 mothers, 871 cooked with wood over an open fire during pregnancy, 489 used a wood stove with a chimney, and 357 employed electricity or gas. The mothers using an open fire had a slightly higher percentage of low birth weight infants, 19.9% versus 16.8% (stove with a chimney) and 16.0% (electricity or gas). Although small, the relationship between maternal use of open cook fires during pregnancy and birth weight reduction was determined to be statistically significant.

The researchers suggest that the well-studied link between ETS and low birth weight may provide a plausible model to explain this result. As with tobacco, combustion of biofuels such as wood produces carbon monoxide. When inhaled, it binds to hemoglobin and forms carboxyhemoglobin, which cannot transport needed oxygen to the body's tissues. A developing fetus, deprived of adequate oxygen, suffers intrauterine growth retardation and subsequent low birth weight.

Although the current study does not directly measure carbon monoxide exposure, other studies in the same geographic area indicate that ETS and cook fire smoke are associated with comparable ambient concentrations of carbon monoxide. Also, persons exposed to either may have similar concentrations of carboxyhemoglobin. In ETS studies, these concentrations have been associated with intrauterine growth retardation and low birth weight. The current study is the first to examine whether exposure to smoke from indoor cook fires might have the same effect.

In interpreting the study results, Boy and his colleagues controlled for numerous confounding factors. For one, fuel type is strongly linked to socioeconomic status—rural families use wood more frequently and also tend to be poorer. Further, there are numerous influences on birth weight, including premature birth and maternal nutrition, health, and socioeconomic status. As in other studies, these factors were significant in the Quetzaltenango study.

The researchers note that, although wood smoke may not carry as great a weight as other environmental pollutants such as ETS, the number of people affected by low birth weight around the world gives *any* risk factor a measure of importance. The researchers acknowledge the difficulties of determining the weight of each factor and suggest that future research might build on their results by quantifying carbon monoxide exposures and detailing them more exactly. **–Julia R. Barrett**

Polluting Your Internal Environment

Homeostasis Is a Factor in Health

Numerous studies conducted around the world have shown a correlation between air pollution and adverse health effects, including premature death. But researchers have been stumped when they try to pin down a specific, consistent mechanism by which different air pollution mixtures affect people with varying susceptibilities living in a range of climates and settings. Researchers at the Johns Hopkins Bloomberg School of Public Health propose an experimental model to test whether a key factor in vulnerability to air pollution–related health effects might be the body's homeostatic capacity, or ability to compensate internally for external stressors and maintain a stable internal environment [***EHP* 110:61–65**]. In particular they are interested in changes in daily death rates.

The concept of homeostatic capacity has been around for decades, but there are few hard data on which indicators of homeostatic capacity are most important, what indicator levels portend a body's impending collapse, or what effects that outside stressors, such as pollutants or pronounced temperature swings, might have on individuals with reduced homeostatic capacity. It is known, however, that homeostatic capacity declines with age and illness, and that as this decline proceeds, the risk of death increases; the number of stressors capable of causing death goes up, and an individual stressor need not be as severe or intense to cause death.

The Johns Hopkins team evaluated a small number of short-lived inbred mice for two indicators of homeostatic capacity: deep-body temperature and electrocardiographic heart rate. After surgically implanting a radiotelemeter that continuously measured the two indicators, researchers monitored the daily routine of the animals.

Deep-body temperature in the mice fell progressively during the weeks before natural death. In one mouse described by the authors, deep-body temperature fell about 9°C over the last 11 weeks of life, and previously predictable temperature oscillations from day to night became erratic. In about the same time frame, heart rate slowed significantly.

Because such significant declines correlated closely with the demise of the animals, they may prove to be good markers of impending health problems in humans as well, says Robert Frank, one of the team leaders. Temperature and heart rate might be particularly good markers for humans because they frequently are measured noninvasively during routine medical examinations and are associated with many key body processes. Other homeostatic indicators that could be monitored include body weight, blood oxygen concentrations, blood viscosity, and extra- and intracellular pH and salinity.

The authors say that regardless of the indicator selected, limited homeostatic capacity in infants and young children, the elderly, and people suffering from a wide range of diseases may be a key element in making them more vulnerable to a wide range of environmental stressors, including not only air pollutants but many other stressors as well. "You're dealing with a population that's gotten to the point where any nonspecific threat can be the proximate cause of death," Frank says. "These are people for whom there is very little benefit to eliminating a single proximate cause of death because there is a whole universe of such causes."

