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The nicotine market: An attempt to estimate the nicotine intake from various sources and the total nicotine consumption in some countries

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Tobacco—particularly smoked products—has been associated with great harm and growing public disapproval and can be expected to suffer in the marketplace. This situation has created opportunities for other less harmful nicotine-containing products such as smokeless tobacco and nicotine replacement products, which are gaining public support. Little is known about the level of nicotine intake in our society. Tobacco sales are known, but how much nicotine is extracted and actually absorbed by users is largely unknown. The present study is a first attempt to estimate uptake of nicotine from tobacco and nicotine replacement products and to map nicotine consumption in a few countries, with special emphasis on Sweden. Relevant pharmacokinetic studies for three types of nicotine-containing products (cigarettes, smokeless tobacco, and nicotine replacement products) were analyzed for bioavailable nicotine. Estimates of nicotine intake from each category were made. These were then multiplied by the amount consumed in the respective countries. Tobacco consumption statistics were usually from official records of taxed sales. In Sweden about 54% of all nicotine intake comes from smoked sources, 45% from nonsmoked tobacco, and 1.3% from nicotine replacement products. For men, 63% of the nicotine consumed comes from nonsmoked tobacco. Per-capita nicotine intake per year for adults aged 15 years or older is 3,321 mg for Austria, 3,043 mg for Sweden, 3,014 mg for Denmark, 2,955 mg for the United States, 2,244 mg for Norway, and 2,023 mg for Finland. Compared with cigarette smokers, snus users seem to have a somewhat higher daily intake (34 mg vs. 25 mg). The cleanest nicotine products, nicotine replacement products, represent a negligible part (about 1%) of the total nicotine consumption in most countries.

Introduction

Nicotine is probably the world's second most used drug after caffeine. About 1.3 billion people worldwide are smokers, and the rate is not declining (Shafey, Dolwick, & Guindon, 2003). Although smokers can be dependent on tobacco or nicotine, most users are likely unaware, or do not admit, that they are seeking nicotine, given that nicotine use without tobacco is exceptionally rare. Tobacco users, most commonly smokers, may not realize that nicotine to a large degree drives their desire for tobacco. However, most smokers probably do know that inhaling smoke into their lungs is unhealthy.

Observers widely believe that it is likely impossible to rid the world completely of nicotine in the near future (i.e., for some generations) (Crane, Blakely, & Hill, 2004; Gray & Boyle, 2003; Warner, Slade & Sweanor, 1997). Despite recent anti-tobacco activities by scientists and activists, worldwide tobacco consumption has not decreased at all (Shafey et al., 2003). The realistic but not optimal reaction to this insight has been to see that nicotine in a pure form could be part of the solution to the tobacco smoking problem. The possible dangers of nicotine itself seem to be dwarfed by the dangers associated with smoking tobacco (Gray & Boyle, 2003). In addition, a distinction has to be made between smoked and nonsmoked tobacco (Foulds, Ramström, Burke, & Fagerström, 2003). So far, pure nicotine has not been associated with increased risk of cancer and

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respiratory diseases. For cardiovascular diseases, pure nicotine is certainly less of a risk factor than is smoking, but, depending on the rate of uptake, nicotine probably poses some small risk (Asplund, 2003; Benowitz, 1999).

If one could entertain the unrealistic assumption that all tobacco users would rapidly switch to clean nicotine tomorrow, we would see an immediate effect on cardiovascular disorders and a delayed effect on respiratory and cancer disorders. In the future, the excess mortality from smoking, which currently runs at 5 million deaths per year (The World Bank, 1999), would almost be eradicated. If such a switch were strongly endorsed by health authorities, it would make nicotine look relatively safe and possibly increase its use. Such an increase would not, however, significantly decrease the positive effect on mortality because pure nicotine has only a small percentage of the harmful effects associated with cigarette smoking (Kozlowski, Strasser, Giovino, Erickson, & Terza, 2001). The more likely effect is that if smokers and potential smokers could not have easy access to cigarettes but instead could use pure nicotine, fewer would do so because nicotine without tobacco is not as reinforcing (Houtsmuller, Henningfield, & Stizer, 2003; Nemeth-Coslett, Henningfield, O'Keeffe, & Griffiths, 1987).

Smoking delivers nicotine fast and in a rewarding way. In addition, tobacco smoke contains other substances that have pharmacological effects, such as carbon monoxide, anabasine, acetaldehyde (Kunin, Latendresse, Gaskin, Smith, & Amit, 2000), and naphthoquinone (Castagnoli, Steyn, Petzer, Van der Schyf, & Castagnoli, 2001) that might be responsible for the monoamine oxidase inhibition seen in smokers (Fowler, Logan, Wang, & Volkow, 2003). Apart from these pharmacological factors, the sensory stimulation, package design, and behavioral features of tobacco and nicotine replacement product use differ and may contribute to the discrepancy in reinforcement power. With such an approach to the tobacco problem, we would need to realize that addiction would remain prevalent. Perhaps it would be easier to deal with the problem of addiction at a later time rather than try to solve the problem of mortality and addiction at the same time.

