Commission of the European Communities
Proposal for a Council recommendation on smoke-free environments
Accompanying document – impact assessment

Some comments on the estimates of EU-wide mortality due to ETS exposure

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Date : 28th July 2009

1. Sources and main claims

The impact assessment[1] is concerned with a number of issues, including estimation of EU-wide mortality due to ETS exposure, estimation of the economic burden of ETS exposure and assessment of the impact of various policy changes. My comments, in this document, are restricted to the estimation of mortality.

The sections of the impact assessment that are relevant to my comments are as follows:

pp 2-3	Summary of the impact assessment	
pp 10-12	Section 3.3.1.	Health burden of ETS exposure
pp 148-158	Annex V	Health effects of ETS exposure
pp 202-241	Annex VII	Quantitative analysis

There is also an associated analysis by Scoggins *et al*[2] from the RAND corporation, which apparently formed the basis of the analysis for the EU Council figures related to the health effects of ETS.

Relevant sections of the RAND report are as follows:

pp 15-18	Chapter 4	
pp 73-114	Chapter 13	Qualitative analysis
pp 173-181	Appendix C	Relative risks

For convenience, I repeat below certain tables appearing in these reports.

Table 1 Relative risk estimates associated with ETS and specific diseases

		Relative risk		
Disease	**ICD-10 Classification**	**Private home**	**Average workplace**	**Pub/bar/nightclub**
Lung cancer	C33-C34	1.24	1.24	1.73
Stroke	I60-I69	1.45	1.45	2.52
Ischaemic heart disease	I20-I25	1.3	1.2	1.61
Chronic lower respiratory disease	J40-J47	1.25	1.25	1.76

This appears in p 10 of the impact assessment[1], p 205 of its Annex VII (without the column for private home), and p 77 of the Rand report[2].

Table 2 Estimated EU-wide mortality due to ETS exposure among smoking and non-smoking staff in 2008

	Non-smokers			**Smokers**			**Smokers and Non-Smokers**
	Offices	**Bars and restaurants**	**Total**	**Offices**	**Bars and restaurants**	**Total**	**Total**
Lung cancer	387	156	542	600	161	761	**1303**
Stroke	378	160	538	601	197	798	**1336**
Heart disease	384	138	522	612	159	771	**1293**
Chronic lower respiratory disease	565	332	897	881	296	1,176	**2073**
Total	**1,714**	**786**	**2,500**	**2,694**	**813**	**3,507**	**6,007**

This appears on p 3 of the summary of the impact assessment[1] on p 11 of the impact assessment itself, on p 239 of its Annex VII (non smoker data only), and on pp 91 and 93 of the Rand report[2] (with data first for non smokers then for smokers).

Table 3 Estimated number of deaths attributable to passive smoking in the 25 countries of the EU in 2002

Condition	Exposure at home			Exposure at work		Total all home plus all workplaces
	Adults <65 years	Adults 65+ years	All home	All workplaces	Hospitality industry	
Lung cancer	6,498	4,443	10,941	2,300	104	13,241
Ischaemic heart disease	10,025	19,873	29,898	2,444	119	32,342
Stroke	5,973	20,557	26,530	2,060	82	28,591
Chronic non-neoplastic respiratory disease	1,269	3,531	4,800	475	21	5,275
Total*	23,765	48,404	72,170	7,280	325	79,449

SOURCE: Smokefree Partnership (2006).

NOTE: *May be affected by rounding in component estimates.

This appears (as Table 4.1) on p 16 of Chapter 4 of the Rand report[2].

Table 4 Estimated number of deaths attributable to passive smoking among Non-smokers in the 25 countries of the EU in 2002

Condition	Exposure at home			Exposure at work		Total all home plus all workplaces
	Adults <65 years	Adults 65+ years	All home	All workplaces	Hospitality industry	
Lung cancer	403	629	1,032	521	16	1,553
Ischaemic heart disease	1,781	6,977	8,758	1,481	48	10,239
Stroke	729	4,954	5,683	596	19	6,279
Chronic non-neoplasm respiratory disease	155	815	970	201	6	1,171
Total*	3,068	13,375	16,443	2,799	89	19,242

SOURCE: Smokefree Partnership (2006).

