SMOKING AND SECONDHAND SMOKE

Global Estimate of SHS Burden

Call it what you will—passive smoking, environmental tobacco smoke, or secondhand smoke (SHS)—worldwide, exposure to the emissions from smokers’ cigarettes caused the premature death of an estimated 603,000 people in 2004, according to a team of academic and World Health Organization (WHO) researchers. This first global assessment of the burden of SHS was led by Mattias Öberg of the Karolinska Institute and sponsored by the Swedish National Board of Health and Welfare and Bloomberg Philanthropies.

SHS was first confirmed in the mid-1980s to cause adverse health effects. The associated effects now include heart disease, lung cancer, worsening of asthma, sudden infant death syndrome, and more. But until now, data on deaths and disease among nonsmokers have not been compiled at the global scale.

"There have been estimates made before for specific countries on the impact of SHS," says epidemiologist Jonathan Samet of the University of Southern California, who did not participate in the new study. "The new paper by Öberg and colleagues is important for putting together a global picture."

The researchers searched the scientific literature, public health reports, and government databases for reliable data on smoking according to age and sex. Where needed data did not exist, they created models to extrapolate from well-studied regions to countries with low data availability. One main data source for exposure among children was the Global Youth Tobacco Survey, cosponsored by the U.S. Centers for Disease Control and Prevention in more than 120 countries. The school-based survey is administered annually to 13- to 15-year-olds to assess children’s use of tobacco products and exposure to SHS.

Öberg says the team took a conservative approach in its assessment. For example, the researchers chose not to include deaths or diseases without clear and strong evidence for a causal relationship to SHS exposure (stroke was one such disease). They excluded diseases that have been causally linked to SHS if strong and comparable international health statistics were unavailable (one such example was sudden infant death syndrome). The team also did not include premenopausal breast cancer, as the relationship between this disease and SHS remains controversial in the scientific community.

“It’s difficult to find really good data [for some of these outcomes],” Öberg says. Moreover, he adds, it’s hard to relate some effects directly to SHS exposure. For instance, despite strong suggestive evidence of links between stroke and chronic

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**Failure is not fatal, but failure to change might be.**
John R. Wooden, basketball coach (1910–2010)

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**SHS around the World**

| Proportion of deaths attributed to SHS* |
|----------------------------------------|
| Women 47% |
| Children 28% |
| Men 26% |

| Proportion of DALYs (ie, disease burden)* |
|-----------------------------------------|
| Children 61% |
| Women 24% |
| Men 16% |

| Percentage of nonsmokers who are exposed to SHS |
|-----------------------------------------------|
| Children 40% |
| Women 35% |
| Men 33% |

*Percentages are rounded and do not total 100.
obstructive pulmonary disease (COPD) and SHS, these links have not been confirmed by strong epidemiologic meta-analyses.1

Finding high-quality peer-reviewed SHS exposure data also has proved difficult; far more data are available on the number of active smokers, Öberg says. He adds that, although smokers themselves are likely affected by passive smoking, they were not included in the team’s main assessment. If they had been included, the estimated morality rate would have increased by about 30%; ex-smokers were included, and without them, the total number of deaths would decrease by 17%.1

In the end the team estimated the global proportion of people exposed to SHS in various settings, as of 2004, at 40% of all children (defined as age 0–14 years), 33% of nonsmoking men, and 35% of nonsmoking women around the world. But those proportions varied by region according to smoking habits, rural versus urban populations, country regulations, and other factors. For example, in the region encompassing Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, the Russian Federation, and Ukraine, around two-thirds of nonsmokers in all age and sex groups were estimated to be exposed. In southern and northeast Africa, only 12% of children and even fewer men and women were estimated to be exposed.1

The burden of morbidity from SHS exposure, as measured by disability-adjusted life years (DALYs), also varied by region, with higher estimates for low-income countries in Southeast Asia and the eastern Mediterranean region compared with Europe. Asthma and ischemic heart disease accounted for the most disease among adults, and lower respiratory infections were the most common outcome among children.1