If pure nicotine is to become a part of the solution to the unparalleled health risks of tobacco smoking, its use needs to be monitored. Tobacco is no longer the only source of nicotine. The market for nicotine replacement products is increasing steadily in most countries. In 2002, nicotine replacement products sold globally for US$1.5 billion, and such products are no longer confined entirely to increasing the chance of a successful quit attempt; they also have a broader use. Several medical authorities have licensed nicotine replacement products for temporary abstinence and reduced smoking. However, long-term use after quitting smoking is likely to be more common than is use for reduced smoking or temporary abstinence.

The contribution of nicotine replacement products to the total amount of nicotine used is presently small but is increasing in most countries. The relative proportion could increase rapidly if nicotine intake from tobacco were to decrease because of, for example, increased awareness of the harmful effects from smoking, increased taxes, and social disapproval.

An assessment of the nicotine consumed from different sources can be carried out for simple descriptive reasons. It also can provide useful information to researchers in much the same way as when total alcohol consumption and its health effects are evaluated. Finally, it is essential to know the source of consumed nicotine, if a switch from the most contaminated delivery system (smoking) to cleaner systems were to be an active tobacco control strategy. This paper offers guidance on how to estimate actual nicotine intake from various nicotine-containing products. The proposed method is applied to an example country, Sweden, where a substantial proportion of all nicotine consumed already comes from sources other than cigarettes. Sweden will be compared with its Scandinavian neighbors and with the United States, all of which have a reasonably good tobacco control climate, and with Austria, which has a much less favorable tobacco control climate, according to a smokers' survey of EU countries (Fagerström, Boyle, Kunze, & Zatonski, 2001).

Method

Estimation of nicotine intake from cigarettes

The seemingly most straightforward measure to estimate nicotine intake from cigarettes is to use the yields obtained by the smoking machines under the ISO standard. However, this method does not replicate well actual human smoking patterns. Many scientists have strongly criticized this method (Jarvis, Boreham, Primatesta, Feyerabend, & Bryant, 2001). One criticism is that the puff volumes are not representative and the method does not account for vent hole blocking (Kozlowski et al., 1998). Smokers vary their nicotine intake considerably from one cigarette to another. For example, several milligrams of nicotine might be inhaled after a long time without smoking (e.g., after a night's sleep), whereas only a fraction of a milligram is inhaled when the smoker lights up a cigarette and forgets it in the ashtray. Several studies have examined actual nicotine intake from cigarettes. Benowitz, Jacob, Fong, and Gupta (1994) found that heavy smokers (defined as those

smoking 29 cigarettes/day [CPD] on average) smoking their own brand consumed 2.5 mg per cigarette. An intake of 1.4 mg was found for another group of heavy smokers (defined as those smoking 27 CPD; Benowitz, Zevin, & Jacob, 1997). In a study of lighter smokers, a 1 mg per cigarette intake was found (Benowitz, Porchet, Sheiner, & Jacob, 1988). In a Japanese study the estimated nicotine intake per cigarette was 0.6, 1.0, 1.3, and 1.4 mg for smokers of ultralow-, low-, medium-, and high-yield cigarettes, respectively (Ueda et al., 2002). A reasonable rough estimate across cigarettes, smokers, and countries may be a nicotine intake of 1.5 mg per cigarette.

The present paper looks at nicotine consumption in Sweden in some detail. Andersson, Kazemi Vala, and Curvall (1997) reported on two studies. All subjects smoked their usual brand for 1 week. At the end of the week, on day 6, urine samples were collected for 24 hr to assess nicotine intake. In one study, 47 subjects smoked, on average, 18.5 CPD. They had an intake of 24.7 mg of nicotine over 24 hr, which gives a nicotine intake of 1.3 mg per cigarette. In the other study, which involved 91 smokers averaging 17.4 CPD, a daily nicotine intake of 24.5 mg was found, corresponding to an absorption of 1.4 mg from each cigarette. The number of cigarettes per day was slightly over the national average of 14, but the nicotine intake per cigarette did not differ with cigarettes per day in these two studies. The average over both studies is 1.4 mg, which is used as the estimate of nicotine intake per cigarette in the present paper.

