Smoke-Filled Rooms

ETS Causes Menstrual Pain

Studies show that women who smoke are twice as likely to experience dysmenorrhea (painful menstruation) as nonsmokers, and smoking prolongs the symptoms of this condition. Fewer data are available on whether secondhand exposure to environmental tobacco smoke (ETS) at home or work also raises the risk for dysmenorrhea in nonsmoking women. In this issue, a team of environmental researchers headed by epidemiologist Changzhong Chen of the Harvard School of Public Health report that ETS exposure does indeed increase the occurrence of dysmenorrhea in nonsmoking women [***EHP* 108:1019–1022**]. Moreover, the more ETS a woman is exposed to daily, the higher her risk for dysmenorrhea.

Dysmenorrhea is a common gynecological problem that not only reduces quality of life but also accounts for significant medical costs and absenteeism from work. Previous studies that looked for an association between ETS and dysmenorrhea were not well controlled. For example, they included retrospective studies that relied on subject recall of symptoms, and involved older women (who have less dysmenorrhea) or women with prior dysmenorrhea (which may have been influenced by factors other than ETS).

Greenwell/EHP

Cramped quarters. Indoor exposure to environmental tobacco smoke may triple a woman's risk of menstrual pain.

The current, better-controlled study followed 165 newly wed, nonsmoking Chinese women through 625 menstrual cycles. The women's average age was 26 years, and they had no past history of dysmenorrhea. Chinese society offers a unique opportunity to study the consequences of ETS exposure because men smoke heavily, whereas women generally do not smoke. Because the women in the study were trying to conceive for the first time, none of them used birth control. This ruled out any impact of previous births or contraceptives, both of which have been implicated in contributing to dysmenorrhea.

Each woman kept a daily diary of menstrual symptoms and the number of cigarettes smoked indoors in her presence. The diaries were collected when a woman either became pregnant or a year had passed without conception.

Three-quarters of the women were exposed to ETS, largely via husbands who smoked around them. The incidence of dysmenorrhea—characterized as pain in the abdomen or lower back on two or more days of menstrual bleeding—varied with the level of ETS exposure, ranging from 9.7% in nonexposed women to as high as 16.9% in women with the highest level of exposure. Compared to women with no exposure to ETS, the researchers calculated that the risk of dysmenorrhea tripled in women with the highest ETS exposure, which corresponded to their husbands' smoking 2.6 or more cigarettes inside per day. In women receiving a middle level of exposure to ETS (0.8–2.5 cigarettes), the risk of dysmenorrhea was 2.5 times greater than in nonexposed women. The researchers estimate that for each day that two more cigarettes are smoked at home, the risk of dysmenorrhea climbs by 30%.

Refraining from smoking and limiting exposure to ETS could benefit the reproductive health of women, the researchers suggest. In future studies, they plan to evaluate whether exposure to ETS makes it more difficult for women to conceive. **–Carol Potera**

A Clearer Look at PM_{10}

Multiday Assessment Provides Better Data

Numerous studies over the years have indicated that airborne particulates can cause health problems. But each of those studies has had drawbacks. Many have looked at just one or two cities, limiting their ability to be extrapolated to other cities in different settings or with different pollutant sources. Others haven't considered factors such as a lag period for health impacts, effects from other pollutants, or a number of sociological influences such as poverty.

In this issue, Antonella Zanobetti and colleagues from the Environmental Epidemiology Program at the Harvard School of Public Health describe their multicity analysis of the relationship between levels of particulate matter smaller than 10 microns (PM_{10}) and hospital admissions for heart and lung disease [***EHP* 108:1071–1077**]. After examining more cities over a longer period with consideration of more confounding variables than any prior study, the team found that the adverse effects of PM_{10} were more accurately assessed by looking at particulate exposures on the day of hospitalization and the previous one or two days. In comparison, say the researchers, previous studies that looked at only a single day of exposure significantly underestimated the effects of particulate matter.

The team analyzed daily PM_{10} levels in 10 U.S. cities: New Haven, Pittsburgh, Canton, Detroit, Chicago, Minneapolis–St. Paul,

Birmingham, Colorado Springs, Spokane, and Seattle. They then reviewed Medicare records for the years 1986–1994 to pin down daily hospitalization counts for people over 65 suffering from cardiovascular disease, chronic obstructive pulmonary disease (COPD), or pneumonia. Finally, they used statistical methods to determine whether hospital admissions were influenced by the time elapsed since exposure to PM_{10} and whether sulfur dioxide, ozone, or carbon monoxide was present. They also factored in meteorologic variables such as humidity, temperature, and barometric pressure, and sociological variables such as poverty rates and minority status.

Although the results differed from city to city, the team concluded from the cumulative results that for each increase of 10 micrograms per cubic meter ($\mu g/m^3$) of PM_{10}, COPD hospitalization rates increased by 2.5%, pneumonia rates increased by 1.95%, and cardiovascular disease rates increased by 1.27%. These figures are especially striking when one considers that daily readings across the United States can fluctuate over a range of 10–150 (or more) $\mu g/m^3$. The results were not shifted significantly by most of the other factors analyzed.

