Declining smoking in Sweden: is Swedish Match getting the credit for Swedish tobacco control’s efforts?

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It is imperative that public policy be based on the most thorough, balanced, and critical appraisal of the available evidence. Regrettably, the two papers \(^1, \(^2\) on which we were invited to comment fall short of those criteria.

**DID SNUFF REALLY REDUCE SMOKING IN SWEDEN?**

Bates \(^et\ al\)\(^3\) concluded snus played a “positive public health role” but did not weigh all available evidence nor critically appraise the methodologies, funding sources, or interpretations of the studies they included. Their “Evidence from Sweden” section included only an unpublished survey with unknown methodology, \(^4\) a newspaper article, \(^5\) and a study from northern Sweden. \(^6\) Foulds \(^et\ al\)\(^7\) concluded that snus had “...a direct effect on the changes in male smoking and health” with little additional evidence. However, both papers ignored published studies and selectively reported findings. A consideration of all the available evidence suggests snus played, at best, a minor role in reducing smoking in Sweden.

A one year Swedish cohort study of persons aged 45–69 years at baseline in 1992–94 examined predictors of smoking cessation or change to non-daily smoking among baseline daily smokers \(n = 3550\)\(^7\). At baseline, 7.0% of men and 0.4% of women used snuff. At follow up, 7.2% of daily smokers had quit and 6.5% were non-daily smokers. The study found: snus use was not associated with smoking cessation; snuff use by non-daily smokers neither predicted cessation nor prevented transition to daily smoking\(^7\); and even if snuff helped some smokers to quit, it accounted for a small fraction of cessation.

In another prospective study, \(n = 5104\) persons aged 16–84 years were interviewed in 1980–81 and followed up in 1988–89. \(^*\) These included \(n = 1546\) daily smokers, \(418\) men who used snuff daily, and \(129\) men who used both snuff and cigarettes. By follow up, 26% of female and 28% of male smokers had quit. Just 5% of male smokers switched to snus and 2% starting using snus in addition to cigarettes. Among male exclusive snus users, 26% quit all tobacco use and 10% took up cigarettes in addition to or instead of snus. Among male dual product users, 56% either continued dual product usage or exclusively smoked, 31% exclusively used snus, and 13% quit all tobacco usage. Even in the one cohort study \(^*\) cited by Foulds \(^et\ al\)\(^7\), where the baseline prevalence of male snus use was 23%, the majority of men and nearly all women who quit smoking did so without ever using snus; 16% of snus users also were using cigarettes at follow up.

Analysis of Ramström’s unpublished Swedish data\(^1\) revealed that 22% of male and 4% of female former smokers used snus as a quitting aid on their last quit attempt, as did 12% of males and 3% of women who were still daily smokers. Smoking quit ratios were not much different for men who used snus (65%) and those who did not (61%). The pattern was similar for women: 52% of female ever smokers who used snus and 55% who did not had quit smoking.

In a 2000 Swedish survey of 1000 former smokers and 985 current daily smokers aged 25–55 years, Gilljam and Galanti\(^9\) found that using snuff at the most recent quit attempt increased the likelihood of abstinence \((\text{odds ratio (OR)} 1.54, 95\% \text{ confidence interval (CI) 1.09 to 2.20})\), with a small difference in using snus for cessation between males who quit (28.7%) and those who tried unsuccessfully (23.0%). More detailed analysis\(^10\) of the TEMO data\(^1\) cited by Bates \(^et\ al\)\(^2\) showed the large majority of men (71%) and women (97%) who quit smoking did not use snus at their last quit attempt, with modest effectiveness for snus as a cessation strategy in that observational study. That is hardly compelling evidence for snus as “an important explanation”\(^11\) for the decline in smoking in Sweden.