Estimation of nicotine intake from smoked tobacco other than cigarettes

Data for consumption of pipe, cigar, and roll-your-own cigarette smoking is given mostly in tonnes. To roughly estimate the nicotine intake from these types of tobacco, an assumption has been made that 1 g of tobacco yields 1.5 mg of absorbed nicotine. This assumption is based on the findings that one cigarette weighs, on average, a little less than 1 g and yields 1.4 mg of nicotine. Therefore, a round figure of 1.5 mg, slightly higher than that from a cigarette, seems realistic to expect from consumption of 1 g of smoking tobacco.

Estimation of nicotine intake from smokeless tobacco

The characteristics of the smokeless tobacco products available worldwide seem to vary more than those of cigarettes, which are manufactured under relatively standardized procedures. For example, the nicotine concentration in U.S. smokeless tobacco products varies from 0.47% to 3.43% of dry weight (Fant, Henningfield, & Nelson, 1999). However, the nicotine obtained from a product is influenced by a number of factors, such as pH (which determines the ratio of unprotonated to free nicotine), the size (weight) of the actual dose, the time it is kept in the mouth, the size of the product surface exposed to the oral mucosa, how much the pinch is handled in the mouth, where it is positioned, and whether a tissue material is between the tobacco and the mucosa (Fant et al., 1999). The bioavailability of nicotine consumed from smokeless tobacco is lower than that from smoking because considerable amounts of nicotine are swallowed in the former case (Svenson, 1987). A large part of the swallowed nicotine, approximately 25%–30%, will not be available as nicotine in the bloodstream beyond the liver because it is metabolized to cotinine (Holm, Jarvis, & Russell, 1992). From a relatively large dose (2.5 g) of smokeless tobacco held in the mouth for 30 min, 3.6 mg of nicotine was absorbed (Benowitz et al., 1988).

The focus in the present paper is on the Swedish smokeless products usually referred to as snus. A dose of portion-packed snus is typically 1 g, and a dose of loose snus, which no longer is as common, is 1–2 g (Andersson, Björnberg, & Curvall, 1994). Today, when the majority of snus is consumed in small, ready-made sachets, the dose varies from 0.3 g to 1 g wet weight. A study of relatively heavy snus users found that 23 users of portion-packed snus consumed, on average, 15.7 g of snus per day. The same amount, 15.7 g per day, was registered for a group of 22 loose-snus users. The nicotine intakes among the portion-packed and loose-snus users were 32.2 and 34.0 mg, respectively. This gives an intake per gram of portion-packed snus of 2.0 mg; the intake per gram of loose snus is 2.2 mg (Andersson et al., 1994).

A recent pharmacokinetic study comprising 12 snus-using men found the following nicotine contents in four different portion-packed snus products: General 1 g, the most popular product, contained 8.8 mg of nicotine; Catch Licorice 1 g, 7.0 mg of nicotine; Catch Mini 0.5 g, 4.5 mg of nicotine; and Catch Dry Mini 0.3g, 4.8 mg of nicotine. The mean amounts of nicotine extracted from General, Catch Licorice, Catch Mini, and Catch Dry Mini were 2.7, 1.6, 2.0, and 1.1 mg, respectively. With an assumption of 55% bioavailability for buccal nicotine products (which has been found repeatedly), the bioavailable dose should be 1.5 mg for General, 0.9 mg for Catch Licorice, 1.1 mg for Catch Mini, and 0.6 for Catch Dry Mini (Lunell, 2005). The bioavailability of nicotine per gram of snus is, hence, approximately 1.5 mg for General, 1.2 mg for Catch Licorice, 2.2 mg for Catch Mini, and 2.0 mg for Catch Dry Mini. Results from these two studies

(Andersson et al., 1994; Lunell, 2005) suggest that the actual nicotine intake from a gram of snus varies between 1.2 and 2.2 mg. The absorption may be somewhat higher from the less common loose snus; however, it is argued here that the estimate of nicotine intake should be based on the more commonly used portion bags. The most popular portion-packed brand, General, is supposed to give an uptake of 1.5 mg/gram, and this figure is used here as the estimate of nicotine intake from snus.

Estimation of nicotine dose from nicotine replacement products

With medications, such as nicotine replacement products, one would assume that estimating nicotine intake would be easier because these products are labeled carefully by regulatory authorities. That is not the case, however. The labeling is inconsistent in the sense that some products (i.e., patches) are labeled according to actual nicotine intake, whereas others (e.g., chewing gum) are labeled according to the product's nicotine content. In addition, as with tobacco, nicotine replacement products vary with respect to usage patterns. For example, gum can be chewed for a long or short time before it is discarded. Further, a residue (at least 0.5 mg) of nicotine always remains, even after long, intense chewing. The largest variability in dose obtained is from the nicotine inhaler, which in principle has only buccal absorption of nicotine (Bergström, Nordberg, & Lunell, 1995). The nominal nicotine content is 10 mg, but more than 2–4 mg seldom is extracted. Large variability in nicotine intake exists depending on the frequency and intensity of the inhalation. The amount of nicotine obtained from nicotine patches is almost identical to the declared dose on the package, although lower levels have been reported (Benowitz, 1995). Regardless, more and better data are available for giving good estimations of nicotine intake from nicotine replacement products.

