



PASSIVE SMOKING: AN INSTITUTIONAL PROBLEM

Fabricated risks attributed to passive smoking

Smoking prohibition and its inherent economic and social costs are based on a false representation of evidence, an evidence that does not demonstrate what smoking ban supporters claim it does. This short document, written for laymen, distills a fact-finding process that took several years. It respects scientific rigour and seeks to explain the fine mechanisms of what may amount to the largest epidemiological fraud ever perpetrated. The fact that this fraud has been adopted by health authorities, governments, and high-ranking figures of medicine should raise serious concerns about the condition of the integrity of our institutions, quite independently of the smoking issue.

The case against passive smoking, also known as environmental tobacco smoke or ETS, is mainly based on the statement that it causes lung cancer or cardiovascular disease in non-smokers. The short analysis that follows examines what is considered the strongest evidence, that on lung cancer. Of course, what follows applies equally to the risk for cardiovascular disease and for any other disease attributed to ETS, as the methodology used in these studies is essentially the same.

The US Environmental Protection Agency Report of 1992, considered a milestone for the elimination of smoking from public places, stated that the risk elevation for lung cancer was 19 per cent. However, in 1998 a federal court established that the EPA report was fraudulent as it was based on flawed science and methods. The scientific objections on which this court decision was based have never been challenged. The EPA appealed on the technical point that, since the agency does not regulate tobacco, its report was informational and therefore not subject to the court's jurisdiction. A 2002 ruling in EPA's favour on this technicality, called by critics a "licence to lie," did not address the fraudulence of the scientific core of the report that was established by the federal court, and thus has no bearing on the original finding that EPA had "cherry-picked" its data to arrive at a preconceived conclusion.

The risk elevation calculated by EPA, based on glaringly contradictory evidence, was and is invalid.

It is exceedingly clear that the judgement of the appeal court was political in nature, made to invalidate a science-based legal precedent that would have stood in the way of the smoking prohibition social programme. It is also equally clear that the tobacco industry, hounded and vilified for years, did not act on the 2002 decision because in the meantime it chose appeasement, by signing the Master Settlement Agreement with the American states. That agreement proscribes the industry from countering any claim of prohibitionist agencies, thus preventing any effective opposition to them. The EPA report is still cited by prohibitionists even as its flawed science and methods are still practised by researchers and government agencies today. Therefore the scientific and logical analysis that follows is still fully applicable.

EPA deduced the risk from 11 studies, of which eight reported a risk elevation and three a risk reduction, that is, protection from lung cancer. These contradictions alone would be sufficient to negate the probability of risk. But even assuming that the 19 per cent average is a valid number, an analysis of how and from which data this number has been calculated leads to extraordinary revelations. It is to be noted that these revelations are applicable to all the studies on ETS that preceded and followed the 1992 EPA report, up to the present time.

1 - **Nonexistent Measurements.** The studies claim that lung cancer risk to non-smokers increases as the exposure to ETS increases, which means that the ETS exposure must be accurately measured in order to quantify risks numerically with the two-decimal precision that the studies show. Because lung cancer is a disease that develops slowly and manifests itself for the most part at an advanced age, this also raises the need to measure exposure to ETS over the non-smoker's lifetime. This is what the studies claim to have done, even though it was not possible to measure exposure starting from any person's birth through the years needed for lung cancer to develop. Rather, attempts were made at a backwards reconstruction of a person's exposure from old age to birth.

Obviously, both measurements are impossible. So impossible, in fact, that ETS exposure has not been measured at all. Instead of an independently objective measure, 60-to-70-year-old non-smokers have been asked to recall what their personal exposure to ETS was during their lives. Typically, such people were asked to recall how many cigarettes, cigars or pipes had been smoked in their presence since early childhood. Their reveries – elicited in a few minutes usually over the phone, or even substituted for by comments provided by the relatives of deceased persons -- were recorded by the studies as precise numbers devoid of error and uncertainty. We all know how difficult it is to remember what we ate one week ago, never mind 20 years ago or during childhood! How is it possible to remember, with an absurd expectation of precision, the exposure to smoke of 30 or 50 years ago? Unless one actually wants to be fooled, the only compelling conclusion is that without dependable measures of exposures, the statistical elaboration of the studies is illusory and so are the claimed risks. These arguments alone are sufficient to discard the studies and their conclusions on risk.

