

REVIEW

Effect of smokeless tobacco (snus) on smoking and public health in Sweden

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Objective: To review the evidence on the effects of moist smokeless tobacco (snus) on smoking and ill health in Sweden.

Method: Narrative review of published papers and other data sources (for example, conference abstracts and internet based information) on snus use, use of other tobacco products, and changes in health status in Sweden.

Results: Snus is manufactured and stored in a manner that causes it to deliver lower concentrations of some harmful chemicals than other tobacco products, although it can deliver high doses of nicotine. It is dependence forming, but does not appear to cause cancer or respiratory diseases. It may cause a slight increase in cardiovascular risks and is likely to be harmful to the unborn fetus, although these risks are lower than those caused by smoking. There has been a larger drop in male daily smoking (from 40% in 1976 to 15% in 2002) than female daily smoking (34% in 1976 to 20% in 2002) in Sweden, with a substantial proportion (around 30%) of male ex-smokers using snus when quitting smoking. Over the same time period, rates of lung cancer and myocardial infarction have dropped significantly faster among Swedish men than women and remain at low levels as compared with other developed countries with a long history of tobacco use.

Conclusions: Snus availability in Sweden appears to have contributed to the unusually low rates of smoking among Swedish men by helping them transfer to a notably less harmful form of nicotine dependence.

In recent times the tobacco industry has been active in developing and marketing new products that might be perceived as less harmful to health than typical cigarettes.^{1,2} At the same time, there has been an increasingly vigorous debate within the public health community over the most appropriate response to the new products being developed by the industry.^{3–5} In this debate, public health advocates have been mindful of the historical precedents set by previous tobacco industry attempts to introduce new product lines that have been perceived as less harmful. It is now clear that so called “light” cigarettes were widely believed to be less harmful (and continue to be by the majority of consumers) but in fact are no less deadly than standard cigarettes.⁶ The introduction and marketing of these products may well have had a serious adverse effect on public health by duping hundreds of millions of smokers into the belief that they could continue to smoke at reduced risk.

In the current debate over tobacco harm reduction, some have cited the “Swedish experience” as an example of tobacco product switching that may have had a positive effect on smoking and public health.⁷ This article aims to review our knowledge about smokeless tobacco use in Sweden and its likely effect on tobacco smoking and public health in that country.

WHAT IS SNUS?

“Snus” is the name given to the form of smokeless snuff tobacco commonly used in Sweden. It is a moist, ground oral tobacco product that is typically placed behind the upper lip, either as loose ground tobacco or contained in sachets appearing like small teabags. The snus is typically held in the mouth (without chewing) for approximately 30 minutes before it is discarded.

MANUFACTURING

Snus both contains and delivers a number of harmful substances, including cancer-causing tobacco specific nitrosamines (TSNAs). It has become clear that different selection and curing methods can affect the levels of nitrites and hence TSNAs present in the raw tobacco before processing.⁸ Over recent decades snus manufacturers have selected tobacco blends that have been air and sun cured (dried), while US moist snuff products tend to include blends high in fire cured tobacco.

After curing, raw cured tobacco is cut into small strips, dried, ground, and sifted before processing. In Sweden, by tradition, snus production has included a process in which the tobacco is heat treated with steam for 24–36 hours (reaching temperatures of approximately 100°C). Ingredients added are: 45–60% water, 1.5–3.5% sodium chloride, 1.5–3.5% humectants, 1.2–3.5% sodium bicarbonate, and less than 1% flavouring. It is claimed that the heating process kills bacteria, producing a relatively sterile product. The product is then packaged in cans and refrigerated during storage. In Sweden the product is also kept in refrigerators by the retailers. One study examined levels of carcinogenic TSNAs in snus kept at temperatures ranging from –20°C to +23°C for 20 weeks.⁹ This exposure to a variety of temperatures over time did not produce a significant increase in concentrations of TSNAs, suggesting that the exposure to heat during

Abbreviations: CI, confidence interval; IOM, US Institute of Medicine; MI, myocardial infarction; OR, odds ratio; OSCC, oesophageal squamous cell carcinoma; RR, relative risk; TSNAs, tobacco specific nitrosamines

manufacturing may itself have prevented microbial activation of nitrites.¹⁰

This manufacturing process contrasts with that traditionally used in the USA, in which the product is fermented (rather than being subject to high temperatures), allowing the continued formation of TSNA. In addition, North American smokeless tobacco is not typically stored in refrigerators. One study found that nitrite and TSNA levels increased significantly in US snuff stored at 37°C for four weeks.¹¹

