

Even a Little Secondhand Smoke Is Dangerous

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AS MORE AND MORE NONSMOKERS HAVE COME TO UNDERSTAND the dangers associated with breathing secondhand smoke,^{1,2} the number of communities enacting ordinances requiring smoke-free workplaces and public places has increased rapidly. As of May 2001, hundreds of communities had enacted laws requiring smoke-free workplaces, smoke-free restaurants, and smoke-free bars. California requires all workplaces, including restaurants and bars, to be smoke-free.^{3,4} The theme for the World Health Organization's World No Tobacco Day in 2001 was "clean indoor air" and communities throughout the world are beginning to clear the air of secondhand smoke. Not only do the laws protect nonsmokers from the toxins in secondhand smoke, but they also create an environment that helps smokers cut down or stop smoking.⁵

The tobacco industry's efforts to slow the spread of smoke-free environments has included a systematic effort to attempt to undermine the scientific evidence that passive smoking causes disease.⁶⁻⁸ One common theme is that the dose of toxins a nonsmoker inhales is tiny compared with the dose the smoker receives, implying that the risks are trivial or nonexistent. Such statements are based on measuring the delivered dose of 1 or more of the 4000 chemicals in secondhand smoke. The problem with such calculations is they can be manipulated by selecting the particular constituent of smoke to be the one that has low absorption or rapid clearance.¹ The real measure of effect should not be the dose of one chemical or another, but rather the biological effect of breathing the secondhand smoke.

The article by Otsuka and colleagues⁹ in this issue of THE JOURNAL adds substantially to the case that short-term passive smoking adversely affects endothelial function in ways that immediately compromise the cardiovascular system.¹⁰ The investigators demonstrated that, in healthy young volunteers, just 30 minutes of exposure to secondhand smoke compromised the endothelial function in coronary arteries of nonsmokers in a way that made the endothelial re-

sponse of nonsmokers indistinguishable from that of habitual smokers.

The investigators measured blood pressure, heart rate, and coronary flow velocity reserve before and after administering adenosine triphosphate using transthoracic Doppler echocardiography of the left anterior descending coronary artery. This innovative noninvasive approach to measuring coronary endothelial function appears to be ideal in these individuals, who have no evidence of coronary disease. Significantly, these substantial changes in endothelial function were not associated with changes in heart rate or blood pressure.

Endothelial dysfunction may be at the heart of the development of atherosclerosis. Normal endothelial cells promote vasodilation and inhibit atherosclerosis and thrombosis, in part because of the release of nitric oxide.¹¹ Dysfunctional cells, on the other hand, contribute to vasoconstriction, atherogenesis, and thrombosis. Risk factors contribute individually to endothelial dysfunction and appear to be additive. One possible unifying hypothesis for the effects of risk factors is that they increase oxidative stress that mediates these effects.¹² Thus, reduction of risk factors improves endothelial function and reduces clinical coronary events. For example, in patients with hyperlipidemia, lipid lowering improves endothelial function both acutely¹³ and chronically.¹⁴

The findings of Otsuka et al⁹ are important not only because they illustrate the importance of preventing nonsmokers from any exposure to secondhand smoke, but also because they help to explain the relatively large risk of death and other cardiac events associated with passive smoking compared with active smoking. Passive smoking increases the risk of cardiac death or morbidity about 30%¹⁵⁻²¹ compared with a doubling to quadrupling of risk associated with active smoking. Thus, the effect of passive smoking is as high as one third the effect of active smoking even though the dose of at least some of the constituents is much less than what the smoker inhales.¹

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The first evidence that nonsmokers were sensitive to a component of tobacco smoke came from studies showing that short-term (30-minute) exposure to secondhand smoke activated nonsmokers' platelets to nearly the extent that they were activated in smokers^{22,23} and that passive smoking increased the presence of endothelial cell morbidity in the blood.²³ These immediate effects on platelets probably act synergistically with the effects on endothelial function. The platelet effects convinced epidemiologists that the dose-response curve for cardiovascular effects associated with tobacco smoke exposure was not linear, but exhibited substantial effects at relatively low doses (at least compared with an active smoker; the doses are high when measured against other environmental toxins) that a passive smoker receives.^{18,20} In addition, animal studies demonstrated that exposure to the secondhand smoke from a single cigarette daily induced atherosclerotic changes.²⁴ The fact that passive smoking does not induce additional effects in smokers^{9,22} suggests that the underlying biochemical and cellular processes saturate at the doses involuntary smokers experience.

While most people think of cancer when they think of active and passive smoking, it is important to emphasize that heart disease is also an important consequence of tobacco smoke exposure. This situation is particularly true for passive smoking; heart disease accounts for about 37 000 of the estimated 53 000 annual deaths attributed to involuntary smoking in the United States.¹⁸ Another important difference between the effects of smoking on risk of cancer compared with risk of heart disease is that the effects on cancer develop and resolve slowly (over a period of years) whereas the effects of smoking on the cardiovascular system occur rapidly.

The findings of the study by Otsuka et al⁹ add to the evidence suggesting that everyone should be protected from even short-term exposure to the toxins in secondhand smoke. Communities should continue to require that workplaces, including restaurants and bars, be smoke-free and mount public education campaigns to encourage smoke-free homes. Not only will everyone breathe better,²⁵ but they will also have healthier hearts.

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