Most striking, children under age 5 years bore the brunt of respiratory infections in poorer countries, where malnutrition or inadequate health care also may lead to higher disease and mortality rates in children with other health problems that are exacerbated by SHS exposure. The team calculated that children overall experienced an estimated 61% of the disease burden from SHS.1

“Children remain exposed in the home,” even in countries with legislation that removes smoking from public places, says Heather Wipfli, a University of Southern California policy expert who, with Samet, coauthored a comment in The Lancet1 on the new research. But Wipfli considers SHS exposure largely a women’s issue: only about 10% of women in the world smoke, she explains, but of the 603,000 SHS-related deaths of nonsmokers estimated in 2004, 47% were among women (compared with 26% among men and 28% among children).3

In one bit of good news, Wipfli says China’s percentage of women smokers has remained low, despite concerns among the public health community that Chinese women would be a market targeted by the tobacco industry—traditionally, women there have not smoked, something that growing wealth and commercialism might have changed. Still, Europe and Asia, and particularly lower-income countries in those regions and countries where almost all parents smoke at home, have extremely high SHS exposures for nonsmokers in general, Wipfli says. She and Samet urge the full implementation of the various components of the WHO Framework Convention on Tobacco Control (an international treaty that works on both supply and demand for tobacco) and associated policy and educational programs.

The WHO will report next year on how many nations have passed bans on smoking in public places, including work sites and restaurants, says Armando Peruga, a program manager with the WHO Tobacco Free Initiative and coauthor of the Lancet report.1 Peruga says the team needs to do additional work to refine their estimates and gather more data for individual countries, particularly those lacking complete reporting data on smoking; he hopes the team may have these calculations completed in a year or so.

Meanwhile, the new study is “an impressive effort at producing an estimate of the global effects of secondhand smoke,” says Bart Ostro, a research scientist at the Centre for Research in Environmental Epidemiology (CREAL) in Barcelona, on temporary leave from the California Environmental Protection Agency. Ostro comments that the epidemiologic method the team used to reach its conclusions is well established and that they “utilized a lot of studies that have been heavily peer-reviewed in the past,” including the U.S. Surgeon General’s seminal 2006 report and a similar 2005 report from the California Environmental Protection Agency.

The researchers’ sensitivity analysis “shows that no matter what assumptions you use, the impact on children and adults is still of great public health significance,” Ostro adds.

Despite remaining gaps in the data, the estimate is “a policy-relevant number and one that should motivate action,” says Samet. “These exercises are intended to provide general guidance and an understanding of the magnitude of the disease burden . . . and how much could be avoided by preventive strategies.”

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NEUROLOGIC HEALTH

Acrolein and Neuro Disorders

Neurologic disorders are among the leading causes of death and illness in the United States. Their causes are poorly understood, but one of the emerging suspected culprits is the substance acrolein, which tends to be significantly elevated in the brains of spinal cords of people who have Alzheimer disease, Parkinson disease, amyotrophic lateral sclerosis (ALS), and other neurologic disorders.1,3,4 A new study adds multiple sclerosis (MS) to the list of disorders potentially affected by this substance.5

Acrolein is produced naturally in the body as a by-product of membrane lipid peroxidation. It also occurs in combustion by-products such as vehicle exhaust, industrial emissions, oil- and coal-fired power plant emissions, cooking fumes, and the smoke from burning cigarettes, wood, and plastics. It’s used as a biocide and to manufacture other chemicals and products such as chemical weapons. The U.S. Environmental Protection Agency (EPA) has determined the ubiquitous pollutant is a major source of respiratory damage.5 But information on the neurologic effects of environmental acrolein is scant.6

In the new study, Riyi Shi of Purdue University and colleagues injected mice with substances known to induce experimental autoimmune encephalomyelitis, an animal model for MS.3 Within 2 weeks acrolein–lysine adduct levels in the spinal cord began to rise, peaking at 65% higher than in controls at about day 20. At the same time, the mice began to display significant muscle control problems. Treatment with the acrolein-scavenging substance hydralazine reduced those problems to a great extent although not significant degree. The researchers also detected significant mitigation of damage to the myelin sheath by hydralazine.