Although different products vary in their pH levels, snus typically has a pH in the range 7.8–8.5.^{12–13} This is important because only nicotine in the free-base form is rapidly absorbed through the mucosal membrane, and the proportion of free-base nicotine available from tobacco is determined by the pH level. For example Brunnemann and Hoffmann compared two brands and found that one brand with a pH of 5.84 had only 1% of the nicotine in the free-base form and another brand with a pH of 7.99 had 59% of the nicotine available in free-base form for absorption.¹⁴ Another study found that a leading Swedish snus brand had a higher pH (and therefore probably more efficient nicotine delivery) than five comparison brands of US smokeless tobacco.¹³

Delivery of harmful substances

Possibly as a result of the differences in manufacturing and storing procedures, snus has been claimed to contain lower levels of some harmful substances than many of the brands available in North America and notably lower levels than exist in the smokeless tobacco used in the Sudan and India.¹⁰ Table 1 below summarises data from five studies^{13–18} of TSNA levels in various samples of different brands marketed in different countries. The total TSNA concentration varied greatly among the US brands from 4.1 to 128 (µg/g dry tobacco). There is little evidence to support claims that TSNA levels have consistently dropped over the past decade in North American snuff (for example, Copenhagen brand in 1994 had a measured TSNA level of 17.2 and in 2000 it was 41.1). Snus brands selected in Sweden from 1990, 1991, and 2000 have generally been lower and have varied from 9.2 to 11.2 µg/g in the three samples in 1990–91 and 2.8 µg/g in 2000. Brunnemann and Hoffmann¹³ also examined the effects of storage for six months at room temperature and found that in two leading US brands, the TSNA levels increased by between 30–130% whereas in the snus brand there was no increase. More recently the manufacturer of

Table 2 Gothiatek standard.¹⁹ Voluntary market based toxicity standard used for snus products by Swedish Match Tobacco Company

Toxin	Limit
Nitrate	3.5 mg/kg
Tobacco specific nitrosamines (TSNA)	5 mg/kg
N-Nitrosodimethylamine (NDMA)	5 µg/kg
Benz(a)pyrene (BaP)	10 µg/kg
Cadmium	0.5 mg/kg
Lead	1.0 mg/kg
Arsenic	0.25 mg/kg
Nickel	2.25 mg/kg
Chromium	1.5 mg/kg

mg/kg, thousandth gram per kilogram product (based on Snus with 50% water content); µg/kg, millionth gram per kilogram product (based on Snus with 50% water content); double the limits for dry weight equivalents.

snus has created and publicised a quality standard, the Gothiatek standard,¹⁹ for its snus products that includes maximum permissible limits for “undesirable substances” (table 2). It is unclear if all Swedish Match smokeless tobacco products produced in Sweden and abroad adhere to the Gothiatek standard.

One method of assessing the potential harmfulness of a tobacco product is to measure the level of circulating mutagens in body fluid after exposure to the tobacco. Curvall and colleagues²⁰ compared the mutagenic activity of urine from snus users, cigarette smokers, and non-tobacco users. They found that smokers had notably increased urinary mutagenic activity, whereas there was no significant difference between snus users and non-tobacco users.

Nicotine delivery

Swedish snus, like some other brands of smokeless tobacco, contains and delivers quantities of nicotine comparable to those typically absorbed from smoking cigarettes. As shown in fig 1, each dose typically provides a venous nicotine “boost” of around 15 ng/ml after half an hour, with steady state levels around 30 ng/ml being typical.^{21–22} These nicotine levels are very similar to those found in cigarette smokers,²¹ with the main difference from smoked tobacco being the slightly slower nicotine absorption and the lack of a higher concentration arterial “bolus” that results from nicotine inhalation. These nicotine levels obtained from snus are

Table 1 Mean nitrosamine content (and range) of moist snuff products from various sources based on dry weight