Based on existing data, eliminating a few select air pollutant stressors would most likely be effective in reducing only acute health problems, which might protect less than one-tenth of 1% of the general population, according to the authors. The researchers' hypothesis and the model they are developing to test the hypothesis were intended to explain premature loss of life measured in days or weeks. They suggest, however, that the same model might be used to test whether chronic exposure to polluted air is associated with subtle long-term shifts in homeostatic capacity, which could contribute to chronic health problems and to greater shortening of the life span. **–Bob Weinhold**

East or West, Cardiac Arrest

Air Pollution Effects Similar Worldwide

In many ways, Hong Kong and London are worlds apart. But researchers from the University of Hong Kong and St. George's Hospital Medical School in London found that air pollution patterns correlated with similar health effects in both, bolstering the argument that air pollution contributes to short-term health problems, no matter where it occurs [***EHP* 110:67–77**]. The team's findings also add to the body of air pollution data by providing the first such information on a large subtropical city; other studies from around the world have been conducted almost entirely in temperate regions. However, the study still leaves questions unanswered, such as the specific mechanism by which these pollutants can cause harm. [See "Polluting Your Internal Environment," preceding page, for a theory about a possible mechanism.]

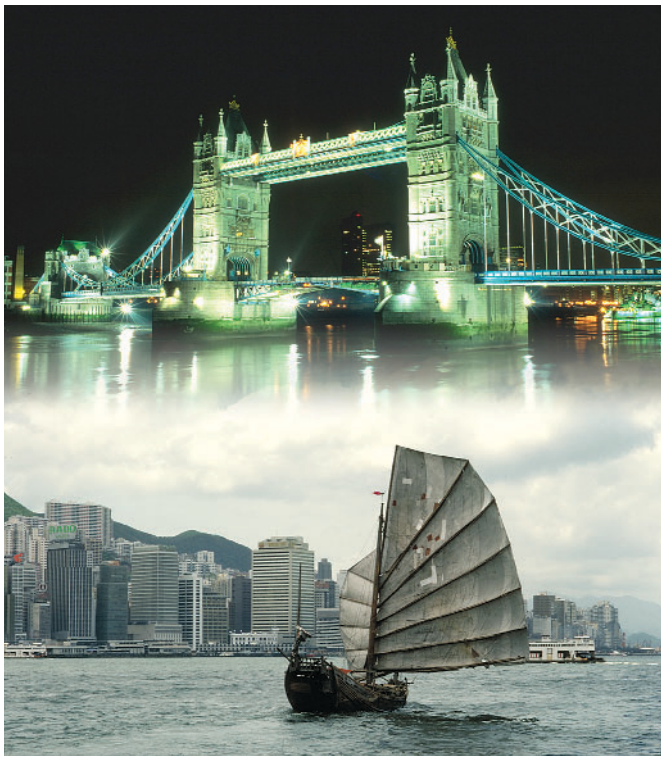
The research team reviewed data for daily emergency hospital admissions for the periods 1992–1994 in London and 1995–1997 in Hong Kong. Selected complaints included asthma in people aged 15–64, a range of respiratory diseases in people 65 and older, a range of cardiac diseases in people of all ages, and one subcategory of cardiac disease, ischemic heart disease, in people of all ages.

Hospital admissions for these complaints were compared with each city's concentrations of particulates 10 microns in diameter and smaller (PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and ozone within three days before admission. The cities had roughly similar concentrations of each pollutant, except for PM_{10}, which was markedly higher in Hong Kong.

To evaluate other factors that can play a part in hospital admissions, the team analyzed variables such as temperature, humidity, day of the week, season, thunderstorm occurrence, and influenza outbreaks. They also analyzed the differences in the two cities in climate, demographics, hospital admission patterns, and other factors that might affect the diseases under consideration. For instance, only 3% of Hong Kong females who are 15 or older smoke, compared to 27% of those in London, while 27–28% of the males in each city smoke.

Despite many differences between the populations, a small but significant number of hospital admissions in each city were generally associated with changes in concentrations of the pollutants studied. This was particularly true for cardiac problems overall, which tended to increase with elevated concentrations of PM_{10}, NO_2, and SO_2 in each city. Ischemic heart disease followed the same general pattern, although admission numbers were statistically insignificant in Hong Kong. Respiratory problems tended to increase with elevated concentrations of PM_{10}, NO_2, SO_2, and ozone in each city in many circumstances. Other common threads appeared. For instance, respiratory illnesses occurred most frequently in each city's time of lowest humidity (the cool season in Hong Kong and the warm season in London).

The researchers acknowledge there were several idiosyncrasies in the data. Cardiac admissions tended to decline as ozone concentrations rose in London, while Hong Kong's ozone concentrations, although very similar to London's, had no significant link with cardiac admissions. In some cases, the effects of a pollutant decreased after a second pollutant was added to an analysis. And there was no significant link in either city between asthma and the pollutants studied. The researchers believe, however, that the similarities outweigh the differences, and that the study therefore "strengthens the argument for the causality of air pollution associations with hospital admissions." **–Bob Weinhold**



The ties that bind. Cities may be as different as night and day in many respects, but the health effects of air pollution are similar worldwide.

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