A general rule with buccal products is that the bioavailability of extracted nicotine is 50%–60% (Molander, Lunell, Andersson, & Kuylenstierna, 1996). The nasal spray also has a bioavailability between 55% and 60% (Benowitz et al., 1997). For nicotine from patches, the bioavailable dose is close to 100% (Benowitz, 1995). For the sake of simplicity, the amount of nicotine consumed from patches is therefore set as the declared dose. For the other products, the following rounded estimates are used: 4-mg gum=2 mg absorbed nicotine, 2-mg gum=1 mg, 10-mg inhaler=2 mg, 2-mg sublingual tablet=1 mg, 4-mg lozenge=2 mg, 2-mg lozenge=1 mg, 1-mg lozenge=0.5 mg, and 1-mg nasal spray=0.5 mg. Table 1 lists the nicotine intake from tobacco and nicotine replacement products.

Table 1. Estimated nicotine intake from nicotine replacement and tobacco products.

Product	Nicotine intake (mg)
Cigarettes	1.4
Other smoked tobacco	1.5/g
Smokeless tobacco (snus)	1.5/g
Gum	
2 mg	1
4 mg	2
Lozenge	
1 mg	0.5
2 mg	1
4 mg	2
Sublingual tablet (2 mg)	1
Inhaler (10 mg)	2
Nasal spray (1 mg)	0.5
Patches	As labeled on package

Consumption and population data

Data for tobacco consumption for the Nordic countries in 2002 was obtained from Nordic Tobacco Statistics (VECA HB, 2003), for Austria in 2002 from Statistics Austria (2003), and for the United States in 2001 from the U.S. Department of Agriculture Economic Research Service (2002). The data for nicotine replacement consumption in Sweden in 2003 was provided by a special statistics report from Apoteket AB (data on file with author). Figures for whole populations and those aged 15 years or older were taken from *Tobacco Control Country Profiles* (Shafey et al., 2003).

Results

In 2002, 4,861 tonnes or 4,861,000,000 g of tobacco were sold as 7,478,000,000 cigarettes in Sweden. A total of 946 tonnes of tobacco for cigars, pipes, and roll-your-own cigarettes was consumed during 2002. The nicotine intake from this source of tobacco is estimated at 1,400,000,000 mg. Thus, total smoked nicotine consumption is 11,800,000,000 mg, the per-capita consumption for the whole population (8,940,000) is 1,328 mg/year, and the per-capita consumption for those aged 15 years or older (7,329,000) is 1,621 mg/year. Daily consumption is 3.6 mg for the whole population and 4.4 mg for those aged 15 years or older. Calculated for only daily smokers (17.8% of 7,329,000 adults=1,304,500; VECA HB, 2003), the estimates are 9,102 mg/year and 24.9 mg/day.

The weight of tobacco sold as snus was 6,752 tonnes or 6,752,000,000 g. The per-capita consumption is 1,133 mg/year for the whole population and 1,400 mg/year for those aged 15 years or older. These estimates give a per-day consumption of 3.1 mg for the whole population and of 3.8 mg for the adult population. Calculated for daily snus users (20% of the adult men [733,000 users] and 2% of the women

[73,300 users], or 806,300 in total; Ramström, 2002), the estimated per-capita intake is 12,566 mg/year and 34.4 mg/day. Table 2 shows the annual total, per-capita, and per-capita user intake as well as the daily per-capita user intake.

A total of 54% (1,328 mg) of all nicotine consumed from tobacco originates from smoked sources. Nicotine replacement therapy contributes with approximately 1.3% (33 mg), and the remaining (1,133 mg) comes from snus. However, the picture is not complete until gender is added because snus use is almost entirely a male phenomena (20% male vs. 2% female; Ramström, 2002). When disregarding nondaily use and daily combined use of snus and cigarettes, it can be estimated that 90% of all smokeless tobacco (9,115,200,000 mg) is consumed by males. For nicotine intake from smoked sources, 46% is consumed by males (5,468,000,000 mg/year vs. 6,419,000,000 mg/year for females). Thus the total amount of nicotine consumed by males each year is 14,583,200,000 mg, of which 62.5% comes from snus. Total annual nicotine consumption for females is 7,432,000,000 mg, of which 1,013,000,000 mg come from snus (13.5%). For this calculation, a smoking prevalence of 16.3% for men and 19.3% for women was used (VECA HB, 2003).

