

# From snus to abuse – prejudice or fact?

The notion that snus or e-cigarettes are gateways to smoking or other intoxicants is beguiling. On closer inspection, however, it is difficult to identify the mechanisms that might explain a causal association between various intoxicants and products containing nicotine.

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The concept of «gateway» is used to illustrate a process by which the use of «soft» drugs such as tobacco, alcohol or cannabis, leads to the use of «hard» drugs such as cocaine or heroin (1). The term is also used to argue that the use of certain nicotine products, such as moist tobacco (snus) or e-cigarettes/nicotine vaporisers, leads to cigarette smoking (2). Nicotine and tobacco products thus occupy a unique position: they are assumed to constitute gateways to «harder» substances, while less harmful nicotine and tobacco products may serve as gateways to more harmful nicotine and tobacco products.

Given the concern that the use of less harmful substances may lead to the use of more harmful substances, the idea of a gateway has often been used by authorities and public-health agencies to underpin restrictive attitudes towards «soft» drugs. For example, the reports from the Norwegian Institute of Public Health on the harmful effects of snus and e-cigarettes/nicotine vaporisers express concern for a possible gateway between these products and smoking (3, 4).

## Background

The origin of the gateway idea is unknown, but must be seen in the context of the increasing concern with the spread of marihuana in the USA in the early 20th century (5). The possibility that marihuana could lead to heroinism was discussed in relation to the introduction of a tax on marihuana in 1937, but was rejected by the director of the US drugs enforcement agency at that time. In a review of the level of penal sanctions for drug offences eleven years later, however, the same man claimed that such an association definitely existed, despite the absence of any scientific evidence (6).

In the scientific community, the gateway idea has been discussed since the mid-1970s. The main concern has been that cannabis might lead to the use of «hard» and/or illicit

substances, such as cocaine and heroin (7). An article by the French-born professor Denise Kandel (1933–), published in *Science* in 1975, was of particular importance. She argued that «... legal drugs are necessary intermediates between non-use and marihuana» and that «Marihuana, in turn, is a crucial step on the way to other illicit drugs» (8).

Like cannabis and alcohol, cigarettes were also regarded as a possible gateway to «hard» substances, although somewhat less important than cannabis. However, the interest in the role of nicotine products as gateways increased in pace with the growing use of snus and chewing tobacco in the USA in the late 1980s (9), and later with the introduction of e-cigarettes/nicotine vaporisers from the mid-2000s.

Although the above quotes from Denise Kandel strongly suggest a *causal* association between the use of various substances, this and other articles from the same period (10, 11) emphasise that such an association must be understood as being of an *associative* nature. In later publications, however, the idea of a causal association is formulated explicitly (12, 13). For example, in 2003 Denise Kandel wrote that the «gateway» hypothesis presupposes that use of a given substance normally precedes another («sequencing»), that use of the first substance raises the likelihood of use of the second («association») and that use of the first substance is in fact the cause of the use of the second («causation») (8).

## Methodological limitations

Irrespective of whether the investigation concerns gateways between various drugs or between different nicotine or tobacco products, it will run into a fundamental problem in attempting to establish that the use of an intoxicant or drug is the *cause* of later use of another. This problem occurs with the use of both cross-sectional and panel data.

The most common solution is to attempt to control for all relevant background variables, which is difficult for obvious reasons. Results from studies that have gone to great lengths to address this problem provide little

support to the gateway hypothesis for nicotine and tobacco products (14–16), as well as for intoxicants in general (14, 17, 18).

Another solution to controlling for social and biological background variables is to undertake twin studies. For example, a Finnish twin study found that taking up smoking was positively associated with taking up cannabis (19). A reasonable objection, however, would be that differences in smoking behaviour between twins indicate that they are not identical with regard to relevant background variables, and that this instead reflects social or psychological differences between the twins (20). Moreover, it is common to use tobacco when smoking cannabis, and controlling for this factor is difficult.

A third, less technical and more theoretical solution involves searching for possible explanatory mechanisms that could render a causal association more likely. For example, it could be that the use of e-cigarettes teaches the body certain motoric and sensory patterns that facilitate smoking of traditional cigarettes. As regards gateways between various intoxicants, some have argued that the use of illegal substances is a ticket to communities where other illegal substances are available (17), or that experimentation with cannabis may cause other intoxicants to appear less risky (21).

In recent years, however, Kandel and collaborators have published a number of articles in which they use studies on mice to argue that certain intoxicants, such as nicotine, predispose the brain to addiction to other substances, such as cocaine. A key precondition, however, is that the intake of nicotine must begin well in advance and continue in parallel to the intake of cocaine (22).

## Theoretical problems

Despite the availability of good-quality data and robust models, the idea of a gateway is riddled with a number of theoretical problems. When investigating possible gateways between substances, the first objection would be that there is no consensus on the definition of «soft» and «hard» drugs. For example, many would disagree with the



Illustration: Ørjan Jensen/Superpop

categorisation of cannabis as «harder» than alcohol (23). Similarly, it is unclear what should be seen as the initial gateway substance, and this leads to an infinite regression. If alcohol and tobacco smoking both are gateways, why not sugar?

A related limitation is that people do not necessarily start using «soft» drugs before turning to «hard» ones, but that the order of use reflects local patterns of use and the local market situation (24). In addition, if a substantial gateway effect exists, we should find a positive correlation between the use of different substances over time. Often, this is not the case. For example, we find that the use of cocaine among young people increased relatively strongly in the late 1990s, while the prevalence of smoking decreased (25). This does not lend support to the idea of predisposition.

Furthermore, the arrow may point in the opposite direction. For example, the increasing use of snus in Norway in recent decades has been accompanied by a marked decrease in tobacco smoking. If there is an association between these two, it appears more likely that snus displaces tobacco smoking than that snus helps recruit tobacco smokers. Nor has the prevalence of snus helped resurrect the use of old-fashioned dry snuff, to provide a historical example.

As regards the idea of biochemical predisposition, this may in theory explain why some people become addicted to a new addictive substance, but it cannot explain why somebody chose to try the substance in the first place. Naturally, this is not a problem in studies of mice.

Nor can predisposition explain a possible transition between different nicotine and tobacco products, since the intoxicant in any case is nicotine. To the extent that nicotine increases the tolerance and urge for more nicotine, users of snus or e-cigarettes/nicotine vaporisers can simply increase their nicotine intake by using snus or e-cigarettes/nicotine vaporisers, without having to resort to tobacco smoking. A possible causal association, if any, must be wholly or partly attributable to other conditions.

Perhaps the strongest objection is that in studies that investigate the use of «hard» drugs, the observed correlation is a direct consequence of examining a selected group, i.e. persons who have used «hard» drugs. If we instead investigate the proportion of users of «hard» drugs among those who at any point in time have used «soft» drugs, the association will naturally be negligible (26). Similarly, this will be a problem if we investigate the effect of snus or e-cigarettes/nicotine vaporisers on smoking.

Does this mean that there can be no possible causal associations between different intoxicants and nicotine and tobacco products? Obviously not. My concern is that in the absence of unambiguous empirical observations, such associations must be based on sound reasons. This is also required in order to adequately model any causal associations. To date, no convincing explanations exist of a causal association between the use of snus and e-cigarettes and smoking. In this respect, the notion of a «gateway» is redundant.

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