Country and brand (year sampled)	Manufacturer	Nicotine (mg/g)	NNK (µg/g)	NNN (µg/g)	Total TSNA (µg/g)
Sweden					
Three brands* (1990–91) ¹⁵	Swed Match		1.4–2.1	5.2–5.7	9.2–11.2
Ettan Snus (2000) ¹³	Swed Match		0.5	1.1	2.8
Sudan (Toombak)					
5 Samples* (1990) ¹⁶		32.2–102.4	630–7870	830–3805	
5 Samples* (1990) ¹⁶		8.4–26.0	1140–2790	420–1550	
3 Samples* (1993) ¹⁷			188–362	241–369	
USA					
2 Samples* (1991) ¹⁵		18.6–20.6	0.5–0.8	4.8–8.0	
1 Sample* (1992) ¹⁵		16.7	0.6	5.6	
Copenhagen (1994) ¹⁸	USSTC	12 (12.7–11.3)	1.9 (1.3–2.5)	8.7 (10.1–7.3)	17.2 (20.2–14.2)
Skool, Original fine cut(1994) ¹⁸	USSTC	11.9 (13.4–10.7)	1.3 (1.4–1.2)	8.2 (9.5–6.9)	14.9 (17.4–12.4)
Skool Bandts Straight(1994) ¹⁸	USSTC	10.1 (10.9–9.3)	0.9 (1.2–0.6)	5.1 (6.1–4.1)	8.2 (9.9–6.5)
Kodiak Wintergreen(1994) ¹⁸	Conwood	10.9 (10.1–11.7)	0.6 (0.8–0.4)	6.3 (7.4–5.2)	11.0 (13.4–8.6)
Hawken Wintergreen(1994) ¹⁸	Conwood	3.2 (3.4–3.0)	0.2 (.24–.16)	3.1 (3.4–2.8)	4.1 (4.5–3.7)
Skool (2000) ¹³	USSTC		4.3	20.8	64.0
Copenhagen(2000) ¹³	USSTC		3.4	14.3	41.1
Timber Wolf(2000) ¹³	Swed. Match		0.95	3.0	7.5
Silver Creek(2000) ¹³	Swisher		17.8	41.4	127.9

All available pertinent data from referenced studies is reported. Blank cells indicate that data were not provided in the referenced study.

*Brand not specified in study.

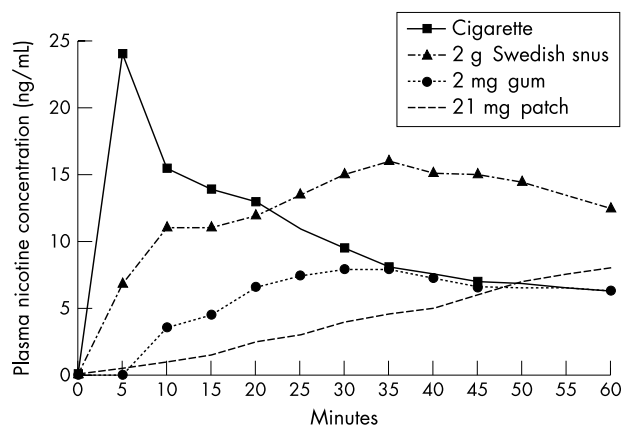


Figure 1 Venous blood concentrations in nanograms of nicotine per millilitre (ng/ml) of plasma as a function of time for various nicotine delivery systems; all plasma nicotine concentrations have been reconfigured such that the pre-absorption level starts at 0 ng/ml (that is, to take out the baseline differences). Cigarette, and 2 mg nicotine gum, adapted from Russell *et al.*,²⁴ and 21 mg patch adapted from Stratton *et al.*,³ page 100. Swedish snus plasma nicotine concentrations in 10 Swedish snus users from a single 2 g pinch of loose snus adapted from Holm *et al.*²¹

about twice as high as the nicotine concentrations typically obtained from nicotine replacement therapy.²³ The nicotine levels shown in fig 1 are from users of loose snus and it is possible that some other brands (particularly portion packed products or those with a lower pH) may give different levels.

IS SNUS HARMFUL TO HEALTH AND IS IT LESS HARMFUL TO AN INDIVIDUAL USER THAN CIGARETTES?

Many of the smokeless tobacco users participating in the older epidemiological studies discussed below may have been exposed to products delivering higher quantities of harmful substances than current versions of these products.

