The Importance of Extinguishing Secondhand Smoke

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Government policies have enormous influence on the health of nations. Arguably, this is illustrated most vividly with tobacco control. However, smoking continues to be a global problem and the major cause of preventable death. The countries with the highest per-capita smoking prevalence rates include (alphabetically) Bangladesh (20.9% of adults), Brazil (16.2% of adults), China (31.4% of adults), Germany (27.2% of adults), Indonesia (34.5% of adults), Japan (43.3% of men and 12% of women), the Russian Federation (60.4% of men, 15.5% of women), Turkey (34.6%), and the United States (23.2%).

Prevalence rates among younger people vary, but in the United States, 18.4% of youths still smoke.

Another approach to assessing population health interventions is to combine evidence from observational epidemiology and from clinical trials using modeling techniques. This can allow projection of the likely effect of a health intervention to a population that may differ from those in which the original studies were undertaken. However, epidemiological models often rely on assumptions that cannot be tested and on simplifications of characteristics of the intervention and the disease process. They also frequently incorporate a large number of estimated parameters. This can lead to a spurious close fit between the model results and the data on which the model parameters were based.

Lightwood and Glantz used a combination of these approaches. Their meta-analysis and the model draw their parameter estimates from different, independent data sources. Hence, the demonstrated consistency between the results allows greater confidence in the results of each approach. This is important because policy makers are informed by such data in balancing costs and effectiveness and in prioritizing between different population health interventions.

A further feature of epidemiological models is that they can highlight important gaps in the data available to inform health policies and strategies. Typically, studies of the risks of secondhand smoke have relied on self-reporting. As discussed by the authors, individual exposures, which are presently measured by levels of cotinine, a stable metabolite of nicotine, were not generally available. However, all the scenarios modeled by Lightwood and Glantz that showed a close agreement with the results of the meta-analysis were based on parameters derived from the study with cotinine levels available.

There are no “control” data to enable comparison with similar contemporary communities without smoke-free ordinances. However, this was presented in some of the individual studies to support the positive impact of legislation. Longer-term data could also have permitted examination of the possible effect of other factors that might have had an impact on secular changes, such as the use of important therapies, including 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors.

The outcome examined in the meta-analysis was hospitalization for acute myocardial infarction. Coronary plaque rupture and thrombosis are critical to this. Reduction in out-of-hospital deaths has also been demonstrated, and those studies with a broader range of end points have shown similar effects to those on incident myocardial infarction.

The term “secondhand smoke” captures the involuntary nature of exposure. This emanates from both “sidestream”
smoke from a smoldering cigarette and “mainstream” smoke exhaled by a smoker. Mainstream smoke is the more important. Secondhand smoking is a leading cause of preventable death, and the 2006 US Surgeon General’s report was entirely devoted to it. The report concluded that the evidence was sufficient to support immediate adverse effects on the cardiovascular system and, importantly, to allow deduction of a causal relationship between secondhand smoke and coronary heart disease (CHD) morbidity and mortality. It was also concluded that the evidence was suggestive of, but not sufficient to infer, a causal relationship with subclinical vascular disease, particularly carotid intimal-medial thickness and with the increased risk of stroke. Although exposure to secondhand smoke has declined over recent decades, the report estimated that 60% of American nonsmokers had biological evidence of exposure to secondhand smoke. The National Health and Nutrition Examination Survey measured cotinine levels among nonsmokers aged 20 years and over in 1999 to 2000, and it was found that 46% living in jurisdictions with smoking legislation had detectable cotinine levels (≥0.05 ng/mL), compared with 13% in jurisdictions without such laws.

Secondhand smoke has been estimated to cause a 25% to 30% increase in risk of CHD events. Exposure has often been estimated by the number of cigarettes smoked daily by a spouse or partner. Even for those with low to moderate exposure (1 to 14 or 1 to 19 cigarettes daily), the relative risk was 1.16 in comparison with those without such exposure. The evidence supporting these associations has been derived from both cohort and case-control studies, which have ranged in follow-up to 20 years, with different exposure measures and variability in the other factors that were controlled for. The association was slightly stronger in the overview of case-control studies, perhaps partly reflecting the bias associated with retrospective recall. There may be other biases. Although exposures may be misclassified, failure to account for background secondhand smoke and the use of only whether or not a spouse of a nonsmoker was a smoker would lead to underestimation of the strength of the association with outcomes. Indeed, estimates of the effects were 2-fold greater when based on cotinine levels, which capture exposure in the range of environments. Whincup et al demonstrated a dose-response relationship between serum cotinine and CHD events in a 20-year prospective study and found that risk was increased by 57% in individuals in the highest quartile of cotinine levels. The effect of any confounding factors such as poorer diet appears to be relatively small.

