Filter ventilation and the risk associated with cigarette smoking

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It has long been clear that cigarette smoking is causally linked to many diseases, particularly lung cancer, chronic obstructive pulmonary disease and various forms of cardiovascular disease. For lung cancer, for example, a meta-analysis my colleagues and I conducted, based on epidemiological evidence published in the 1900s [1], estimated that current smoker/never smoker relative risks, were around 10 in North America and Europe, though lower (around three) in Asia. They were higher for squamous cell carcinoma than for adenocarcinoma, were very strongly related to amount smoked, and declined with increasing years of cessation of smoking.

Around the middle of the last century, nearly all cigarettes smoked had no filters [2]. However, after epidemiological studies from the 1950s onwards associated cigarette smoking and lung cancer [3,4] various research programs were initiated to better understand the toxicity of mainstream cigarette smoke condensate. Based on animal studies of the tumorigenic activity of cigarette smoke [5] the lowering of cigarette smoke yields was then viewed, both by public health scientists and governmental authorities, as a means of reducing the adverse health effects associated with smoking [6-12]. Beginning in the 1950s and 1960s, US and European government-initiated programs were implemented to reduce the total aerosol residue (tar) yields of cigarettes [13], with various measures considered to do this, including the use of filter tips, porous cigarette paper, expanded and reconstituted tobacco and filter ventilation.

The effect the introduction of filters and tar reduction has had on the risks associated with cigarette smoking has been much discussed. In 2001 the National Cancer Institute published their Monograph 13 [14], which concluded that filter cigarettes, or cigarettes with lowered machine-measured tar yields, had not been demonstrated to provide any reduction at all in the risk of diseases related to smoking. One argument the Monograph made is that smokers compensate when smoking lower delivery products so as to maintain a constant nicotine yield. Such compensation might occur by increased inhalation of the smoke or by increasing the number of cigarettes smoked per day.

This argument seems to be rather weak based on a detailed look at the evidence. Thus, a meta-analysis of relevant epidemiological studies we published in 2004 [15] reported filter/plain relative risks for lung cancer of 0.61 (95% confidence interval 0.54-0.70) for data unadjusted for amount smoked and of 0.66 (0.38-0.76) for adjusted data. Similarly, relative risks for lung cancer were significantly lowered for the lower tar/higher tar comparison, with unadjusted estimates of 0.60 (0.45-0.81) and adjusted estimates of 0.73 (0.64-0.83). The reductions were evident in studies of different sexes, locations, periods or designs. Consistent with this are results from cross-sectional and brand switching studies summarized in a publication [16] which combined study-specific estimates of a “compensation index”, with values of 1 indicating complete compensation and of 0 indicating no compensation. Here estimates of the index were 0.78 (0.72-0.84) from the cross-sectional studies and 0.74 (0.68-0.81) from the brand switching studies, indicating that compensation is incomplete. This review concluded that compensation was predominantly by altering puff volume, with little change in cigarette consumption. An experimental study [17] which found no evidence of deeper inhalation when smoking low- versus high-yield cigarettes was considered by the authors to be consistent with “published literature indicating that smoking low-yield cigarettes does not increase the depth of inhalation”.

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It has also been claimed that filter ventilation has contributed to the increase in lung adenocarcinomas relative to other study types [18], a claim some felt sufficient to support a ban on filter ventilation [19]. However, this publication did not consider evidence from a review [20] showing that changes in diagnostic criteria over time were at least partly responsible for the shift from squamous cell carcinoma to adenocarcinoma. That review also pointed out clear evidence that the shift occurred in those who have never smoked, as well as in smokers, and that the shift began before the rise in use of low tar filtered cigarettes. As noted by Travis et al. 2013 [21] the shift may have occurred because in the past non-small-cell carcinomas were lumped together without separation into squamous cell carcinoma and adenocarcinoma as there was then no therapeutic reason to separate the types, a situation which changed in more recent years.

There appears to be no good epidemiological evidence relating risk of smoking-related diseases in smokers of filtered cigarettes specifically to the degree of ventilation of the filters used. Obtaining valid results would in any case be difficult, as this information is not provided on the pack. It is also possible that some smokers may partially block the ventilation holes [22,23], although researchers [14,24] have concluded that vent blocking only occurs in a minority of smokers some of the time, with little effect on smoke yields.

However, some publications have related biomarkers of exposure (BOEs) and biomarkers of potential harm (BOPHs) to the degree of ventilation in the cigarettes tested.

One publication [25] reviewed data from 11 studies in nine countries and concluded that the data suggested that smokers of highly ventilated filter cigarettes were on average exposed to lower amounts of tar and nicotine per cigarette than were smokers of less ventilated filter cigarettes.

Another publication [26] related the contributions of tobacco nicotine content and filter ventilation to machine yields of tar, nicotine and carbon monoxide, based on data from 92 UK, US and Canadian cigarette brands. They concluded that filter ventilation appeared to be the main method for reducing yields, but also noted that some brands contain about twice as much nicotine as do other brands.