Nicotine dependence

Given the pattern of nicotine absorption described above there can be no doubt that snus is dependence forming in much the same way as other forms of tobacco consumption. There is some evidence that the dependence potential of nicotine and other psychoactive drugs is related to their speed of delivery to the brain^{23, 25} and so one would expect snus and other non-inhaled forms of nicotine delivery to be proportionately less addictive than inhaled tobacco smoke. However, there is clear evidence that users of products with snus-like nicotine delivery profiles develop cravings and nicotine withdrawal symptoms when attempting to abstain, and find it difficult to quit.^{21, 26} While snus probably does not produce stronger nicotine dependence than smoking, it has just minimal, if any, advantages over cigarettes or other smokeless nicotine delivery products in terms of its lower potential to induce dependence. In fact, its high nicotine delivery and hence dependence potential (relative to most other non-smoked delivery modalities) may be a critical factor enabling it to compete with the more rapidly absorbed nicotine from smoked tobacco.

Oral cancer

One of the biggest concerns about the use of smokeless tobacco stems from the relatively large body of evidence from a number of countries showing that oral tobacco use can cause cancer of the mouth, head, and neck. With regards to its use in India, the 2001 US Institute of Medicine (IOM) report³ stated that, "A large number of studies in India,

including cohort, case-control, and intervention studies, support an association between oral cancer and smokeless tobacco, and these studies are consistent, strong, coherent and temporally plausible" (p 427). The IOM report stated that toombak users in Sudan also have a much higher relative risk (RR) of oral cancer than non-users and that "In spite of conflicting US data, it can be concluded that snuff use in the United States also increases the risk of oropharyngeal cancers" (p428). In contrast, there is consistent evidence from two case-control studies in Sweden showing no increased risk of cancer of the head, neck, or mouth among snus users.

Schildt and colleagues²⁷ investigated whether snus leads to increased risk of oral cancer by comparing various risk factors in 410 cases of oral cancer and 410 matched controls identified during the period 1980–89. Ninety six per cent of the identified cases and 91% of identified controls participated in the study (leaving full data from 354 matched pairs) and 20% of the overall sample were current or ex snus users. Univariate analyses found significant increased risk of oral cancer as a result of smoking (odds ratio (OR) 1.8 for active smokers), and alcohol (OR 1.9 for beer drinkers) but no increased risk for active snus use (OR 0.7, 95% confidence interval (CI) 0.4 to 1.1). The authors concluded: "Our results do not support any association between use of oral snuff and oral cancer."

Lewin and colleagues²⁸ conducted a similarly designed study, identifying cases of head and neck cancer in two regions of Sweden between 1988 and 1991 and matched controls. Interviews were conducted with high proportions of identified cases (90%) and controls (85%). This study found significantly increased risks of head and neck cancers associated with alcohol use and smoking, but no increased risk associated with former or current snus use. The RR for head and neck cancer among snus users as compared with non-snus users, after adjusting for age, region, alcohol, and smoking was 1.0 (95% CI 0.6 to 1.6). Similarly there were no significant relations between duration of snus use or lifetime consumption and head/neck cancer.

A recent systematic review of the health effects of smokeless tobacco concluded: "Chewing betel quid and tobacco is associated with a substantial risk of oral cancers in India. Most recent studies from the US and Scandinavia are not statistically significant, but moderate positive associations cannot be ruled out due to lack of statistical power."²⁹ Snus causes a number of non-malignant oral diseases, including oral lesions³⁰ and dental caries.³¹ However, it appears as though the lesions produced by snus are reversible and disappear if snus use ceases.³⁰

Other cancers

Ye and colleagues³² conducted a case control study (504 cases and 1164 controls) examining the effects of smoking, alcohol, and snus use on gastric cancer in Sweden. They found a significant dose and duration related increase in gastric cancer risk with smoking, but no effect of snus or alcohol. They concluded that "smoking, but not the oral use of tobacco in the form of moist snuff, is positively associated with risk of gastric cancer".

Lagergen *et al.*³³ conducted a case-control study designed to test the association between smoking, snus, and alcohol use, and cancer of the oesophagus and gastric cardia in Sweden. Combined smoking and alcohol use was strongly associated with oesophageal squamous cell carcinoma (OSCC) (OR 23.1 for heavy users compared with never users), but snus use was not significantly associated with any of the cancer sites under study in multivariate analyses. There was some indication of a possible link between snus use and OSCC in that the odds ratio was 2.0 for use for over 25 years versus never snus use,

although because of the relatively small size of this sub-sample ($n = 14$ cases) this was not significant (95% CI 0.9 to 4.1). The authors concluded: "we found no statistically significant association between snuff dipping and risk of any of the studied tumors."