NOTE: *May be affected by rounding in component estimates.

This appears (as Table 4.2) on p 17 of Chapter 4 of the Rand report[2].

Tables 2-4 support a statement in the explanatory memorandum to the proposal that:

"According to conservative estimates, 7 300 adults [the 7,280 in Table 3] including 2 800 [the 2,799 in Table 4] non-smokers died as a result of ETS exposure at their workplace in the EU-25 in 2002. The deaths of a further

72 000 people [the 72,170 in Table 3], including 16 400 non-smokers [the 16,443 in Table 4], were caused by ETS exposure at home[3]. The Impact Assessment accompanying this proposal estimates that workplace exposure to ETS accounted for 6 000 deaths [the 6,007 in Table 2], including 2 500 non-smokers [again in Table 2], in the EU in 2008."

2. General approach

For a given country, cause of death and exposure (e.g. exposure in offices) suppose we have estimates of:

N total number of deaths from the cause in the year of interest
p proportion of the population exposed
R relative risk associated with exposure

Suppose that B is the background rate of death in the unexposed group. In the actual population, the rate will be increased to $B((1-p)+pR) = B(1+p(R-1))$, so the attributable fraction A (the excess as a proportion of the total) will be estimated by:

$$A = p(R-1)/(1+p(R-1))$$

NA will then be the number of deaths associated with the exposure.

These numbers are then added over country, cause of death and type of exposure to generate the results. This is the basic methodology used in the impact assessment[1] and in the Rand report[2], though for the workplace calculations the deaths were restricted to those of working age (20-64 years).

3. Specific comments

3.1 Relative risks used

One might have expected that the relative risk estimates used in the calculations would have been derived from up-to-date meta-analyses of the relevant literature. This is not the case at all. Annex VII of the impact assessment[2], on p 205, states that the relative risks "that were applied in the

calculations were identical to those reported by Jamrozik (The Smoke Free Partnership 2006) and the Royal College of Physicians (2005) in the UK. They are based on median figures obtained through meta-review of existing literature and are consistent with the ranges reported in Annex V." Furthermore, these references themselves cite earlier papers. A little digging revealed that the 2006 report cited[3] merely cited the 2005 report[4], and chapter 4 of that report "Deaths from exposure to environmental tobacco smoke in the UK" cited approaches developed in New Zealand, mentioning a paper by Kawachi *et al* in 1989[5] and by Woodward and Laugesen in 2000[6]. Even then, these reports cited yet other sources. Also relevant is a paper by Jamrozik in the BMJ in 2005[7] which used the same relative risks for lung cancer, IHD and stroke as used here.

Having investigated these sources, I think I am able to determine the source of the various estimates.

Lung cancer

The 1.24 for private home exposure comes from the 1997 paper by Hackshaw and Law[8] which presents a meta-analysis of risk in never smoking women in relation to smoking by the spouse. Use of the same figure for workplace exposure derives from Woodward and Laugesen (2000)[6] "based on a review of epidemiological studies". The figure of 1.73 for pub/bar/nightclub exposure comes from Jamrozik (2005)[7] where he suggested multiplying the workplace estimates by 3.04 based on a single salivary cotinine study[9].

Ischaemic heart disease

The estimate of 1.3 for private home exposure comes from a meta-analysis by Law *et al*[10]. That for workplace exposure of 1.2 is stated by Woodward and Laugesen[6] to come from a 1998 paper by Wells[11] and from a 1999 paper by Steenland[12]. Actually, Wells gives 1.18 and Steenland 1.21.

Stroke

The estimate of 1.45 for private home exposure derives from the Jamrozik BMJ paper[7]. It was based on the median from seven studies[13-19]. Jamrozik assumed the same figure for workplace exposure given a lack of evidence.

COPD

The estimate of 1.25 for private home exposure derives from a paper by Law and Hackshaw in 1996[20]. The 2005 report by the Royal College of Physicians[4] assumed that the same estimate applied to workplace exposure.