Foulds \(^et\ al\)\(^2\) and Bates \(^et\ al\) did not examine whether the population subgroup driving the recent growth in snus usage was the same one quitting smoking. We addressed that question by conducting a birth cohort analysis using published Swedish tobacco prevalence data\(^11, \(^13\)\) (table 1). The largest increase in daily snus use in the 1990s was among males who were aged 16–24 years in 1989; most other birth cohorts experienced a slight decrease. Daily smoking declined for all birth cohorts between 1989 and 2000 (table 1); the smallest decline was among the same birth cohort that experienced the greatest increase in snus use. Applying the proportions in table 1 to Swedish population figures for 1999, \(^14\) we estimated a net gain of 26 859 male daily snus users between 1989 and 1999; \(43\,540\) males became users and 16 681 quit. Of the \(43\,540\) new users, \(40\,347\) (93%) were age 16–24 at the beginning of that decade. In contrast, there was a net decrease of 230 391 male daily smokers during the decade; males aged 16–24 years in 1989 accounted for \(10\,099\) (4%) of them. To reduce the effects of cigarette related mortality on observed smoking prevalence, we repeated the analysis limited to males less than 44 years old in 1999. In that analysis, males age 16–24 in 1988/89 accounted for 100% of the increase in snus use and just 13% of the decrease in daily smoking. The groups quitting smoking in the 1990s were not the ones taking up snus.

Bates \(^et\ al\)\(^2\) implied snus use prevented smoking among young males, but cross sectional\(^13\) and cohort\(^14\) studies contradict that assertion. For example, 41% of male fifth graders in Stockholm County who used only snus in 1997 were smoking one year later.\(^15\) That finding is consistent with one US four year follow up study in which 40% of youth took up cigarettes instead of or in addition to use of oral tobacco\(^16, \(^17\)\) yet cited by Foulds \(^et\ al\)\(^2\) as evidence that snus was not a gateway to smoking. Although the relevance of US data to Sweden is not clear, oral tobacco use apparently predicts cigarette smoking for a proportion of Swedish and US youth. A 2001 Swedish survey of 15–16 year olds found a higher prevalence of daily smoking for girls (16%) than for boys (10%);\(^18\) daily snus use was very low for girls (2%) and quite high for boys (18%). Perhaps some boys who would have smoked used snus instead, but boys’ snus use far exceeded the sex difference in daily smoking; snus use may have added to total tobacco use by young males more than it “prevented”...
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Univariate analysis of snus use cited by Foulds et al.

...the prevalence of daily snuff use among 16–24 year olds declined from 37.1% to 18.7% for females and from 27.7% to 14.0% for males.

Daily use of snuff remained relatively constant among 16–24 year old males during the 1990s, at 21% to 23%.

They clearly limit themselves to an assessment of smoking and then largely ignore differences in smoking among young females and the large majority of the decline among males.

Health Effects of Snuff: A Critical Appraisal

Examination of the reviews by Foulds et al. and Bates et al. for just one disease is illustrative of the type of simplistic conclusions that might be reached when the nuances of epidemiologic research are not appreciated, findings are not fully evaluated, and published critical reviews are ignored.

Foulds et al. concluded that snuff is not a risk factor for oral cancer. However, critical appraisal of the two case-control studies they cited suggests both suffered from small sample size and low statistical power. There was also misinterpretation of the findings from the Lewin et al. study. That study, in fact, found that snuff use was associated with a substantially elevated risk for oral cancer compared to ever smoking cigarettes (OR 4.7, 95% CI 1.1 to 18.6) and former snuff use (OR 10.5, 95% CI 1.4 to 83.3). Current snuff use (OR 3.3, 95% CI 1.6 to 6.7) was comparable to ever smoking cigarettes for oral cancer associated with ever using snuff (OR 4.7, 95% CI 1.1 to 18.6). Those results differ from the many case-control studies that found a strong independent risk for cigarette smoking in the development of oral cancer, suggesting possible methodological problems. There were similar limitations in other studies uncritically cited by Foulds et al., such as very small sample size and uncontrolled confounding by smoking in the multivariate analysis of snuff and stroke. As a minimum, better studies not funded by snuff manufacturers are needed to evaluate the risk for adverse health effects associated with oral tobacco use.