It remains possible, but unlikely, that a carcinogenic effect of snus only emerges after very long term use. Bolinder and colleagues³⁴ found a non-significant RR of death from cancer of 1.1 for snus users compared with never tobacco users (95% CI 0.9 to 1.4) in a prospective study of Swedish construction workers that included a relatively large sample, many of whom had used snus for over 40 years. The RR for cancer death was 1.0 (compared with non-tobacco users) for the 1734 snus users aged 55–65 years, most of whom would presumably have used snus for over 35 years. However, this study found significantly increased all cause mortality in snus users compared with never tobacco users, largely from elevated cardiovascular mortality. The RR for lung cancer among snus users compared with never tobacco users was 0.8 among men aged 55–65, whereas the RR was 30.6 (95% CI 14.6 to 64.1) for smokers of at least 15 cigarettes per day (again compared with never tobacco users).

Overall, the results of the five large studies examining snus in relation to cancer are consistent in finding no increased cancer risk among snus users. All of the Swedish studies of the relation between snus and cancer were robust enough to detect significant effects for tobacco smoking (often involving very large effect sizes), and the studies of oral cancer were also able to detect significant relations with alcohol use. The lack of relation with snus is therefore unlikely to be caused by methodological problems such as low statistical power.

Cardiovascular disease

Bolinder and colleagues conducted a series of epidemiological and clinical studies^{34–39} examining the effects of long term snus use on health, focusing on cardiovascular risk factors and myocardial infarction. Their first report³⁵ focused on a cross sectional study of almost 98 000 Swedish construction workers undergoing health examinations in 1971–4, including over 5000 exclusive snus users. This study found an increased prevalence of circulatory and respiratory symptoms among snus users and heavy smokers as compared to non-tobacco users, and an increased prevalence of hypertension in snus users compared to non-tobacco users. Surprisingly this study found the lowest prevalence of hypertension among smokers of at least 15 cigarettes per day. The results were based on univariate analyses, and did not control for potential confounders other than age.³⁵

Bolinder's second study³⁴ examined the relation between tobacco use and cardiovascular mortality in a larger sample ($n = 135\ 036$) of Swedish male construction workers recruited at a health examination in 1971–4 and followed up 12 years later.

This study found that snus users had a significantly higher risk of dying from a cardiovascular event than never tobacco users (RR 1.4, 95% CI 1.2 to 1.6). This excess risk was comparable to that of ex-smokers who had quit in the past five years, but smaller than heavy smokers (RR 1.9 compared with never tobacco users). The analyses in this study adjusted for age and region of origin, and (for at least some analyses, although it was not always stated) also adjusted for body mass index, blood pressure, diabetes, and heart problems at the time of entering the study. Alcohol consumption and cholesterol were not measured and so could not be controlled for.

Subsequent studies focused on a smaller sample of Swedish firemen (around 140, split approximately equally between snus users, smokers, and non-tobacco users). These

studies found that snus use did not influence exercise capacity,³⁶ or play a major role in the atherosclerotic process^{37,38} (both of which were adversely affected by smoking). However, they replicated the previous finding of higher daytime (but not night time) heart rate and blood pressure among both snus users and smokers as compared to non-tobacco users.³⁹ Overall, these studies by Bolinder and colleagues are suggestive of an increased cardiovascular risk from snus use, that is probably mediated by nicotine's sympathetic stimulant effects, and is of a smaller magnitude than the excess cardiovascular risks caused by smoking. It was suggested that snus' effects on blood pressure may be related to its sodium content (1.3–3.5% sodium chloride and 1.5–3.5% sodium bicarbonate).

However, two subsequent case-control studies by Huhtasaari and colleagues^{40,41} did not find a significantly increased risk of myocardial infarction among snus users as compared to non-tobacco users. Both of these studies were based on data collected in northern Sweden as part of the World Health Organization MONICA (multinational monitoring of trends and determinants in cardiovascular diseases) project. In both reports, the cases and controls were identified in the 1990s.