The impact of secondhand smoke on the risk of CHD is larger than anticipated, based on the 80% increase in relative risk associated with active smoking and the fact that the measured exposure to tobacco smoke to which nonsmokers are exposed is only about 1% of that from smoking 20 cigarettes daily. However, such large effects are biologically plausible and are consistent with nonlinear effects of tobacco exposure at low doses, including important actions on platelet and endothelial function. The effects on platelet and endothelial function, arterial stiffness, oxidative stress, and inflammatory markers are approximately 80% to 100% of those associated with active smoking. Other deleterious effects of secondhand smoking include those on matrix metalloproteinases, which might contribute to instability and rupture of atherosclerotic plaques; high-density lipoprotein cholesterol, and mitochondrial energy utilization.

The effects not only are large but occur rapidly. An important early study showed that platelet activation and aggregation and endothelial cell damage occurred in nonsmokers within 20 minutes of exposure to secondhand smoke, with no further activation among active smokers. Similarly, 30 minutes of breathing secondhand smoke caused endothelial dysfunction to a degree similar to that in active smokers. The effects of endothelial function may be slow to recover after long-term higher levels of exposure to secondhand smoking ends. However, there will be a rapid decrease in platelet aggregation, a key factor in acute coronary syndromes.

Exposure to secondhand smoke can occur in several environments, particularly in the home and workplace but also in restaurants, bars, gambling venues, and automobiles. It is not shared equally. Women are less often active smokers but sustain most of the burden of secondhand smoking. Effects may be particularly hazardous for children. They have smaller airways, breathe more quickly, and take in 3 to 4 times as much air and, potentially, secondhand smoke relative to their body weights as adults. The association with subclinical atherosclerosis in children is unproven. However, a relationship with carotid intimal-medial thickness has been shown in the Atherosclerosis Risk in Communities Study and children have many years to manifest a disease such as atherosclerosis, which has a long latency period. Disturbingly, the declines in cotinine levels in the time intervals between 1988 to 1991 and 1999 to 2002 have been less among children than adults. In 1999 to 2002, 59.6% of American children aged 3 to 11 years had cotinine levels ≥0.05 ng/mL, and their median cotinine concentration was 0.09 ng/mL, compared with 0.035 ng/mL in older adults. Nonsmokers in lower socioeconomic groups are also vulnerable because of higher active smoking rates and other environmental conditions to which they are exposed. In indigenous populations with high smoking prevalence, reduction in smoking, which would extend to secondhand smoke, may be the single most important short-term action to improve their life expectancy.

The World Health Organisation Framework Convention on Tobacco Control had 166 parties as of July 2009. Protection from secondhand smoke is among the 6 most important and effective policies outlined in the Framework, together with raising taxes and prices, health warnings, QUIT programs, banning of advertising and sponsorship, and careful surveillance of the tobacco epidemic and prevention policies. This should include cotinine or alternative biomarker data if possible. The present meta-analysis strengthens the evidence base to support laws promoting smoke-free environments. The effects are significant and, although immediate, increase with time. It is also important that the very high rates of CHD magnify the public health impact of secondhand smoke to underscore the importance of legislation. The California Environmental Protection Agency estimated that in the United States alone, in 2005 secondhand smoke resulted in...
46,000 deaths due to CHD, compared with 3400 due to lung cancer in adult nonsmokers and 430 deaths related to sudden infant death syndrome.16

Such regulation can also have an impact on active smoking. A systematic review of 26 studies showed that smoke-free workplaces reduced smoking prevalence by 3.8% and the amount smoked by 3.1 cigarettes daily in those continuing to smoke, together constituting a 29% decrease in total cigarette consumption.19 One of the studies included in the meta-analysis2 found that acute coronary syndrome admissions were decreased in smokers as well as nonsmokers.7 Furthermore, rather than having a negative impact on businesses, smoking bans can increase patronage of restaurants and drinking venues.

Clinicians should advise their patients to avoid public places that permit smoking, and families should be counseled not to smoke at home or in a vehicle with patients. Healthcare professionals can also be powerful advocates, and research such as that described in this issue2 strengthens the case for government action.

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References

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