Reducing Carcinogen Levels in Cigarette Smoke

To the Editors: Grizt et al. published an editorial calling for increased research efforts to reduce the risk of lung cancer in smokers (1). They emphasized research on smoking behavior and preventing the development of cancer but do not mention safer cigarettes, which offer the most immediate promise for reducing the risk of lung cancer. Most smoking-related lung cancer is almost certainly induced, or at least initiated, by chemical carcinogens in cigarette smoke, especially the tobacco-specific nitrosamines (TSNA) 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and polycyclic aromatic hydrocarbons such as benzo(a)pyrene (2, 3). Cigarettes from different countries show wide variations in TSNA content (e.g., those from the United States had TSNA levels seven times higher than those from Bangladesh; ref. 4). Cigarette TSNA levels were strongly correlated with those for nitrate (3, 5), suggesting that TSNA arise from the reaction of amines with nitrite derived from nitrate in the tobacco. TSNA level was not correlated with the tar content of mainstream cigarette smoke (6). Most of the evidence indicates that TSNA in cigarette smoke arise by transfer from TSNA in cigarettes and that lowering TSNA levels in cigarettes would reduce the exposure of smokers to TSNA (4, 7-9).

The mean TSNA content of Swedish snuff (“ethan snus”) was 2.8 μg/g compared with 33 μg/g for all U.S. snuff brands (10). Apparently, the low TSNA level in snus occurs mainly because snus is manufactured by heating freshly picked tobacco leaves with steam, which produces relatively sterile tobacco, whereas U.S. cigarette and snuff tobacco is stored for several weeks in curing barns (10). Storage leads to proliferation of bacteria that reduce nitrate to nitrite, which produces nitrosamines by bacteria-catalyzed or chemical nitrosation (3, 11). Snus is also kept refrigerated in stores selling the product (10). I suggest that similar treatments could be adopted for the manufacture and sale of cigarettes.

Previous efforts to reduce the risk of smoking by marketing low-tar, low-nicotine cigarettes failed because smokers compensated by smoking more intensely or more often to maintain their blood levels of nicotine (12). Instead, I recommend that we reduce TSNA levels in cigarettes, cigarette smoke, snuff, and chewing tobacco by reducing the application of nitrate fertilizer by tobacco farmers, using tobacco strains that produce low yields of TSNA and, especially, curing and storing tobacco by the methods used for snus. Polycyclic aromatic hydrocarbon levels might be reduced by altering combustion conditions during smoking. Such measures are unlikely to lower the incidence of smoking-induced deaths from heart disease, stroke, and chronic obstructive pulmonary disease, which account for most of the deaths due to smoking and are probably not due to TSNA or polycyclic aromatic hydrocarbon. However, these changes are likely to prevent a large proportion of the deaths due to lung cancer (currently 160,000 per year in the United States) and other smoking-related cancers that may be induced by TSNA and polycyclic aromatic hydrocarbons (2). I thank Stephen S. Hecht (University of Minnesota Cancer Center, Minneapolis, MN) for advice and help with this letter.

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References

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