Huhtasaari and colleagues⁴⁰ found an age adjusted OR for myocardial infarction (MI) of 0.89 (95% CI 0.62 to 1.29) for snus use versus no tobacco use, whereas smoking significantly increased risk of an MI (OR 1.87, 95% CI 1.40 to 2.48). In multivariate analyses smoking remained significantly associated with MI, whereas snus use was not.

Huhtasaari subsequently conducted a larger study than the one reported in 1992, and included more detailed tobacco use histories and closer matching of cases and controls (matched for sex, date of birth, and area of residence).⁴¹ This study found (after adjustment for multiple cardiovascular risk factors) that cigarette smoking significantly increased risk of an MI (OR 3.53, 95% CI 2.48 to 5.03), whereas snus use significantly reduced the risk (OR 0.58, 95% CI 0.35 to 0.94) compared with men who never became regular tobacco users. When the analysis focused only on fatal cases, there was a tendency towards increased risk in snus users, but this was not significant (OR 1.5, 95% CI 0.45 to 5.03).

There is no clear explanation for the difference in results between the Bolinder³⁴ and Huhtasaari^{40,41} studies, although the different study populations, time periods covered, and outcomes measured (sudden death versus non-fatal MI) may have contributed. The similar magnitude of effect for fatal cardiovascular events found in these studies is suggestive of a slightly increased risk overall. On the other hand it remains possible that the effect of snus in the Bolinder study was caused by some unmeasured (and therefore uncontrolled) confounding factor, with dietary habits and alcohol consumption being examples of baseline variables not measured in that study. This possibility is supported by a recent report of the effects of smokeless tobacco in the USA, based on analyses of the First National Health and Nutrition Examination Survey epidemiologic followup study (NHANES-1) data.⁴² This study had 96% follow up of the original 14 407 participants and 98% identification of death certificates for the 4604 decedents by 1992. Male smokeless tobacco users were found to have moderately increased risks of some disorders, but all of these excess risks disappeared when variables such as race and poverty were controlled for. For example, the crude hazard ratios for male smokeless users versus non-tobacco users were 1.5 and 2.1 for circulatory and respiratory diseases before adjustment, but after adjustment for confounders these hazard ratios became 1.0 and 0.9. One potentially serious flaw with this study⁴² is that pipe and cigar users were included in the "non-tobacco user" comparison group, seriously undermining confidence

in their conclusion that US smokeless tobacco users have similar mortality outcomes to non-tobacco users. We cite this paper as an example of the changes in outcomes that can result from controlling for baseline variables, rather than as evidence of the safety of US smokeless tobacco.

Bolinder *et al*'s first study³⁵ found snus users to be at excess risk of a number of respiratory symptoms. For example, the OR for "cough in the morning" for snus users versus never tobacco users was 2.1 (95% CI 1.8 to 2.4), as compared with an OR of 7.9 for smokers versus never tobacco users. It is not easy to think of a plausible mechanism whereby exclusive snus use might cause respiratory symptoms. This study excluded all those who reported mixed use of snus and cigarettes or reported being an ex-smoker (n = 59, 864 excluded). However, the increased respiratory symptoms suggest the possibility that some of those reporting exclusive snus use were actually occasional or ex-smokers. Passive smoke exposure is another possible confounding factor that could potentially contribute to these findings. This study was initially funded by a health insurance group with the purpose of examining factors affecting sick leave and disability pensions. Some participants may have under-reported their recent or ex-smoking due to their belief that it either was not worth mentioning, or out of a concern that it may somehow affect their future benefits.

In reviewing the evidence from a range of clinical and experimental studies, Benowitz⁴³ concluded: "Overall, the epidemiologic and experimental data suggest that nicotine absorbed from smokeless tobacco, nicotine gum or transdermal nicotine is not a significant risk factor for accelerating coronary artery disease or causing acute cardiovascular events." This conclusion is supported by a recent case-control study that examined risk factors for stroke among Swedish men.⁴⁴ In multivariate analyses, controlling for other risk factors, smoking was related to increased risk of stroke (OR 1.74) whereas snus use was not (OR 0.87, 95% CI 0.41 to 1.83).

