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Submission to Senate Select Committee on Tobacco Harm Reduction

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From Ron Borland PhD

Thank you for the opportunity to make a submission to this Inquiry.

I am currently a Professor in the School of Psychological Sciences at the University of Melbourne since July 2019. Prior to that I was the Nigel Gray Distinguished Fellow in Cancer Prevention, at The Cancer Council Victoria. I have been researching issues around tobacco control since 1987. I have published around 450 peer reviewed publications most that relate to aspects of tobacco control. Among other things I conducted the first longitudinal study of the impact of workplace smoking bans on smoking behaviour and the first longitudinal study on the impact of health warnings on smokers. I, along with colleagues, have pioneered the use of strong empirical methods to evaluate the impact of tobacco control policies and programs more generally. In this regard I was senior Editor on the prestigious IARC Handbook which lays out the methods for population-based policy evaluation within tobacco control.

Any serious evaluation of the potential and risks of a harm reduction approach needs to consider all relevant information, not be restricted to evidence that supports one's pre-existing position. I have attempted to do this in what follows. I start by evaluating some of the arguments that are being pursued by proponents of either side that are inconsistent with the totality of relevant evidence.

This submission does not consider harm reduction activities, like smokefree places for which there is near universal public support, or failed harm reduction efforts such as those associated with ill-conceived efforts to restrict tar intakes which failed because the industry subverted the efforts through use of filter venting, but to focus on the potential of lower toxin products as potential replacements for smoking, not simply as short term smoking cessation aids (as is currently the case with Nicotine replacement products).

In this submission I consider three main types of product: low toxin smokeless tobacco such as that used widely in Scandinavia (referred to as Snus, the Swedish word for snuff); nicotine vaping products (NVPs) where a solution containing nicotine is heated to produce an aerosol that is inhaled; and heated tobacco products (HTPs) where specially treated tobacco is heated (not burned) and a resultant aerosol containing nicotine is inhaled.

It is likely that the Committee will be provided with claims along the lines that "we have no real idea of how harmful vaping is likely to be" and some may go further to claim "it might be comparably harmful to smoking." Such claims fail to consider the science. A core function of science is drawing understanding from observations to identify mechanisms that can be applied to predict future effects with increased precision (rarely 100% successful in the biological and behavioral sciences, but increasingly accurate). That is, science helps us predict uncertain

futures with greater accuracy than any other method. As a result, this analysis is grounded in scientific thinking because there cannot be empirical evidence around some possible outcomes for many years. However, in some cases, especially with respect to youth uptake, we now have data that should have alleviated some of the concerns of those opposed to the introduction of alternative products.

1. Relative safety of NVPs

The estimate that "the risk of using NVPs long term is unlikely to be more than 5% of risk of active smoking and may be considerably less" is a sound estimate based on considerable science. [1]

The claim is grounded in good science, which is not the same as direct evidence. Of course there is no direct evidence as to what the effects of vaping will be over a lifetime of use: only the very earliest adopters of this technology could have been vaping for as much as 10 years, or of what benefits shifting to NVPs from smoking may provide at different ages, and how this compares with quitting nicotine altogether at the same age. It will take decades of use to establish actual outcomes. That is why we need science, the evidence grounded elaboration of mechanisms to help us predict likely outcomes.

A version of the 5% estimate of harm (ie, 95% les harmful) first emerged from a Delphi-like exercise using a pool of a dozen experts. [2] The conclusion, as I understand it, was based on toxicological considerations and known patterns of harm from related products. Since then, estimates of harm have been examined by the Royal College of Physicians [1] and Public Health England [3] (largely the same group but using a different process). They endorsed the estimate after re-considering the evidence base and found no evidence to shift from it as they consider it a likely conservative claim based on the evidence that has emerged since it was made.

I find it notable that while a lot has been written about 5% not being evidence based, no-one has come up with a credible alternative estimate; that is, an estimate grounded in a scientific understanding of the determinants of harm. I think this is because it simply can't be done.

