Accepting premature deaths from smoking

W A Farone

Could partial reduction of some toxic components in cigarettes lead to reductions in premature deaths from smoking?

If one analyses the suggested means of solving the problem of premature death and disease caused by smoking, the strategies break down into four broad groups. Listed in the order in which I perceive these to have the highest probability of resulting in the most reduction in death to the least these are:

1) Smoking cessation
2) Replacement of the pharmacologic (pleasant) sensations of nicotine in cigarette products with non-cigarette products that can be viewed as providing either medicinal nicotine or analogue chemicals with similar function
3) Reduction in toxic components of cigarette smoke immediately to levels that are suggested by current science as “acceptable” based on existing dose–response data for cumulative lifetime exposure. (This is a central part of the theme of the California Environmental Protection Agency’s requirements—that is, that there are levels of chemicals for which the risk is not more than “background”.)
4) Partial reductions in some toxic components over time to eventually lead to products with reduced premature death rates

Should the last strategy be regarded as acceptable in light of the more definitive reductions in the other strategies? Is the reduced level of premature death hypothesised to follow a slow response to reducing toxicity for cigarettes something that should be supported?

REducing nitrosamines

In the paper by Gray and Boyle1 in this issue, a case is made for the reduction of one class of known carcinogen in cigarette smoke, the tobacco specific nitrosamines (TSNAs). They argue that the known global variations in TSNAs among cigarette brands are unacceptable and that the lowest levels able to be produced should be required. The fact that lower levels in some cigarettes have been achieved implies that all cigarettes could have the same lower levels.

Would there be any reduction in premature deaths if only TSNAs were reduced and, if so, would this reduction make such modified tobacco products more acceptable to current and future smokers? Or would a TSNAs reduction only lead to the same result as the tar reduction derbies of the 1960s and 1970s—that is, no significant reduction in premature death rates. From the 1950s to about 1980 delivery levels of cigarettes in the USA experienced a reduction in the amount of chemicals delivered based on the standardised Federal Trade Commission test. This reduction was seen in the sales weighted “tar” average reported during the period. Today, these lower levels have been maintained, but not reduced further. The reductions were of the order of 70% over that period. Although there is the issue of smoker compensation in the lower delivery cigarettes, one might still have expected that the level of disease would have been significantly reduced based on the risk associated with exposure. For example, one might have expected that on average the risk would have dropped from that of a two pack a day smoker to that of a one pack a day smoker. Unfortunately dose–response curves are not linear. Further, the synergy of so many potent toxins was not considered.

The combination of sufficient toxins and the dosing of the newer cigarettes created essentially the same disease rates.

IMPLYING REDUCED RISK

The manufacturers of cigarettes would like nothing better than to keep products on the market that are virtually unchanged from existing products. If the act of removing one toxin or even a few of the many toxins in cigarettes could be advertised as an achievement with the implications of reduced risk, more smokers may be deterred from quitting and more young non-smokers may feel reassured that the risk is small enough to begin smoking. This is directly analogous to the implicit advertising promise of the 1970s until today that lower tar cigarettes convey less risk.

There are several technologies that reduce TSNAs without reducing other toxic chemicals to any great extent. For example, the extraction technology used by Philip Morris in the denicotinised NEXT cigarette removes the bulk of the TSNAs along with the nicotinic alkaloids. In principle, replacing the alkaloids while leaving out the TSNAs would result in a great reduction in TSNAs, even more so than TSNAs reduction resulting from improvements in the technology of curing tobacco. The TSNAs that would be delivered to a smoker would “only” be those that formed during the reactions of the alkaloids during the smoking and smoke aging process. A Philip Morris reference indicates that two thirds of the TSNAs in Burley tobacco smoke and 33% in Bright tobacco cigarette smoke is produced because of the high temperatures that occur during the burning of a cigarette, that is pyrosynthesised. In this case the amount of TSNAs in the cigarette itself can be misleading and the pyrosynthesised quantities are not eliminated by extraction or curing. Additional treatments, such as the removal of nitrosamine precursors like nitrate that produce nitric oxide, are needed.

Table 1 presents some of the information concerning levels of some of the carcinogenic materials in smoke considered to be “acceptable” based on a daily exposure value compared to a range of these chemicals reported in cigarette smoke on a “per cigarette” basis.

The list in table 1 is only for carcinogens and is a very limited selection chosen to make the specific point that many carcinogens are now being delivered at levels far in excess of reasonable levels. The number of cigarettes smoked per day will multiply the values in the “Amount in cigarettes” column. Eventually all of the carcinogens and teratogens on the California Proposition 65 list will have levels defined that are nominated as values of daily exposure that will increase risk by less than 1 in 100 000. These levels are a reasonable starting point but still do not take into account cumulative and synergistic effects.

Regulating levels

If such levels were regulated, the cigarette industry would no longer be able to argue as they do now that they are “trying to reduce levels”. The levels of the chemicals and the risk associated with the levels would be defined. Levels that do not achieve some scientifically
Table 1 Comparison of levels of some carcinogens in cigarette smoke

<table>
<thead>
<tr>
<th>Compound</th>
<th>Safe harbour level*</th>
<th>Amount in cigarettes</th>
<th>Safe harbour level*</th>
<th>Amount in cigarettes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaldehyde</td>
<td>90 Micrograms</td>
<td>500–1400 Micrograms</td>
<td>90 Micrograms</td>
<td>500–1400 Micrograms</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>40 Micrograms</td>
<td>70–100 Micrograms</td>
<td>40 Micrograms</td>
<td>70–100 Micrograms</td>
</tr>
<tr>
<td>Benzo(a)pyrene</td>
<td>60 Nanograms</td>
<td>20–40 Nanograms</td>
<td>60 Nanograms</td>
<td>20–40 Nanograms</td>
</tr>
<tr>
<td>Benzo(a)anthracene</td>
<td>40 Nanograms</td>
<td>20–70 Nanograms</td>
<td>40 Nanograms</td>
<td>20–70 Nanograms</td>
</tr>
<tr>
<td>Dibenzo(a)pyrene</td>
<td>2 Nanograms</td>
<td>1–7–3.2 Nanograms</td>
<td>2 Nanograms</td>
<td>1–7–3.2 Nanograms</td>
</tr>
<tr>
<td>4-(N-Nitrosomethylamino)</td>
<td>14 Nanograms</td>
<td>80–770 Nanograms</td>
<td>14 Nanograms</td>
<td>80–770 Nanograms</td>
</tr>
</tbody>
</table>

*Data on safe harbour levels (levels of a chemical that are estimated to provide an increased risk of cancer of no more than 1 in 100 000) are from the California Environmental Protection Agency. 

References

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