Levels of Toxins in Oral Snuff

Foulds et al. presented data on toxins in oral snuff, reported Swedish Match’s “quality” standard of tobacco specific N-nitrosamine (TSNA) levels below 10 000 µg/kg, and cited one industry study on urine mutagenicity as a method for assessing reduction in harm. Bates et al. highlighted Swedish Match’s voluntary standard as a possible standard for European Union regulation. However, the authors of both papers ignored the Institute of Medicine report that examined the issue of TSNA levels in Swedish snuff and other snuff and called for more research on its cellular and genetic toxicity before promoting it as a harm reduction agent.

The US Department of Agriculture and the Food and Drug Administration have set permissible limits for N-nitrosamines of 5 µg/kg for bacon and 5 µg/kg for beer. The Swedish standard of 10 000 µg/kg dwarfs the levels for those other consumer products and its role as a “safer” product is questionable.

Patterns of Use

Foulds et al. and Bates et al imply that snuff use actually reduced initiation of cigarette smoking. Yet, could any health professional seriously advocate taking up oral tobacco as a means of preventing cigarette smoking? This seems dangerously close to advocating oral opioid narcotics such as codeine as a means of avoiding heroin use.

Implicit in the Foulds et al. paper is that how a product is used is as important a determinant of its health effects as how it is made. This is the crux of the issue of whether oral tobacco can contribute to overall reductions in tobacco related disease and if there are subpopulations for whom oral tobacco is either without benefit or harmful. This lesson was well established by the experience with low tar or “light” cigarette marketing as well as the promotion of oral tobacco by United States Smokeless Tobacco Company (UST). UST’s youth directed health image promotion helps explain why oral tobacco was the main growth segment of the US tobacco industry in the 1970s and 1980s. Most of the growth was from recruitment of young non-tobacco users to a course of tobacco addiction, not adult smokers switching to snuff.

How a product is used can be strongly influenced by marketing, cultural practice, taxes, and access. Unfortunately, Foulds et al. pay little attention to these other plausible determinants of patterns of tobacco use in Sweden. For example, Sweden was among the world’s leaders in its vigorous anti-smoking campaigns of the 1970s and 1980s that continue today. Whether snuff uptake contributed to this reduction or simply became a new hazardous behaviour in men who otherwise would have been tobacco-free is not clear.

An equally plausible explanation for the slightly higher prevalence of smoking among women may be related to sex differences in employment status and therefore differential exposure to smoke-free workplace regulations, which began...
in Sweden in the early 1980s and have since expanded. Swedish men are more likely than women at nearly all ages to be in paid employment, and employed women are far more likely than men to be working part-time (41% v 8%). Therefore, men would be more likely than women to be impacted by smoke-free workplace regulations. The Nordic countries to which Bates et al and Foulds et al compared Sweden—Denmark and Norway—have substantially fewer restrictions on smoking in public spaces and began tobacco control efforts quite some time after Sweden. One Nordic country not mentioned in either paper, Finland, has had a fairly aggressive tobacco control movement and now has bans on indoor smoking comparable to Sweden. Finland’s per capita cigarette consumption has been declining since the early 1970s and consequently so has the male lung cancer mortality rate, which continues to move toward the rate for Sweden’s males. Snuff usage was low in Finland for most of that period, except for a sharp increase from the late 1980s until its sale was banned in 1995.

There is a considerable dual use of cigarettes and snus in Sweden. If snus by smokers reduced the pressure to quit then snus may be contributing to increased health risks by delaying smoking cessation. It is disappointing that rather than consider this serious concern and explore how it could be minimised, Foulds et al do not even address it.

WHAT CAN THE EXPERIENCE OF SWEDISH SNUS TEACH US ABOUT HARM REDUCTION?