It is clear that the relative risk estimates in Table 1 do not provide an appropriate summary of the evidence on the associations of ETS with the diseases in question. They are seriously out of date and they omitted relevant references at the time they were produced. This is particularly clear for stroke where Jamrozik[7] overlooked seven studies[21-27] available at the time his paper was produced, most of which found little or no relationship with ETS. Also, the relative risk for stroke was derived using medians, an inappropriate and non-standard technique for summarizing data of this type.

I regularly update meta-analyses for all four diseases, and it is of interest to compare the estimates used (see Table 1 above) with my own most recent estimates[28-30] based on random-effects meta-analysis of the complete available evidence. This gives:

	At home		At work	
	Report	P N Lee	Report	P N Lee
Lung cancer	1.24	1.16	1.24	1.24
Stroke	1.45	1.27	1.45	*
IHD	1.30	1.17	1.20	1.10
COPD	1.25	1.20	1.25	*

(* virtually no data)

Generally the estimates used in the impact assessment[1] and the Rand report[2] are somewhat too high. For lung cancer, this may be due to recent

studies showing lower risks, while for IHD it may reflect failure to consider relevant large studies (e.g. LeVois 1995 and Enstrom 2003[31,32]).

3.2 Interpretation of the summary relative risk estimates

A relative risk is a measure of association, and does not necessarily mean the increase results from a cause and effect relationship. Although, as demonstrated, in Annex V of the impact assessment[1] and Appendix C of the Rand report[2], many sources have reported (as I also have) summary relative risks in excess of 1, this does not mean that these estimates are necessarily reliable estimates of the effect of ETS exposure. They may all be subject to common biases.

For lung cancer, as I have argued at length elsewhere[33,34], the apparent risk increase may be mainly, if not wholly, a result of various sources of bias (including misclassification of active smoking, confounding and including studies that do not adjust for age). For the other causes of death, I have also argued[29,30,35] that causation has not been demonstrated. Even if there is a true effect, it may be substantially different from that indicated by the relative risk.

However, the heading of Table 2 shown in section 1 "… mortality due to ETS exposure" makes it clear that these relative risk estimates, with their many weaknesses, are interpreted as not only causal, but also accurate estimates of the increased risk due to ETS. This is despite the fact that the impact assessment[1] states on p 10 that there is only "suggestive evidence that ETS may cause stroke, .. and chronic obstructive pulmonary disease (COPD) in adults …" (emphasis added).

3.3 Applying never smoker relative risk estimates to smokers

All the relative risk estimates used in the calculations in the impact assessment[1] and the Rand report[2] are from studies of never smokers (lifelong non-smokers). However, the estimates are used to calculate deaths due to ETS for non-smokers (never plus former smokers) and for smokers (current smokers). This renders the analysis speculative to say the least. Also, it is far

from clear how the relative risk estimates have been used to calculate the deaths due to ETS.

The appropriate way to do this is to define a model thought appropriate for the joint effect of smoking and ETS, to calculate the deaths that did occur for each smoking/ETS combination, and then to calculate the deaths that would have occurred had no-one been exposed to ETS. Let me illustrate this by a hypothetical example, in which one is concerned with lung cancer and it is assumed that, in the absence of smoking, ETS multiplies risk by 1.24, and, in the absence of ETS, smoking multiplies risk by 10. Under a multiplicative model, the risk (relative to a non ETS-exposed smoker) is then 12.4, under an additive model it is 10.24. Suppose also there are 10,000 deaths in total, and that the proportions in the lower smoking/ETS groups are as given below.

The calculations then proceed as follows:

Multiplicative model

		Smoking ETS	No No	No Yes	Yes No	Yes Yes	Total Total
1	Proportion exposed		0.4	0.2	0.1	0.3	
2	Relative risk		1	1.24	10	12.4	
3	Relative numbers of deaths		0.4	0.248	1.0	3.72	5.368
4	Actual numbers of deaths		745	462	1,863	6,930	10,000
5	Number if no ETS		745	373	1,863	5,589	8,569
6	Due to ETS		0	89	0	1,341	1,431

Here row 3 comes by multiplying rows 1 and 2, row 4 comes by scaling row 3 to add to the total number of deaths, row 5 comes by multiplying the ETS exposed numbers by 1/1.24, and row 6 by subtraction of the total in row 5 by that in row 4.

