

Gateway effects and electronic cigarettes: a response to J-F Etter

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Competing interests: None

This is a response to an essay by J-F Etter published in Addiction in 2017 (see reference #1). It was sent to Addiction which offered a 500 word letter. The full response is posted here)

Etter's valuable contribution [1] to the debate on the plausibility of gateway effects as they might apply to e-cigarettes is most welcome. He makes many salient points that comprehensively circumscribe key points of the debate. However, there are several key issues that we believe he has not addressed satisfactorily.

Lack of coherence with population data on teenage smoking?

Etter repeats a frequently made argument that the gateway hypothesis is incompatible with evidence from the USA [2] and UK [3] of declining adolescent smoking. The argument here runs that vaping has been rising while smoking continues to fall, so vaping cannot be causing smoking to any significant degree at the adolescent population level.[4]

This argument relies on an assumption that the population-wide net impact of any putative gateway effect of e-cigarette use would be larger than the combined net impact of all other policies, programs and factors which are responsible for reducing adolescent smoking prevalence (for example, tobacco tax and retail price [5], measures of the denormalisation of smoking[6], exposure of children to adult-targeted quit campaigns[7], retail display bans [8], health warnings [9] and plain packaging[10]). This is a ridiculously high bar that gateway critics demand that anyone suggesting gateway effects must jump over.

It is clearly possible that significant numbers of vaping teenagers who might otherwise not have smoked could take up smoking in an environment where there was a larger preventive effect occurring in response to comprehensive efforts to reduce smoking uptake. The combined impact of such factors in preventing uptake could thereby easily mask considerable smoking uptake that might have not occurred in the absence of e-cigarettes.

With smoking prevalence at record lows in both the US and England, only longitudinal studies with very large numbers of participants would have the statistical power to consider with any confidence the factors responsible for changes in the absolute proportions of adolescents who smoked. Cross-sectional studies could never be definitive.

For this reason, longitudinal prospective cohorts which control for factors known to be associated with smoking uptake are vital to examining potential gateway effects. Nine of these have recently been included in a meta-analysis [11]. Adjusting for demographic, psychosocial, and behavioural risk factors for cigarette smoking, the odds of subsequent cigarette smoking by non-smokers who had any experience of vaping more than tripled among e-cigarette users compared to those with no vaping experience.

Double standards on gateway and “reverse” gateway effects?

E-cigarette enthusiasts often argue that vaping is demonstrably a reverse gateway *out of* smoking for those who quit, while being scathing about suggestions that it could ever be a gateway *into* smoking.

Soundbites like “kids who will try stuff, will try stuff” and “kids who will smoke, will smoke” have been repeatedly held aloft like an omnipotent crucifix before a gateway vampire. These responses are voiced as self-evident truisms, with their circularity being seductive at first blush. However, any cessation researcher offering the equally trite “smokers who will quit, will quit” as a serious contribution to understanding the complexity of transitioning out of smoking, would be rightly pilloried for their primitive understanding of the complex trajectories with multi-factorial elements that can conclude with permanent smoking cessation.

There is a vast literature on the efficacy of smoking cessation interventions (to which Etter, and other prominent critics of e-cigarette gateway claims, have often contributed) where relevant mediating variables (for example: level of addiction, self-efficacy, levels of personal and professional support, planned v unplanned and gradual v rapid quit attempts) are measured, and then adjusted for in estimates of the contribution of the cessation drug or intervention.

Yet Etter argues that a common liability model (“a propensity to use nicotine” in any form) can explain all the main claims of the gateway hypothesis. All we need to say about anyone who smokes regularly is that that they had a propensity to do so. If this hard determinism was all that was needed to be invoked in understanding smoking uptake, how then can we explain the dramatic falls in uptake that have been seen in nations which have robust tobacco control programs? What eroded that “propensity”? Nicotine liability may well be a predisposing factor. But what of the known tractable reinforcing and enabling factors [12] that tobacco control has so successfully identified and addressed over decades?

Implausibility of experimental, occasional vaping transitioning to smoking?

In criticising studies which do not differentiate adolescent occasional, experimental vaping from more regular vaping, Etter argues that it is “hardly plausible that a simple puff or a few puffs on an e-cigarette can cause subsequent regular smoking.” But of course *every* regular smoker started with a “simple puff”, mostly in adolescence. They then typically progressed through experimental, then less-than-daily smoking and finally onto regular, daily smoking. Many young smokers categorised in snapshot, cross-sectional studies as experimental (“a few puffs”) or light, irregular smokers will soon change their status. Just as no young smoker commences their smoking career by smoking a pack on their first day, few if any adolescent vapers commence vaping with heavy, daily use.

Moreover, Etter’s assertion about implausibility ignores an important body of evidence regarding the high susceptibility of children and adolescents to the psychotropic and addictive effects of nicotine. For example, Fidler et al [13] and others [14] have highlighted

that children only require a very minimal exposure to develop an important and identified “sleeper effect”: a vulnerability to smoking after trying just a single cigarette, that can lie dormant for three years, or more:

“From a neurobiological viewpoint, neural reward pathways might be changed as a consequence of a single exposure to nicotine, thus potentially increasing vulnerability to later smoking uptake” [13]

Others have referred to an established body of evidence relating to youth nicotine exposure:

“Importantly, several studies support that a single drug exposure can lead to changes in synaptic strength that are associated with learning and memory. Ultimately, these cellular changes could underlie the long-lasting effects of drugs” [14]

The high susceptibility of children and youth to the “neurobiological insult” of nicotine was recently been highlighted in the US Surgeon General’s report on the potential risks of nicotine and electronic cigarettes to youth [15]. The implications of this material appear to escape Etter’s analysis entirely.