Table 3 compares Sweden with the other countries. In Denmark, 7,205,000,000 cigarettes were consumed, which amounts to a nicotine intake of 10,000,000,000 mg. Among the Danish population (4,370,000) aged 15 years or older, the per-capita consumption is 2,319 mg/year and 6.3 mg/day. In Denmark, a considerable amount of other smoked tobacco (1,982 tonnes) was consumed. This amounts to 2,900,000,000 mg of nicotine consumed or 683 mg/year and 1.9 mg/day. In addition, 36 tonnes of smokeless tobacco was consumed, resulting in a nicotine intake of 54,000,000 mg or a per-capita rate of 12 mg/year. The consumption of nicotine replacement products in the other Nordic countries is not known with precision, but the market penetration is higher in Sweden, where these products represent 1.3% of all nicotine consumed. In Finland, Denmark, Austria, and Norway, nicotine replacement products can be assumed to add at most about 1%. Without losing too much precision, one can therefore calculate the total nicotine consumed in the other countries from only tobacco products. In summary, the total daily per-capita consumption in Denmark and Sweden is 8.2 mg. The corresponding figures for Norway, Finland, Austria, and the United States are 6.1, 5.5, 8.9, and 8.1 mg, respectively.

Discussion

This paper is probably the first attempt to estimate effective nicotine intake in populations and users. The estimations were intended to determine the amount of nicotine that actually enters the blood-stream of users and thus could have a pharmacological effect. Apart from estimating the nicotine intake in whole populations, nicotine consumption also was broken down by users and sources of nicotine.

In Sweden, a unique pattern of nicotine consumption was found in which 46% of nicotine originated from unburned sources. The contribution from nicotine replacement products is small (1.3%), despite Sweden having the world's second highest per-capita consumption, except for Iceland (personal communication, Jörgen Johnsson Pharmacia Consumer Healthcare, 2001). Thus the Swedish product snus accounts for almost all unburned nicotine intake. Among the nicotine replacement

Table 2. Nicotine intake in Sweden (in milligrams).

	Total intake per year	Annual per-capita intake	Annual intake per user	Daily intake per user
Tobacco				
Cigarettes	10,469,000,000	1,170	8,025	
Pipe, cigars, roll-your-own	1,419,000,000	159	1,087	
Total smoked	11,888,000,000	1,328	9,102	24.9
Snus	10,128,000,000	1,133	12,566	34.4
Total tobacco	22,016,000,000	2,462		
Nicotine replacement				
Patch (24 hr)	31,000,000	3.5	—	—
Patch (16 hr)	12,000,000	1.3	—	—
Gum (2 mg)	146,000,000	16.3	—	—
Gum (4 mg)	87,000,000	9.7	—	—
Lozenge (1 mg)	4,400,000	0.5	—	—
Lozenge (2 mg)	2,800,000	0.3	—	—
Sublingual (2 mg)	11,000,000	1.2	—	—
Inhaler (10 mg)	2,600,000	0.3	—	—
Nasal spray (1 mg)	32,000	0.0	—	—
Total nicotine replacement	296,832,000	33.1	—	—
Total tobacco and nicotine replacement	22,312,832,000	2,495.1	—	—

Note. Dash represents values not known.

Table 3. Nicotine intake for the population aged 15 years or older (milligrams).

		Nicotine intake (mg)		
Country	Amount per year	Per year	Per-capita per year	Per-capita per day
Sweden				
Cigarettes	7,478,000,000 pieces	10,400,000,000	1,447	3.9
Pipe, cigars, roll-your-own	946 tonnes[a]	1,400,000,000	196	0.5
Smokeless tobacco	6,752 tonnes[a]	10,128,000,000	1,400	3.8
Total			3,043	8.2
Denmark				
Cigarettes	7,205,000,000 pieces	10,000,000,000	2,319	6.3
Pipe, cigars, roll-your-own	19,832 tonnes[a]	2,970,000,000	683	1.9
Smokeless tobacco	36 tonnes[a]	54,000,000	12	0
Total			3,014	8.2
Finland				
Cigarettes	4,295,000,000 pieces	6,900,000,000	1,626	4.5
Pipe, cigars, roll-your-own	1,122 tonnes[a]	1,680,000,000	397	1.1
Smokeless tobacco	Not on sale			
Total			2,023	5.6
Norway				
Cigarettes	2,570,000,000 pieces	3,800,000,000	1,075	2.9
Pipe, cigars, roll-your-own	2,367 tonnes[a]	3,500,000,000	989	2.7
Smokeless tobacco	431 tonnes[a]	646,000,000	180	0.5
Total			2,244	6.1
United States				
Cigarettes	410,000,000,000 pieces	57,000,000,000	2,588	7.1
Pipe, cigars, roll-your-own	3,849 tonnes[a]	5,700,000,000	26	0.1
Smokeless tobacco	50,400 tonnes[a]	75,600,000,000	341	0.9
Total			2,955	8.1
Austria				
Cigarettes	15,274,000,000 pieces	21,300,000,000	3,174	8.6
Pipe, cigars, roll-your-own	653 tonnes[a]	980,000,000	145	0.3
Smokeless tobacco	9 tonnes[a]	13,000,000	2	0.0
Total			3,321	8.9