2 - **Fatal Flaws.** Although the measures of exposure are not valid, let us assume now for the sake of argument that they are. Summaries, conducted by groups that have an interest in finding risks on ETS, report that the average of all the 75 primary studies on lung cancer and passive smoking published up to May 2006 indicate a risk elevation of 20 per cent, basically the same imagined by EPA in 1992. Such a relatively low elevation is not credible because the studies have not accounted for a whole series of prejudices, biases and confounders that are inevitably present and that are in themselves more than sufficient to cancel the claimed risk elevation. For example, it is known that people with lung cancer are more prone to amplify their recalls of exposure - recall bias - than those who are not so affected – and this is for obvious emotional reasons. Another example is that some state that they are non-smokers but do not say that they have been smokers, so they end up in the wrong category – (misclassification bias). And yet another one: there are over 30 risk factors for lung cancer reported in the professional literature, about 300 of them for cardiovascular diseases, and their very likely interference in ETS studies has never been credibly measured and corrected for. It is therefore exceedingly probable that the small risk elevation of 20 per cent is fictitious because of interference that is not and cannot be calculated.

Singly or combined, these considerations are sufficient to explain the glaring inconsistencies of different studies, where some claim an increased risk, others protective effects, or, in several studies, neither one nor the other, and erase the credibility of the claimed risk of ETS.

3 - The Absurd Methodology. A third category of irrecoverable flaws invalidates the studies on passive smoking independently from the first two, and therefore invalidates the statements of causal link. The overwhelming majority of these studies do not define risk on the basis of higher or lower frequency of cancer in function of higher or lower exposures to ETS. Rather, as we have seen, risk is defined on the basis of different memories between the groups that are compared. One set of groups consists of self-declared non-smokers all with lung cancer and exposed to ETS; the other set of groups consists of self-declared non-smokers without lung cancer and also exposed to ETS, and this is because people who have never been exposed to passive smoking are impossible to find. To illustrate, studies may have reported that people without lung cancer recalled ETS exposure at, say, a 100 rate, and people with lung cancer recalled exposure at a 120 rate. Strangely, the studies presume that having remembered 20 per cent more represents 20 per cent more risk!

Such an incredible presumption also implies the absurd reasoning that having remembered 20 per cent excess exposure, which is impossible to verify or measure in the first place, has been responsible for all the lung cancer of the group with the disease, while those who remember a little less remain totally immune from cancer. Furthermore, the majority of studies did not find differences of exposure, while others found that some groups already affected by cancer recalled a lower exposure, thus paradoxically indicating that passive smoking would have protected them from the cancer they already had.

Conclusions

- No study of ETS and lung cancer has provided a credible and accurate measurement of ETS exposure.
- The overwhelming majority of the studies has not measured different frequencies of lung cancer in different groups.
- Lacking reliable measurements, the statistical analyses of the studies are illusory.
- No study can guarantee that some of the non-smokers studied were in fact smokers, or had been smokers.
- No study could exclude that the lung cancers observed might have been caused by other risks and not by ETS.
- The overwhelming majority of studies adopted improper and absurd methods of risk calculations.
- The majority of studies did not report differences of risk, and many claimed a reduction of risk.
- Independently or combined, these considerations negate the credibility of claimed ETS risk for lung cancer, and are equally applicable to ETS studies of cardiovascular and other diseases.

The statements of the US Surgeon General, as well as those of other health authorities around the world, are based solely on the studies discussed above.

It follows that all smoking bans in the world are also based solely on this body of evidence.

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