Title
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Permalink
https://escholarship.org/uc/item/7t75f9k7

Journal
Chest, 148(1)

ISSN
0012-3692

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Publication Date
2015-01-01

DOI
10.1378/chest.15-0340

Peer reviewed
The Civil Liberty of Smoking

Some political figures have made a point recently about the importance of civil liberties. These individuals point to the importance of freedom and discourage government interference in personal matters. Despite the known health risks, 20% of the US population smokes conventional tobacco cigarettes. Many smokers argue that they enjoy the habit and do not believe that others should have the right to regulate their behavior. Smokers represent a financial burden on the health-care system, but pay considerable consumption tax during their lifetime and, thus, their economic impact could be debated.

The argument changed from one of personal freedom to include issues of public health when the impact of secondhand smoke (SHS) was recognized. No longer were smokers simply affecting themselves, but also their children, spouses, and other innocent bystanders. Indeed, roughly 40% of children worldwide are regularly exposed to SHS, and 600,000 people are predicted to die annually from SHS. Some data also suggest that smoke exposure may have transgenerational consequences such that an individual’s adult health may be affected by prior exposures experienced by one’s mother or grandmother.

In addition to cigarette smoking, other behaviors have been debated. Vaccinations in children are being provided inconsistently, in part due to influential voices who advocate against them. Some of these individuals point to potential risk of vaccines despite no compelling data and ignore the transformative benefits that vaccines confer. Clearly, the issue of withholding vaccinations from children is not just one of civil liberties but of potentially endangering the population at large.

Similarly, seat-belt legislation was resisted by many based on civil liberties, but some measures have produced widespread benefits for society. Sleep deprivation was also considered a matter of individual choice, but once data showed a risk of motor vehicle accidents and other occupational risks, the matter no longer became limited to the individual, because the wellbeing of the community needed to be considered. Finally, required hand washing for those in the food services industry, for the simple and very clear reason of preventing transmission of diseases such as hepatitis A and pathogenic Escherichia coli to the community, is being challenged as impinging on civil liberties.

In this issue of CHEST, Adams et al provide a sophisticated analysis of the mechanisms underlying cardiovascular risk of SHS. Using an elegant technique developed by the Jelic laboratory, the authors isolated endothelial cells from venous scrapings and studied the inflammatory and vasodilatory properties of these cells. The authors argue that mechanisms underlying cardiovascular risk in people exposed to SHS are poorly understood and hypothesize that the vascular biology of endothelial cells from people exposed to SHS is affected by the exposure. Indeed, the authors observed that expression of endothelial nitric oxide synthase (eNOS) and phospho-eNOS was decreased similarly in cigarette smokers and individuals exposed to SHS. Additionally, the authors observed that expression of endothelial nitric oxide synthase (eNOS) and phospho-eNOS was decreased similarly in cigarette smokers and individuals exposed to SHS. Also, levels of nuclear factor-kB, an important inflammatory transcription factor, were increased similarly in cigarette smokers and individuals exposed to SHS. The findings were compared with endothelial cells from nonsmokers who did not display these same abnormalities. To test the clinical relevance of the observed abnormalities, the authors also performed flow-mediated dilation of the brachial artery in response to ischemic stimuli, and observed a similar degree of impairment in smokers as compared with individuals exposed to SHS. Clearly, both smokers and innocent bystanders may be affected by this toxic inhalation. The data provide compelling rationale for limiting exposure of irritants to all involved.
Despite these important results, many questions remain. First, of the thousands of toxic chemicals in cigarette smoke, what is the molecular mechanism of reduced eNOS and increased nuclear factor-κB expression in smokers and individuals exposed to SHS? Are there therapeutic targets that could be used to minimize cardiovascular risk among exposed individuals? Second, do similar toxicities exist for other forms of smoking (e.g., electronic cigarettes [e-cigarettes]) also affect vascular risk for the willing recipient as well as innocent bystanders? Recent data suggest that e-cigarettes may have unique toxicities to airway cells and may affect bacterial pathogenesis, suggesting the need for independent study of each injurious agent. Third, what is the difference in chemical delivery to the lungs of various different exposed individuals? Sidestream smoke may be more toxic than mainstream smoke because it is unfiltered, and it generates more toxins when it mixes with room air for 0.5 to 2 h. Moreover, carbon monoxide (CO) concentrations in the exhaled breath of cigarette smokers are higher compared with SHS-exposed individuals; in theory, inhaled CO may have potential benefits in very low concentrations. Inspired CO is entering clinical trials in various inflammatory lung diseases, due to its antiinflammatory, antiapoptotic, antiproliferative, and vasodilatory effects, although the clinical benefits remain unclear. We congratulate Adams et al on the provocative findings and for informing public policy with a strong scientific foundation. Clearly, any discussion around the civil liberty of smoking cigarettes is complex when the broader population is considered.

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