The cities included in the study cover many regions of the United States, but the need to have long-term daily PM_{10} data and to eliminate influences such as windblown dust led to a high number of cities in the northeastern quadrant of the country. (The cities chosen had daily PM_{10} data for anywhere from 5.5 to 10 years.) The team concluded that the range of climates covered by the selected cities was sufficient to make the results applicable to other cities. That was confirmed when they added four more cities (including two more in the West) for a similar analysis as part of another study and saw no significant change in their findings.

But one puzzling result still has the researchers stumped. Lower PM_{10} levels, at one-third or less of the federal standard of 150 $\mu g/m^3$, boosted hospital admissions for all three diseases by 20% or more. While the reason is unknown, the team speculates that the rise could be triggered by factors such as the specific sources of particulate pollutants or changing behavior patterns such as leaving windows open more on presumably "clean" days. The authors state that it is crucial for public health impact assessment to know whether the associations are dominated by only a few high pollution days or whether they persist at the concentrations seen on most days. What they actually found is that the effects persist at common concentrations well below the current air quality standards, indicating that rising particulate levels lead to more hospitalizations for these three illnesses, and that the link shows up even at low levels. **–Bob Weinhold**

Childhood Tooth Decay

Is It Linked to Lead?

Both epidemiologic and animal studies suggest that childhood lead exposure is associated with dental caries, or tooth decay, but proof based on human studies remains elusive. Although lead poisoning occurs in all socioeconomic groups, urban minorities are particularly affected because they frequently live in older housing, which often contains lead-based paint and lead-contaminated dust. This population also suffers a high incidence of tooth decay, causing some researchers to consider whether lead exposure could be a factor. In a study published this month, a team at the University of Rochester School of Medicine and Dentistry in New York explores the possibility of a link but does not find a definitive answer [***EHP* 108:1099–1102**].

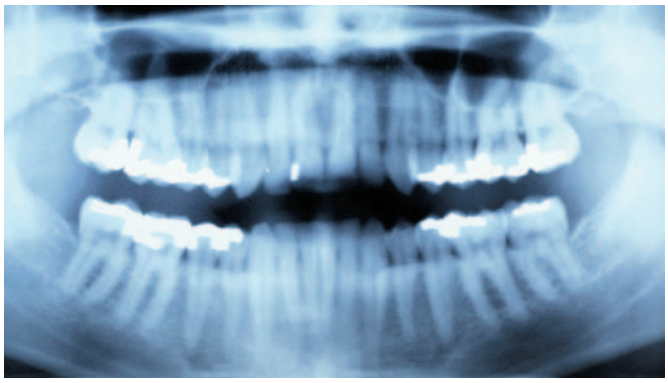
The team's objective was to see whether children with higher lead exposures at toddler age, when permanent teeth are developing, had more caries at school age than children with lower exposures. Headed by James R. Campbell of the Department of Pediatrics, the team drew on data collected by the Eastman Dental Center, which conducts a dental examination program for second- and fifth-graders in Rochester's public schools. Further data were provided by the Monroe County Health Department, which has maintained a database of blood lead concentrations in county children, including those in Rochester, since 1986.

By comparing data from both sources, the researchers identified a primary sample of 248 children who received dental examinations during the 1995–1996 and 1996–1997 school years and for whom information on blood lead concentrations at 18–37 months of age was available. Blood lead concentrations peak at about 2 years—up until that age, nearly everything a child touches goes into his mouth. The threshold for blood lead is established by the Centers for Disease Control and Prevention at 10 micrograms per deciliter (μg/dL), and the Rochester team used this level to define low and high lead exposure in their study population.

Because tooth decay has several causes, the team attempted to control for confounding factors through interviews with the students' parents. Parental input provided information on demographics, fluoride exposure, diet, oral hygiene, and medical history for a secondary sample of 154 students. Keeping in mind three hypotheses regarding lead's involvement in tooth decay that suggest that deciduous (baby) and permanent teeth may be affected differently by lead, the researchers also noted which type of teeth were affected by decay.

Blood lead concentrations in the primary sample had ranged between 0 and 46 μg/dL, with 34% of the children having had blood lead concentrations exceeding the defined threshold. In a simple comparison, 27% of the children with high blood lead concentrations had permanent tooth decay, as compared with 15% with low concentrations. Similarly, 59% of the children with high blood lead concentrations had deciduous tooth decay, compared with 46% with low concentrations. However, the children with high blood lead concentrations were older and had more permanent tooth surfaces—not only had they had more time to develop caries in permanent teeth, they simply had had more time to get permanent teeth. When these variables were included alongside information supplied by parents in more sophisticated statistical analyses, no valid, significant association was found.

Based on these analyses, the researchers conclude that tooth decay is not strongly linked to lead exposure, but they cannot rule out that a weak association exists. The authors also speculate that the difference between the two groups may not be significant because none of the children are so old as to see much permanent tooth decay. Significance might be better determined when these children are older. **–Julia R. Barrett**



PhotoDisc

Lead link largely lacking. Although earlier studies suggested a connection between childhood lead exposure and cavities, findings from a Rochester, New York–based study indicate the two are not strongly linked.