Let us consider some relevant evidence. For a start, estimates of total harm-weighted toxin exposure from NVPs is generally accepted to be less than 1% of that from smoking. [4] As some of the data used to estimate toxin deliveries came from early studies of more primitive devices which delivered more extraneous toxins than more modern models, this estimate may have to be refined down (certainly not up). All, or nearly all, of the studies that have found concerning levels of toxins (as distinct from any), have used the vaping devices under unnatural conditions which lead to overheating and generation of high levels of toxins like formaldehyde eg. [5]. However, such levels are aversive to the user. Hence, sustained use, or in any real-life use, under such conditions is highly unlikely. Moreover, most modern devices use thermistors to prevent overheating, further reducing any risk of acutely high intakes of such compounds.

What do we know from patterns of tobacco use and disease around the world? First, most of the harm comes from combustion, either at the time of use (with smoking), or through charring used in preparation of some forms of smokeless tobacco. The epidemiology of smokeless tobacco

indicates that when products are engineered to minimizes toxin levels, as was pioneered in Sweden, there are no clear increased risks of premature mortality in otherwise healthy individuals from extended use. There is evidence that nicotine, as a stimulant, is a risk factor for heart attack in those who have recently had heart disease, but then so is any stimulant or activity that, like exercise that can cause phasic rapid increases in heart rate.

The epidemiology of smoking indicates that in broad terms less than 10 years of regular (essentially daily) smoking is not associated with measurable increases in premature mortality, some studies would extend that out to closer to 20 years. Studies have not been able to find any increased premature mortality in those who quit smoking under 30, and as most who smoke have taken up daily use by 20, thus at least 10 years of use. However, beyond the age of around 30, the risk begins to rise, slowly at first, but more rapidly after around 40, such that the average lifelong smoker dies around 10 years younger than a comparable never smoker. [6] They also suffer at least the same amount of disability reduced years of life within their allotted span. Somewhere between one in two and 2 in 3 long term smokers will die prematurely as a result of their smoking. [7, 8]

While it is true that "every cigarette is doing you damage", the overwhelming majority of the damage is corrected by the body's repair systems. However, over time there is an increased probability that the repair systems will fail (ie, be overwhelmed or wear out), leading to sustained and expanding damage. It only takes a fractional failure in recovery to kick start a disease process like cancer which can rapidly get out of control and end up being fatal. We should not assume that several years of smoking generates no risk, but it is clearly small.

It is important to note that the dose-response effect on toxins with harm is presumed to be monotonic increasing but is not always linear across the range from no-measurable effect to levels associated with serious adverse effects.

The evidence from smoking is that while both play important roles, the dose of toxins per day is less important in estimating risk than the duration of exposure. [9] In both conference presentations and in personal conversations the distinguished British epidemiologist Sir Richard Peto has indicated that duration (years of use) is around 3 times more important than amount per day in predicting adverse effects from cigarette smoking, but such estimates are naturally imprecise and may not generalise to levels of exposure much different to the patterns typically found in the studies of the health effects of smoking on which these estimates are based (average daily consumption of 10-20 cigarettes per day among daily smokers).

On a purely dose related basis, we might expect vaping to carry less than 1% of the harm. If vapers continue to vape for as long as smokers smoke, then length of exposure would be an issue, which should raise the likely risk profile up, but by how much we do not know for sure, but 3 times would raise the upper limit to around 3% of the harm. However, the risk profile is not likely to be linear. For smoking the risk of passive smoking causing heart disease clearly indicates a lower threshold of harm and recent work indicates that smoking at very low daily levels only approximately halves the risk of heart disease. [10] Daily vaping is more like having one puff per day, spread over several hours, and what the impact here will be is also uncertain,

but it is likely considerably less than for the levels of passive smoking that have been demonstrated to increase disease risk. Other major causes of mortality have a more linear relationship with disease risk, so the reduction of risk with lower consumption is far greater than for heart disease.