Note. Population data from *Tobacco Control Country Profiles* (Shafey et al., 2003).
[a]Tonnes are metric tonnes.

products, the nicotine gum alone is responsible for 80% of the nicotine intake from these products, and nicotine patches contribute 15%. In most other countries, the patches have a larger share, but given Sweden's good antismoking climate (Fagerström et al., 2001), the gum is likely to be used more for long-term substitution and possibly for temporary abstinence and reduced smoking.

The findings also indicate that snus users have a somewhat higher nicotine intake (34.4 mg), compared with smokers (24.9 mg). However, other studies looking at blood nicotine concentrations in Swedish snus users and smokers have found identical (Holm et al., 1992) or quite similar nicotine concentrations (Andersson, Axell, & Curvall, 1995; Andersson et al., 1997). The 24.9-mg intake from cigarette smokers may not be representative of smokers in other countries because Sweden has a large proportion of ex-smokers, more woman than men smoke, and many current former highly dependent male smokers use snus.

When nicotine intake was broken down by sex, the present analysis found that men use almost twice as much nicotine as women and that, for men, 62.5% of all nicotine intake come from snus. Nicotine replacement consumption was not counted because its use by sex is not known, and its general share is only 1.3%.

In a comparison of the Scandinavian countries, Sweden and Denmark appear to have relatively similar nicotine intakes (3,043 and 3,014 mg, respectively, per capita per year for the population aged 15 years or older). Norway has a lower per-capita nicotine intake (2,244 mg), but roll-your-own cigarettes represent a big share of the market, and nicotine absorption from this source is difficult to estimate. If anything, the 1.5-mg estimate might be on the low side. Also, some border trade exists in which Norwegians buy cheaper cigarettes in Sweden. Finland clearly has the lowest nicotine intake (2,023 mg per-capita per year for the adult population). The low level of nicotine intake in Finland is most likely related to the high number of ex-smokers among the men and the fact that Finnish woman have not yet taken up smoking to the same degree as in the other Scandinavian countries. The U.S. data, which are from 2001, are close to those of Sweden and Denmark. Austria, the country with the least developed antismoking climate (Fagerström et al., 2001) of these countries has the highest consumption (3,321 mg per year for the adult population).

It would be of great interest not only to map current nicotine intake in populations and subpopulations but also to follow it over time. Where good statistics exist, one could go back in time and do

these estimations for nicotine intake. However, valuable data from various nicotine sources can be found almost only in Sweden and the United States because in most other countries cigarettes represent nearly the entire nicotine intake. An interesting and maybe disappointing finding is that nicotine replacement products, as currently marketed, have not been able to gain a bigger share of the nicotine market. If nicotine consumption is looked at by source and followed over time, it should be of great interest to see how consumption reflects on morbidity or mortality, particularly if the cigarette monopoly on nicotine intake could be broken in countries other than Sweden.

A study such as the present one has many limitations. It is difficult to estimate correctly the actual nicotine intake from various sources. Most important would be to estimate correctly the intake from cigarettes because this is the dominant source for nicotine consumption. The study relied on here for the 1.4-mg intake estimate per cigarette had limitations. Only men were included, and their average cigarette consumption was somewhat higher than the national average. A study using a representative sample of smokers, allowing them to smoke ad libitum in their normal environment, and making an accurate assessment of cigarettes smoked would solve most of the problems associated with estimating nicotine intake from cigarettes as well as intake from other chemicals.

Also, in the estimation of nicotine intake from snus, the data are far from perfect. The intake was estimated from the most popular brand, General portion-packed. However, because the brand has less than 50% market share, other brands, most with a lower nicotine intake (Lunell, 2005), would be more common. Further, the information about actual consumption of tobacco products in a country is imperfect. For tobacco products, only those that have been taxed are registered, which does not include, for example, duty-free buying, legal border trade, and products smuggled into the country.

Numerous other limitations are associated with the assumptions and calculations presented. The results should thus be seen as a first crude attempt to assess nicotine intake, and the study could, it is hoped, serve as a springboard for raising interest in the topic and lead to more and better studies.