Given the inconsistencies in the results of these studies, it remains possible that snus users have a slightly increased cardiovascular risk as compared to never tobacco users, even after controlling for other confounding factors. However, all of the large studies of the effects of tobacco use on cardiovascular disease in Sweden are in agreement that "the use of smokeless tobacco (with snuff being the most studied variant) involves a much lower risk for adverse cardiovascular effects than smoking does".⁴⁵

Respiratory diseases

A Pubmed search did not identify any studies that specifically examined the effect of snus on respiratory diseases; similarly the IOM report did not address the effects of smokeless tobacco on respiratory illnesses.³ The reason for this is presumably that there is no plausible causal mechanism whereby smokeless tobacco could cause respiratory disease. A recent study of mortality in US smokeless users reported no increased risk of respiratory diseases in smokeless users.⁴² This contrasts heavily with the effect of continued smoking on chronic obstructive pulmonary disease, with 50% of elderly Swedish smokers developing the condition as compared with less than 20% of never smokers.⁴⁶

Diabetes

Bolinder³⁷ found that smokers had significantly higher fasting blood glucose values than never tobacco users whereas snus users were not significantly different from never users. Eliasson and colleagues⁴⁷ found that neither smoking nor snus use was associated with changed glucose tolerance or insulin concentrations. However, a more recent study by Persson⁴⁸ found an increased risk of (asymptomatic)

type 2 diabetes among both heavy smokers (25+ cigarettes per day) and heavy snus users (3+ cans per week), with significant odds ratios of 2.7 and 2.6, respectively, for these two groups as compared with non-tobacco users. It should be noted that this study specifically recruited men over 35 years old, 50% of whom had a family history of diabetes. The effects of snus on risks for diabetes are unclear and it may be that any effects are restricted to heavy users and/or those with a family history of diabetes.

Pregnancy

A Pubmed search did not identify any studies that had specifically examined the effects of snus use during pregnancy. However, given that animal studies have implicated nicotine as a cause of some of the widely known adverse effects of tobacco exposure during pregnancy (on both the health of the mother and healthy development of the fetus), it follows that snus use during pregnancy is likely to incur some of the risks associated with smoking during pregnancy.⁴⁹ The preliminary results of one study (as yet unpublished) have been presented at a conference earlier this year.⁵⁰ The study examined data from the Swedish Birth Register for women who delivered babies during 1999–2000. The study compared 789 snus users to 11 242 cigarette smokers and 11 500 women not using any tobacco. Smokers gave birth to babies weighing an average of 206 g (7.3 ounces) less than non-tobacco users. Snus users gave birth to babies weighing an average of 40 g (1.4 ounces) less than non-tobacco users. Snus users were also about twice as likely as non-tobacco users to deliver prematurely (perhaps partially explaining the slightly lower birth weight), and were more likely than both smokers and non-tobacco users to suffer pre-eclampsia. Clearly, the full results of this study and additional studies on this topic are required before coming to conclusions, particularly given the possibilities for confounding variables to cause small sized effects. However, given the known risks of nicotine in pregnancy, and the preliminary results of this unpublished study, it seems likely that snus use can cause adverse health effects in pregnancy and should not be promoted as safe for use in pregnancy. It would be a particular cause for concern were there to be evidence of increased snus use among women of reproductive age, without an equal or greater reduction in smoking in that group. Given that smoking during early pregnancy in Sweden has already declined from 31% in 1983 to 12% in 2000,³¹ it could be argued that the potential for snus to have a "positive" impact on smoking in pregnancy has similarly shrunk. It would seem as though Swedish women are on a positive trend towards tobacco-free pregnancies without snus, and that it would be best kept that way.

THE PATTERN OF NICOTINE USE IN SWEDEN OVER THE PAST CENTURY

Total consumption of snus and cigarettes in Sweden have changed dramatically over the past century, with the most pronounced changes occurring over the past 20 years when cigarette consumption has reduced significantly and at the same time snus consumption has risen significantly. Figure 2 provides only a crude snapshot of overall sales, that hides sex-specific changes and changes in the size of the population. Adult (over 14) cigarette consumption went from approximately 0.2 kg/person in the 1920s to 1.1 kg/person in 1970 and then down to 0.6 kg/person at the end of the 20th century. Across the same time points Snus consumption fell from 1.4 kg/person to 0.4 kg/person and then has increased again to 0.9 kg/person by 2000. Figure 2 also serves as a reminder of some of the other factors affecting cigarette consumption; the large drop in cigarette sales in 1997 was probably related to an 18% price increase in January of that