Finally, the epidemiology of use of the cleanest forms of smokeless tobacco in Sweden where there is long-term use finds no clear evidence of raised mortality among otherwise healthy people, but a risk of continued use for death among those with heart disease, something expected for a stimulant. [11] This data suggests any additional effect on length of use is likely small. This understanding of dose related effects is important for public policy in considering the relative risks of use of HTPs as against NVPs. Taking Stephens estimate as a guide, smoking a HTP at an equivalent rate to smoking (ie smoking15-20 cigarettes per day), would result in exposures equivalent to around 1 cigarette per day, a level which the research (including research on passive smoking) suggests is associated with considerable harm, although much less than smoking (especially for heart disease). [4] However, equivalent levels of vaping would represent no more than 1 puff of a cigarette per day, levels where risks must be considerably less, and such exposures have not been linked clearly to disease to date (due to difficulties of measurement and low prevalence of such levels of active smoking among other things). This means from a public health point of view, far greater caution is required around HTPs than NVPs.

The Committee is likely to be told that there is more and more evidence about the harms of vaping. It is true, there are more studies, but do they change the conclusions? The studies that claim to find harms from vaping are mainly of two broad types: those reporting acute effects, some of which can hardly be considered harms. These include short term rises in heart rate, a phenomenon that occurs at similar levels when using other stimulants like caffeine (not to mention engaging in physical activity). In my doctoral work in the 1980s, I demonstrated that having someone smile at you caused a short-term increase in heart rate. Similarly, short term stiffening of blood vessels is to accommodate the increased blood flow needed to support increased activity. Some is also short-term damage that is routinely repaired, analogous to the damage to muscle tissue caused by any episode of vigorous exercise. The point being these are only problems if they convert into chronic changes from normal. That is, they stress the body beyond the capacity of normal repair mechanisms to adapt.

Some of the second set of studies claim longer term effects, but some of these use retrospective data and claim cases attributable to vaping that have occurred before vaping was prevalent. At least one such paper has been retracted, [12] and several others are being questioned. The other problem is that virtually all these studied participants are fairly recent ex-smokers and so far it has been impossible to control for effects of past smoking. In time, it would be surprising if better controlled studies did not find small effects, especially when comparing continuing vapers with those quitting all nicotine, but nothing the authors point to meets such a standard at present.

There is limited evidence on the other side of the ledger for benefits. The longitudinal study comparing a sample of vapers to continuing smokers of Polosa and colleagues focusing on symptoms of COPD shows clear benefits of shifting to vaping, but this study does not have a sample of those quitting all tobacco to compare with, and the sample size is small, so it cannot be

thought of as definitive. [13] Further in work we are about to submit for publication, we find that reported ability to cope with stress and reported day to day health are reported as better by ex-smokers who vape compared with ex-smokers who quit all nicotine, although his seems to be restricted to the first year or so post quitting, suggesting it may only ease the readjustment needed in moving from a daily smoking. It is hard to imagine getting results like this if actual health and adjustment was worse among vapers.

These are collectively a compelling set of facts which with science-grounded reasoning leads to the conclusion that it is very likely that the level of harms will be very much lower for vaping than for cigarettes because the exposure is much lower. In short, the estimate that NVPs are 95% less harmful is a reasonable estimate of the differential risk, but one that needs to be kept under constant review if new evidence emerges that suggests some of the assumptions are flawed.

The real question about vaping is whether the level of harm that is likely is sufficient to justify constraints on their availability and to what degree.

2. NVPs help people quit smoking

There is evidence that NVPs do assist people to quit smoking. The latest meta-analysis, from the Cochrane collaboration drew the conclusion that there is moderate evidence that NVP use is superior to state of the art recommendations on NRT use for smoking cessation. [14] The qualification of the conclusion (ie moderate rather than strong) was based on the number of trials, and the limited number of vaping devices tested. In this context it is important to note that the levels of evidence used in this case is far more stringent than that applied to domains where randomized trials are not possible; ie, the evidence although not rated as strong is far stronger than for any effects of vaping on uptake of smoking or of effects of promotion on use.