McNeill, who has been persistently critical of gateway effects [4, 16] authored two heavily cited papers which noted that

“The first symptoms of nicotine dependence can appear within days to weeks of the onset of occasional use, often before the onset of daily smoking” [17].

Moreover, in a 30 month follow-up of the same subjects, it was noted that

“Symptoms of tobacco dependence commonly develop rapidly after the onset of intermittent smoking, although individuals differ widely in this regard. ...*There does not appear to be a minimum nicotine dose or duration of use as a prerequisite for symptoms to appear.* The development of a single symptom strongly predicted continued use, supporting the theory that the loss of autonomy over tobacco use begins with the first symptom of dependence” (our emphasis) [18].

The clear contrast between the well-established understanding of cigarette smokers' rapid onset of symptoms of nicotine dependence with efforts to trivialise concerns about initial infrequent use of e-cigarettes is therefore noteworthy.

Schneider and Diehl in their e-cigarettes as “catalysts” model [19], reviewed features of vaping that make it both attractive to adolescents (perceived lower health risks, attractive tastes, lower price, inconspicuous use, higher acceptance among peers and others) and why “increasing familiarity with nicotine could lead to ...potential transition to tobacco smoking”.

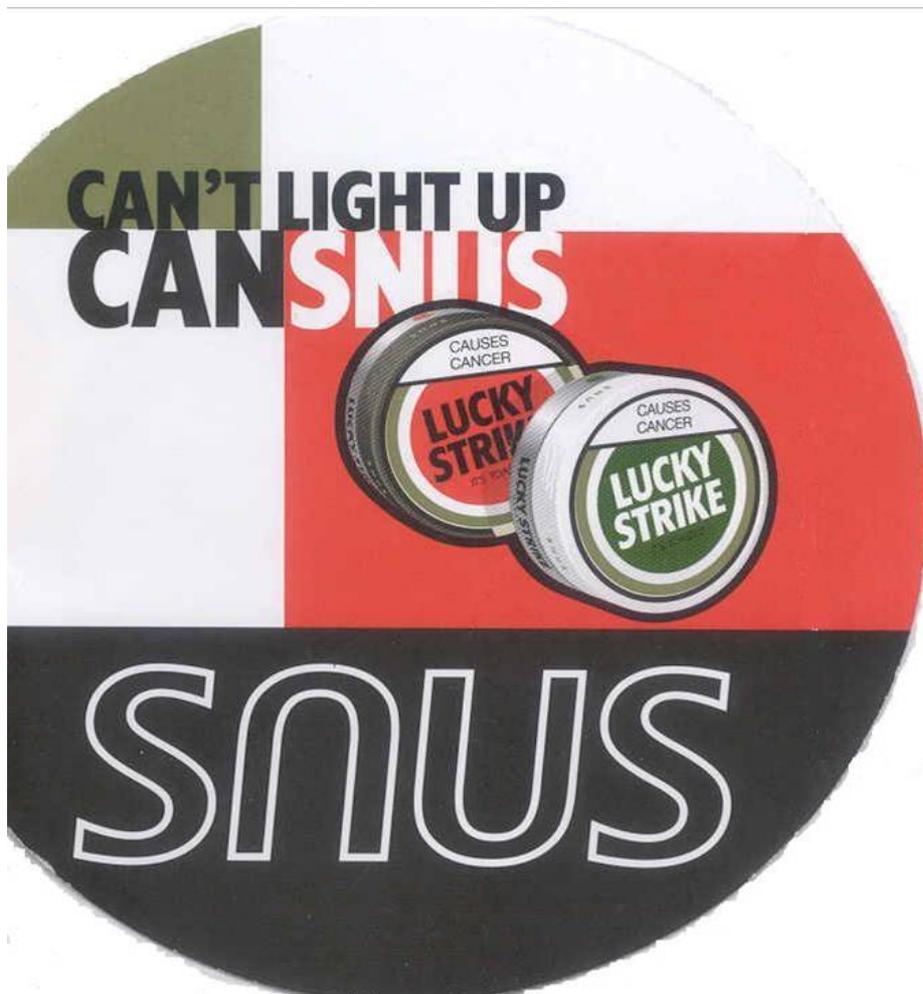
They offer several cogent and highly plausible reasons for such transition that Etter does not consider. These include:

Accessibility: E-cigarettes and cigarettes are often sold alongside one another. Adolescents who might otherwise never visit a tobacco retailer and be exposed to retail promotions, discount offers and curiosity push cues would be thus now exposed.

Experience: As they state “Becoming used to the habitual and ritual procedures of smoking such as poise, handling, smoke breaks and body language” may erode negative feelings about smoking in some adolescents and facilitate experimentation with cigarettes.

Finally, despite high profile claims from transnational tobacco companies that they hope to transition as many smokers as possible from smoking to vaping, an obvious alternative and highly plausible business model for these companies would be to facilitate dual use: smoking *as well as* vaping, rather than smoking *instead of* smoking. With smoking not allowed in many locations where smokers spend many hours every week, it would be very much in the commercial interests of the industry to promote both forms of consumption.

Strategies like retail placement of cigarettes with e-cigarettes, retailer incentive promotions to encourage dual use, cross-branding, and promotional activity in poorly regulated environments (especially the internet) promoting messages like “when you can’t smoke, vape” seem likely to follow similar parallel campaigns used to promote smokeless tobacco (see Figures)



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