Does the present study offer any alternatives to the use of prevalence as a measure for nicotine intake? Prevalence by itself can be used as a starting point for estimating nicotine intake. When using the number of daily smokers in Sweden and multiplying it by average daily cigarette consumption $\times$ 1.4 mg $\times$ 365 days, we calculated a yearly intake of 1,048 mg per capita compared with the 1,170 mg obtained from sales data (Table 2). The use of prevalence data may give lower values for nicotine consumption because the number of cigarettes smoked may be underestimated and the number of daily smokers may be under-reported. It is thus believed that the methodology presented here, although rough and preliminary, is superior to prevalence measures if the objective is to measure nicotine intake.

The present study has yielded a benchmark for nicotine intake in individuals and populations. The study showed that (a) relatively large variability exists in nicotine intake among countries, (b) the dirtiest vehicle (smoked tobacco) in most countries accounts for more than 90% of nicotine intake, (c) nicotine replacement products still have a negligible role in total nicotine intake, and (d) Sweden is very different from other countries, with almost half of all nicotine consumed originating from unburned sources. Swedish males have the lowest rate of smoking-related morbidity in Europe (Peto, Lopez, Boreham, & Tuhn, 1996), the lowest smoking prevalence, and the highest nicotine intake from unburned sources. Whether these three factors are related and even causally so is currently much debated (Foulds et al., 2003; Tomar, Connolly, Wilkenfeld, & Henningfield, 2003).

References

Andersson, G., Axell, T., & Curvall, M. (1995). Reduction in nicotine intake and oral mucosal changes among Swedish oral moist snuff after switching to a low-nicotine product. *Journal of Oral Pathology & Medicine*, *24*, 244–250.

Andersson, G., Björnberg, G., & Curvall, M. (1994). Oral mucosal changes and nicotine disposition in users of Swedish smokeless tobacco products: A comparative study. *Journal of Oral Pathology & Medicine*, *23*, 161–167.

Andersson, G., Kazemi Vala, E., & Curvall, M. (1997). The influence of cigarette consumption and smoking machine yields of tar and nicotine on the uptake and oral mucosal lesions in smokers. *Journal of Oral Pathology & Medicine*, *26*, 117–123.

Asplund, K. (2003). Smokeless tobacco and cardiovascular disease. *Progress in Cardiovascular Diseases*, *45*, 383–394.

Benowitz, N. L. (1995). Clinical pharmacology of nicotine patches. *European Journal of Pharmaceutics and Biopharmaceutics*, *41*, 168–174.

Benowitz, N. L. (1999). Snuff, nicotine and cardiovascular disease: Implications for tobacco control. *Journal of the American College of Cardiology*, *34*, 1791–1793.

Benowitz, N. L., Jacob, P., Fong, I., & Gupta, S. (1994). Nicotine metabolic profile in man: Comparison of cigarette smoking and transdermal nicotine. *Journal of Pharmacology and Experimental Therapeutics*, *268*, 296–303.

Benowitz, N. L., Porchet, H., Sheiner, L., & Jacob, P., III. (1988). Nicotine absorption and cardiovascular effects with smokeless tobacco use: Comparison with cigarettes and nicotine gum. *Clinical Pharmacology and Therapeutics*, *44*, 23–28.

Benowitz, N. L., Zevin, S., & Jacob, P., III. (1997). Sources of variability in nicotine and cotinine levels with use of nicotine nasal spray, transdermal nicotine, and cigarette smoking. *British Journal of Clinical Pharmacology*, *43*, 259–267.

Bergström, M., Nordberg, A., & Lunell, E. (1995). Regional disposition of inhaled 11C-nicotine vapor in the human airway visualized by positron emission tomography. *Clinical Pharmacology and Therapeutics*, *57*, 309–317.

Castagnoli, K. P., Steyn, S. J., Petzer, J. P., Van der Schyf, C. J., & Castagnoli, N. (2001). Neuroprotection in the MPTP Parkinsonian C57BL/6 mouse model by a compound isolated from tobacco. *Chemical Research in Toxicology*, *14*, 523–527.

Crane, J., Blakely, T., & Hill, S. (2004). Time for major road-works on the tobacco road? *The New Zealand Medical Journal*, *1190*, U801.

Fagerström, K. O., Boyle, P., Kunze, M., & Zatonski, W. (2001). The anti-smoking climate in EU countries and Poland. *Lung Cancer*, *32*, 1–5.

Fant, R. V., Henningfield, J. E., & Nelson, R. A. (1999). Pharmacokinetics and pharmacodynamics of moist snuff in humans. *Tobacco Control*, *8*, 387–392.

Foulds, J., Ramström, L., burke, M., & Fagerström, K. (2003). Effect of smokeless tobacco (snus) on smoking and public health in Sweden. *Tobacco Control*, *12*, 349–359.

