Given our strong agreement with Gwinn et al.’s (1) position on the importance of integrating studies of genetic and environmental contributors to health risk, we were intrigued by their citation of our own commentary (2) as a counterargument. They appear to have misinterpreted our position as a global objection to the study of genetic variants associated with smoking and its complications. To the contrary, we have noted many potential benefits from research on the genetics of smoking, including a better understanding of substance abuse and insights into the pathophysiology of diseases associated with smoking (2, 3). This research has the potential to generate new ideas about the treatment of cancer, heart disease, and the many other adverse health consequences of smoking.

Our skepticism is limited to the use of genetics as a tool for smoking cessation; we question the value of research focused on this goal. As Gwinn et al. note (1), smoking is a continuing health problem in the United States and a growing problem internationally. While genetic research may shed light on the mechanisms by which smoking leads to many diseases, the appropriate public policies for prevention of those diseases are clear: Increase the reach of smoke-free environments, block tobacco advertising (particularly advertising directed at children and young adults), and assure universal access to proven treatments for smoking cessation.

The concept of genetics as a way to individualize smoking cessation resonates with popular presentations of genetics but is questionable on 2 counts. The science does not support it: Several trials have failed to show significant effects, and decision modeling suggests that success is unlikely under realistic projections (4, 5). Equally important, the demographics of smoking point to a different, more productive approach: Smokers in the United States are disproportionately from socially disadvantaged populations, with limited access to smoke-free environments and smoking cessation counseling and treatments. The problem is not that we lack the ability to help people stop smoking—dramatic drops in smoking rates among affluent and educated segments of our population testify to the success of current efforts—it is that we are not doing enough to reach less advantaged groups (6).

We agree with Gwinn et al. regarding the strong value of using studies of genetics and smoking to investigate larger questions about gene-environment interaction and to improve methods for complex data integration. Such knowledge would be extremely helpful as we attempt to address, for example, the equally complex and widespread problem of air pollution health effects, for which (in contrast to smoking) there is no single exposure that can be targeted for removal as an unnecessary societal scourge. This contrast points to the importance of the broader epidemiologic context in defining the research agenda.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

REFERENCES


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DOI: 10.1093/aje/kwp397; Advance Access publication November 25, 2009