Very recently, Carroll et al. published a paper in Cancer, Epidemiology, Biomarkers and Prevention [27] based on data from filter ventilation levels in cigarette brands together with data from Wave 1 of the US Population Assessment of Tobacco Use and Health (PATH) study conducted in 2013 and 2014. Restricting attention to smokers who reported a usual brand and did not regularly use other tobacco products, they related the degree of ventilation (ranging from 0.2% to 61.1%) to nine BOEs and five BOPHs. The BOEs included nicotine, tobacco-specific nitrosamines, volatile organic compounds and polycyclic aromatic hydrocarbons, while the BOPHs measured inflammation and oxidative stress. In the abstract, the authors noted that “Filter ventilation was not associated with BOE or BOPH” though they earlier stated that significant positive relationships were seen after adjustment for one BOE (the volatile organic compound N-acetyl-S-(phenyl)-L-cysteine) and for one BOPH (high-sensitivity-C-reactive protein). They also noted that smokers of more highly ventilated filter cigarettes, compared to smokers of less highly ventilated filter cigarettes, perceived their brand to be less harmful.

My invitation to prepare this article was based on a letter my colleague John Fry and I wrote to the same journal [28] commenting on the paper by Carroll et al. 2021 [27], a letter to which the original authors replied [29]. Our letter made two main points. The first was that the analyses that Carroll et al. had conducted did not, as is usual, provide information on a per cigarette basis, and that, if one did so, clear trends of decreasing levels with increasing ventilation, often highly statistically significant, were found with four of the five BOPH, and with some of the BOE, notably total nicotine equivalents. We also noted that our analyses were only approximate as we did not have the individual person biomarker data available, and suggested that Carroll et al. might report such analyses, a suggestion that was not taken up.

The other main point was that we had discovered, when analyzing data from the PATH study for another publication [30], that there are erroneous data on amount smoked for some individuals, due to confusion in data entry between cigarettes and packs smoked per day. We gave the example of one individual who reported smoking 300 cigarettes per day at Wave 1, 10 at Wave 2 and 15 at Wave 3, when the Wave 1 data should presumably have been 15 cigarettes per day. Carroll, et al. did not comment at all on this, and therefore one does not know whether they accounted for these erroneous data.

Another point we could have made, but did not, due to limitations on the length of letters to the journal, was that the adjusted data presented by Carroll et al. not only adjusted for age, sex, race, education, smoking duration, menthol status and quit effort, but also accounted for multiple comparisons. Adjustment for multiple comparisons is a somewhat dubious procedure, as it makes inferences for one biomarker depend on how many other biomarkers were considered, which seems inappropriate to this author.

Ultimately, while it is possible that changes in the type of cigarette smoked over time have reduced the risk of smoking-related diseases, it is clear that any such reductions, if true and verifiable, are not large, and far less, proportionately, than the substantial reduction in tar level that has occurred since the days when this exceeded 30 mg per cigarette. Quitting smoking clearly causes a much greater reduction, where we estimate that the excess risk associated with cigarette smoking is approximately halved every 10 years after quitting for lung cancer [31] and for chronic obstructive pulmonary disease (COPD) [32], with the reduction in excess risk declining about twice as rapidly as this for ischemic heart disease (IHD) [33] and for stroke [34].

From the point of view of risk reduction, smokers clearly do best to quit, as there are no safe cigarettes. Yet, a significant proportion of smokers do not want to do so, preferring to retain their dose of nicotine, coupled with their user behaviour and taste-related pleasure. For such smokers, using, or switching to, any type of combustible cigarette including lower tar ventilated filter cigarettes would hardly be a much safer option. They do, however, have access in certain countries to alternative products which involve no exposure to smoke and deliver similar nicotine doses with the potential to reduce their risk of harm. Swedish moist snuff (“snus”) is one possibility, where quite extensive epidemiological evidence shows little or no increased risk at all of smoking-related disease [35,36]. Other alternative available products, while not harmless, that have the potential to reduce the risks associated with smoking, are electronic cigarettes.
(e-cigarettes), heated tobacco products and nicotine pouches, where chemical and biomarker data indicate a much-reduced exposure to biomarkers of relevant toxicants [37-42]. While clear demonstration of harm reduction requires robust epidemiological data, various studies confirm the reduced emission of harmful compounds from heated tobacco and e-cigarettes compared with combustible cigarettes [43-46]. Risk assessment studies indicate that products with reduced exposure of harmful compounds have the potential to reduce the risks associated with smoking [47-49]. Separately, clinical research with adult smokers using electronic cigarettes or heated tobacco products instead of combustible cigarettes have shown measurable changes in biomarkers of exposure and biomarkers of biological effect [39,50,51], and a less detrimental impact or even improvement in pulmonary and vascular health [52-55].

To get a good picture of the health effects of e-cigarettes, heat-not-burn cigarettes, or nicotine pouches one really needs results from large cohort studies comparing risk of disease onset in those who at baseline were continuing cigarette smokers with those who had switched to the new product (and perhaps also in those who used the new product but never cigarettes), with adjustment for relevant factors. While it may be too early to get relevant results for lung cancer and COPD, where it takes a long time to see clear effects of quitting, this is not so for IHD and stroke.

While results from a large, but quite short-term follow-up cohort study in South Korea [52] do suggest that switching from combustible cigarettes (CC) to heat-not-burn cigarettes is associated with a lower risk of cardiovascular disease than continued CC smoking, no corresponding results are available for e-cigarettes. Unfounded claims that e-cigarette use is associated with a lower risk of cardiovascular disease than continued CC smoking, no corresponding results are available for e-cigarettes.

Conflict of Interest

The author is a long-term consultant to various tobacco companies and organizations, including the funders of this commentary.

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