Fowler, J. S., Logan, J., Wang, G. J., & Volkow, N. D. (2003). Monoamine oxidase and cigarette smoking. *Neurotoxicology*, *24*, 75–82.

Gray, N., & Boyle, P. (2003). The future of the nicotine addiction market. *The Lancet*, *362*, 845–846.

Holm, H., Jarvis, M. J., & Russell, M. A. H. (1992). Nicotine intake and dependence in Swedish snuff takers. *Psychopharmacology*, *108*, 507–511.

Houtsmuller, E. J., Henningfield, J. E., & Stitzer, M. (2003). Subjective effects of the nicotine lozenge: Assessment of abuse liability. *Psychopharmacology*, *167*, 20–27.

Jarvis, M. J., Boreham, R., Primatesta, P., Feyerabend, C., & Bryant, A. (2001). Nicotine yield from machine-smoked cigarettes and nicotine intake in smokers: Evidence from a representative population survey. *Journal of the National Cancer Institute*, *93*, 134–138.

Kozlowski, L. T., Metha, N. Y., Sweeney, C. T., Schwartz, S. S., Vogler, G. P., Jarvis, M. J., & West, R. J. (1998). Filter ventilation and nicotine content of cigarettes from Canada, the United Kingdom, and the United States. *Tobacco Control*, *7*, 369–375.

Kozlowski, L. T., Strasser, A. A., Giovino, G., Erickson, P. A., & Terza, J. V. (2001). Applying the risk/use equilibrium: Use medicinal nicotine now for harm reduction. *Tobacco Control*, *10*, 201–203.

Kunin, D., Latendresse, M. W., Gaskin, S., Smith, B. R., & Amit, Z. (2000). Pre-exposure effects of nicotine and acetaldehyde on conditioned taste aversion induced by both drugs. *Pharmacology, Biochemistry, and Behavior*, *66*, 695–699.

Lunell, E., & Lunell, M. (2005). Steady-state nicotine plasma levels following use of four different types of Swedish snus compared with 2-mg Nicorette chewing gum: a crossover study. *Nicotine & Tobacco Research*, *7*, 397–403.

Molander, L., Lunell, E., Andersson, B., & Kuylenstierna, F. (1996). Dose related and absolute bioavailability of nicotine from a nicotine vapor inhaler. *Clinical Pharmacology and Therapeutics*, *59*, 394–400.

Nemeth-Coslett, R., Henningfield, J. E., O'Keeffe, M. K., & Griffiths, R. R. (1987). Nicotine gum: Dose related effects on cigarette smoking and subjective ratings. *Psychopharmacology*, *92*, 424–430.

Peto, R., Lopez, A. D., Boreham, J., & Tuhn, M. (1996). Mortality from smoking worldwide. *British Medical Bulletin*, *52*, 12–21.

Ramström, L. (2002, October). *Patterns of use: A gate leading to smoking, or a way o give up?* Paper presented at the fourth annual meeting of the Society for Research on Nicotine and Tobacco–Europe, Santander, Spain.

Shafey, O., Dolwick, S., & Guindon, G. E. (2003) *Tobacco control country profiles* (2nd ed.). Atlanta, GA: American Cancer Society.

Statistics Austria. (2003). Austrian tobacco statistics, Table 23.12. Retrieved November 30, 2004, from www.statistik.at/cgi-bin/jahrbuch.pl?kapitel=23&sprache=d

Svenson, C. K. (1987). Clinical pharmacokinetics of nicotine. *Clinical Pharmacokinetics*, *12*, 30–40.

Tomar, S. L., Connolly, G., Wilkenfeld, J., & Henningfield, J. (2003). Declining smoking in Sweden: Is Swedish Match getting the credit for Swedish tobacco control's efforts? *Tobacco Control*, *12*, 368–371.

Ueda, K., Kawachi, I., Nakamura, M., Nogami, H., Shirokawa, N., Masui, S., Okayama, A., & Oshima, A. (2002). Cigarette nicotine yields and nicotine intake among Japanese male workers. *Tobacco Control*, *11*, 55–60.

U.S. Department of Agriculture Economic Research Service. (2002). U.S. tobacco statistics. Retrieved November 30, 2004, from http://mannlib.cornell.edu/reports/erssor/speciality/tbs-bb/2003/tbs2003.pdf

VECA HB. (2003). Nordic tobacco statistics. Retrieved from www.statveca.com

Warner, K. E., Slade, J., & Sweanor, D. (1997). The emerging market for long term nicotine maintenance. *The Journal of the American Medical Association*, *278*, 1087–1092.

The World Bank. (1999). *Curbing the epidemic*. Washington, DC: Author.