The large trial of Hajek et al, found a vaping intervention to be almost twice as effective as what was close to current state of the art use of pharmacotherapy; ie not just showing an effect, but that vaping is superior to the existing recommended products. [15] Another trial by Walker et al also found a significant improvement of adding vaping to a group only using nicotine replacement therapy. [16] These two recent trials used more modern vaping devices than those used in the earlier trials where the effects were more modest. Admittedly, much of the benefit of vaping appears to be due to higher levels of persistent use, which may be important for relapse prevention. It is also important to consider in the context of the actual role vaping plays. It has some of its effect, perhaps most, by acting as a replacement for smoking, rather than as a short-term cessation aid.

In addition, in countries where vaping is most prevalent, rates of smoking appear to be coming down, which is consistent with population level effects. I accept there are studies of community samples comparing those who vape to quit and those who use nothing which do not show positive effects (some do), but there is evidence that those who choose vaping to quit, as for other forms of help, tend to be more dependent, so the use of the nicotine, may be only levelling the playing field, not providing a fundamental advantage to those who use it. NB. Differences in who use interventions is a key reason we need randomized trials where they are possible.

3. Vaping is likely to be less addictive than smoking

It seems to be assumed by those who oppose harm reduced products that the products will prove as dependence forming (ie addictive) as smoking cigarettes. This is not altogether consistent with the weight of limited evidence suggesting vaping is likely less addictive, although it remains plausible. [17, 18] Our understanding of the determinants of dependence are limited but there are good reasons to hypothesize that levels of dependence might be less. Regardless of dependence, if use of low-toxin nicotine is much less harmful, the public health consequences of long-term use are still going to be much les than for smoking.

4. Vaping is not a gateway to smoking, despite considerable evidence consistent with this hypothesis.

First, I acknowledge that, there is overwhelming evidence that young people who have never smoked but have tried vaping are more likely to try smoking in the next year than those who have used neither. [19] This is the core of the argument in favour of a gateway effect; ie that vaping increases the likelihood of subsequent smoking (eg, by creating an appetite for nicotine and/or because of the behavioural similarity of vaping to smoking). However, these studies are also completely consistent with other possible relationships, most notably that the kinds of adolescents who try vaping would, in the absence of vaping, have tried smoking anyway and perhaps even sooner. That is, initial vaping may be no more than a marker for future risky behaviour, not a cause, and it could even have a protective effect (ie those who try vaping being less likely to start smoking as a result, as long as they are still more likely than those who have never tried any such product). [20]

To make a causal inference much more is needed than a longitudinal association. If there is a systematic causal relationship of vaping leading to smoking, then a rise in vaping levels should be associated with a rise in smoking levels at population level within the comparable time period. As the studies typically have one year follow-up, this would mean year to year increases in smoking as vaping increased. This has not been found in the countries where uptake of smoking is assessed regularly (largely the same countries, eg USA from which the observational studies come). Indeed, there is evidence that the rate of decline in smoking actually increased over the period when vaping first become popular in the USA. [21]

It is also worth noting that none of the studies had enough cases of daily smoking to see if the associations found for any use and monthly use extend to regular use. In this regard, there is clear evidence from Sweden that uptake of daily snus use is protective against future daily smoking and also is associated with increased cessation among those who smoke (either following initial snus use or who took up snus after becoming daily smokers). [22]

Smoking rates are going down for every country where vaping has increased where I have seen data. This is also true in Australia, even though rates of vaping are lower as a result of nicotine vaping products being effectively illegal. If vaping is leading to more smoking, as a gateway predicts, how can we explain the two behaviours moving in opposite directions? The data is more consistent with a substitution effect. A substitution model would generally accept the likelihood of some degree of use of both products, but with a net tendency to choose the

substitute (ie vaping), although this could occur without ever trying the alternative, something more likely where the existing product is perceived as undesirable in some respects (ie harmful) as in this case. The substitution model is also grounded in there being differences in people's attraction to the use of nicotine. For example, other mild stimulants like caffeine, while some people find use highly rewarding, for others it does nothing much and yet others find the stimulatory effect mildly unpleasant, so don't use. There is a lot of evidence that adolescents attracted ot smoking have different characteristics than those who are not.