Whatever one concludes about the role that snus may have played in reducing smoking in Sweden, its applicability to other countries and other regulatory regimens is debatable. A product’s role as a cessation aid or harm reducing measure depends on too many factors to be accepted cross culturally. Manufacturers’ responses to consumer preferences and economic factors—such as additives, type of tobacco, curing methods, pasteurisation processes, and storage issues—will affect the its harm profile. For example, some Swedish Match products sold in the USA have higher TSNA levels than those sold in Sweden. Differences between countries in the marketing of products certainly affect their usage. Sweden has banned advertising claims, including health claims, and snus cannot be overtly marketed to children through sponsorship of youth events or the type of juvenile advertising so often employed in the USA.

Usage patterns are also very culturally dependent. What groups traditionally have used oral tobacco? Is there a custom or does it need to be introduced to a naive population? Are there existing social barriers to using snuff? Finally, differences in the regulatory environment are crucial. In the absence of government regulation of the product, as is the case in the USA, the manufacturer will not necessarily produce the least hazardous product possible and consumers will not necessarily have the type of complete information necessary to make an informed choice. Unfortunately, the “Swedish experience” already has been cited in the USA by an oral tobacco company to support marketing intentions that bear little resemblance to what has happened in Sweden and with products far higher in TSNAs, as well as in testimony before the US Congress. At the end of the Bates et al paper, various caveats and cautions are issued; it is unfortunate that these were not in the introduction nor given the same prominence. If there is a role for oral tobacco in a comprehensive effort to reduce the death toll from tobacco use, then its manufacture and marketing must be overseen by an agency with comprehensive regulatory authority. A regulatory agency should be open to all strategies that are scientifically based and that will save lives. However, the decision about what role oral tobacco plays in that overall scheme is a decision that can only be made by an agency that has all of the relevant information.

This is the one area in which we are in total agreement with Bates et al: there must be meaningful regulation of the product itself, as well as proper review of any substantiation for claims and marketing of snus before harm reduction claims should be made.

There are two final concerns: while considering what role snuff can play in harm reduction, we should also weigh the relative cost/benefits to public health of the use of various nicotine sources available for cessation/harm reduction. Where does snuff fit within this continuum? If a new regulatory regimen is necessary to ensure that snuff plays a positive role, the effort to achieve that reform should also include looking at the current regulatory constraints on existing, “cleaner” (that is, pharmaceutical) nicotine sources. Should these restrictions be examined to make clean nicotine more effective, less expensive, and easier to use? For example, in the USA, the FDA already has regulatory authority over medicinal nicotine products. These products have been established as safe, at least for short term use, but little has been done to encourage their improvement or to explore their long term use and potential for harm reduction. Any attempt to enhance the harm reduction market by including new products or new uses for current products must include the question of the proper regulatory environment for medicinal nicotine.

Finally, as the debate goes forward, we need to keep it focused on the main issue, which is how to reduce the death and disease caused by tobacco use. The rhetoric needs to be toned down, because everyone in this debate has a common interest, even if we focus on different ways to achieve that goal. Those who favour a more cautious regulation-first approach are no more believers in a “quit or die” strategy than those who view snus as an element of a harm reduction strategy are “industry stooges”. In many cases, the difference in focus result from differing degrees of regulatory authority and advertising restrictions, the differing behaviour of national oral tobacco companies, as well as differences in the product itself. We need to find a common vocabulary that allows us to debate the science and the political realities, while still remaining focused on the goal.

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REFERENCES

*Copy testing by the Federal Trade Commission indicates that health claims, even those with disclaimers, may mislead a substantial number of consumers as to the relative health risks and benefits of a product. Health claims can provide a halo effect to the advertised product, which make it hard for consumers to understand the risks still inherent in using the product. See FTC, A Joint Report of the Bureau’s of Economics and Consumer Protection, Generic copy test of food health claims in advertising (November 1998).
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