Shi and colleagues say their study provides the first evidence that endogenous acrolein plays a key role in MS. Shi says it’s plausible that environmental acrolein can act in the same general way. “There’s no reason not to believe that the same type of damage could occur.”

Richard LoPachin, a neurochemist and director of research at Montefiore Medical Center in New York, partially agrees. “Because acrolein is highly reactive with proteins at the site of exposure, it has limited distribution in the body and, therefore, limited access to the brain,” he says. But acrolein is just one of many type-2 alkenes, a large family of environmental and food contaminants that includes acrylamide, methyl vinyl ketone, methyl acrylate, and 4-hydroxyenononal. LoPachin says type-2 alkenes share a common mechanism of action at nerve terminals in the brain, and he thinks the combined effects of these substances could contribute to some neurologic disorders.

Robert Kavlock, director of the EPA National Center for Computational Toxicology, says acrolein’s physical properties make it difficult to assess the compound using the agency’s ToxCast™ high-throughput chemical screening program using currently available technology. But pinning down the causes of these neurologic disorders could help millions of people. In the United States alone, about 5.3 million people have Alzheimer disease, about 1.5 million have Parkinson disease, about 400,000 have MS, and about 30,000 have ALS.9

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A new study has reopened an old debate over the potential health risks that fish farms pose to wild fish populations, concluding that “productivity of wild salmon is not negatively affected by either farm lice numbers or farm fish production.” The paper by Gary Marty, a research associate at the University of California, Davis, and fish pathologist with the British Columbia Ministry of Agriculture, and two colleagues pooled data from fish farms in western Canada with data first presented in 2007 by Krkošek et al. The earlier paper concluded that infestations of ectoparasitic sea lice from salmon farms were driving a decline in wild pink salmon (Oncorhynchus gorbuscha) populations in British Columbia’s Broughton Archipelago and that extinction would occur if the infestations continued.

Sea lice pose no direct threat to humans who consume the fish; furthermore they’re removed during the harvesting process. But the new study contributes to the larger ongoing discussion of whether a large-scale aquaculture industry can be sustainable in terms of human and ecosystem health.

Marty says earlier analyses omitted relevant factors from a medical perspective—that is, a diagnostic approach to fish health and epidemiologic factors, rather than a model-driven analysis. For the new study, he and his colleagues obtained proprietary monthly sea lice data from fish farms in the region, giving what they call a fuller picture of the salmon decline in 2001–2002 than the previous analyses, which relied on sea lice counts from wild fish only.

According to Marty, the new analysis suggests pink salmon populations are within a natural pattern of fluctuation. “Our paper estimates that sea lice numbers on farmed fish were greater in 2000 than in 2001, and the wild pink salmon exposed to those sea lice in 2000 came back in record high numbers in 2001,” he says.

Martin Krkošek, lead author of the 2007 paper and a lecturer in zoology at the University of Otago in Dunedin, New Zealand, says the new analysis was limited by the omission of data from the affected region prior to infestations as well as nearby regions where there are no salmon farms. Analyses that used the spatial and temporal controls from a larger picture of salmon abundance in the Broughton Archipelago, Krkošek says, “have found effects of sea lice.”

Jeff Silverstein, national aquaculture program leader for the U.S. Department of Agriculture, notes that although the earlier paper suggested sea lice from salmon farms caused wild salmon declines, “The recent study has managed to show that the correlations don’t appear to be causative.” Ian Bricknell, director of the Aquaculture Research Institute at the University of Maine in Orono, adds, “The epidemiological approach . . . is a much more effective way of analyzing this data.” Bricknell says Marty et al. “looked at many more variables than just lice and salmon (as was done earlier) and have backed it up by testing their model with biological data.”

Krkošek agrees other factors may have contributed to the 2002 decline but strongly disagrees with the conclusion that sea lice do not negatively affect wild salmon productivity. “While one may speculate about the possibility of other factors that could have contributed, it is known—not speculated—that lice numbers were very high on those fish,” he says.

Marty insists the study’s most important impact lies in showing how medical analysis brings a broader perspective to fish population studies. “I want people to focus on what is actually causing salmon populations to go up and down,” he says. “We should still look at sea lice but include other factors as well.”

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