It is instructive to consider evidence on effects of other nicotine products on smoking rates. The best long-term data comes from Scandinavia where In Sweden most men use smokeless tobacco (Snus, Swedish for snuff) and smoking rates are the lowest in Europe. Recently snusing (use of snus) has become popular as an alternative to smoking among girls in neighboring Norway and the latest data indicates only 1% of Norwegian adolescent girls smoke regularly [23] an extremely large decline from levels of a few years ago and unprecedented anywhere. In Sweden, there is strong evidence that not only does daily Snus use protect against ever becoming a daily smoker, but ever daily snus use is strongly predictive of quitting smoking (ie being an ex-smoker if ever smoked). [22]

The findings do not mean that under no conditions can vaping encourage smoking, but that it is not happening in the places where vaping is increasing. The myth of a gateway continues to be propagated despite the evidence of youth smoking rates declining. It shows a misunderstanding of basic scientific inference.

5. The tobacco industry

The tobacco industry means different things to different people. For me it is primarily the large marketers of cigarettes, a market dominated by a small number of extremely large companies. These companies have a minority share of the vaping market but completely dominate tobacco markets for cigarettes and HTPs. They are most threatened by open systems vaping as producing pharmaceutical grade nicotine is relatively cheap and does not require massive infrastructure, unlike most tobacco products, where it is easier to control a market. The uptake of HTPs in some countries is simply amazing, and there is a real risk the world will end up being dominated by these lower, but likely substantial, risk products, rather than the almost certainly far less harmful vaping products.

Emerging reality; Australia is increasingly out of step with comparable other countries and they are doing better than us at reducing smoking. The countries we tend to compare ourselves with most: New Zealand, Canada, the UK, US, and Europe all have much more liberal policies around vaping, indeed in the case of NZ and Canada, both have moved in the last few years from the same historical position Australia began with to explicitly legalize vaping in various forms and have put in place (or are putting in place) regulatory frameworks to minimize the risk. As far as we can tell, from those countries with enough data, smoking rates are declining at least as fast, probably faster in those other countries than in Australia.

Yes, Australia has always been a leader, but the evidence is accumulating that we are being passed by some countries, and others appear to be catching up. We cannot be certain the vaping

policies here are the reason, but it is hard to think of an alternative reason. We have been increasing the price of cigarettes more than most comparison countries and have much stronger policies of restricting promotion than some others (especially the USA). Even countries that have traditionally been very weak on tobacco control, like Japan are seeing historically unprecedented declines in cigarettes consumption, seemingly replaced (mainly) in this case by HTPs rather than NVPs (which are effectively illegal like in Australia). [24]

There is no doubt Australia has been a leader in tobacco control and I have spent most of my career contributing to documenting the impacts of policies on tobacco use, many of which were pioneered in Australia. Unfortunately, I fear that Australia's supremacy has gone. We still lead on price and led on standardized packaging and in all respects, apart from harm reduction, can be thought of as leading, or in the leading bunch, so why are our smoking rates declining somewhat slower than in other countries that in some cases (eg, US) are way behind us on conventional policies but have access to harm reduced alternatives?

COI: I have a long-term philosophical commitment to harm reduction approaches to drug use and other ways people should be encouraged to act to minimize risks from potentially harmful activities they may choose to indulge. In this regard, I favour nudges as ways to make desirable options easier and undesirable ones a bit harder as an approach rather than prohibition. I think the evidence is clear, attempts at prohibition for activities that people want to engage in is a flawed strategy, it can lower levels of the behaviour (but not always) but not eliminate them, and it is with large social costs.

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