Additive model

		Smoking ETS	No No	No Yes	Yes No	Yes Yes	Total Total
1	Proportion exposed		0.4	0.2	0.1	0.3	
2	Relative risk		1	1.24	10	10.24	
3	Relative numbers of deaths		0.4	0.248	1	3.072	
4	Actual numbers of deaths		847	525	2,119	6,508	10,000
5	Number if no ETS		847	424	2,119	6,356	9,746
6	Due to ETS		0	102	0	153	254

It can be seen that the choice of model makes a huge difference, the additive model (inherently more plausible) giving an answer 5 to 6 times less than the multiplicative model.

My initial impression from reading the impact assessment[1] and the Rand report[2] is that they had assumed a multiplicative model, as they talked in various places of basing an attributable fraction merely on the proportion exposed and the relative risk. However, I now believe this is not the case for two reasons.

First, the numbers of deaths attributed to ETS in smokers would, under the multiplicative model, be very much higher than the number of deaths attributed to ETS in non-smokers, and this is not the situation. For lung cancer, the ratio in Table 2 of 761 to 542 = 1.4 is much more like the ratio of 153 to 102 = 1.5 in my additive example than the ratio of 1,341 to 89 = 15.1 in my multiplicative example.

Second, there is reference on page 11 of the impact assessment[1] to having revised the estimates used in the 2006 report "Lifting the Smokescreen"[3] and that refers (on pp 26 and 27) to having first estimated the deaths due to active smoking, then subtracted these from the total, and then calculated the deaths due to ETS from the reduced total. I cannot find any statistical basis for this method of calculation, but if it is applied to the data sets above, the totals attributable to ETS drop from 1,431 to 224 with the multiplicative model, and from 254 to 120 with the additive model. I am not sure how serendipitous it is that the figure of 224 is reasonably close to the correct figure of 254, but it does seem likely there has not been a huge overstatement of deaths in smokers due to ETS.

It still remains true, however, that the estimated number of deaths in smokers is hugely speculative (and also that both the impact assessment[1] and the Rand report[2] are extremely poor in explaining their methodology).

3.4 Incorrect consideration of two exposures

Where there are two exposures (here offices and bars/restaurants), the proper way to do the attributable risk calculation is to consider estimates for four groups – no ETS exposure, office exposure only, bar/restaurant exposure only, and both exposures. The two exposures are likely to be correlated, and as a result the over-simple approach used overestimates risk. [One can see this by considering the simple hypothetical situation where everyone either has both exposures or neither – then as each relative risk relates to both exposures, one ends up with twice the correct numbers of attributable deaths.]

3.5 Age and sex not taken into account

As far as I can see, the calculations for Table 2 are based on estimates of numbers of deaths and population exposed for men and women combined for age 20-64. There seems no indication that separate calculations were made for specific narrower age groups and for the two sexes and the numbers of deaths then combined. Failure to do this would seem highly dubious. Staff working in bars and restaurants are often young, whereas deaths occur predominantly in the older age groups. Failure to take age into account would cause marked overestimation of attributable deaths. This is illustrated in the hypothetical example below, where it is assumed the numbers in each age group are the same and the relative risk is 1.3.

Age	Deaths	Exposed	Attributable fraction	Attributable deaths
20-34	100	0.3	0.0826	8.26
35-40	500	0.2	0.0566	28.30
50-64	2500	0.1	0.0291	72.82
Total (ignoring age)	3100	0.2	0.0566	175.47

The total of 175.47 attributable deaths calculated ignoring age is substantially greater than the total for the three age groups, 109.38.

3.6 Failure to take the healthy worker effect into account

It is well known that numbers of the working population have a lower risk of many diseases, particularly cardiorespiratory, than do those who do not work. This “healthy worker effect” arises because the chronically ill tend not

to work. As the impact assessment[1] and the Rand report[2] have failed to take this into account, the resulting estimated numbers of deaths due to ETS will be somewhat too high.

3.7 What is exposure?

Estimates of prevalence of ETS exposure were obtained (see Annex VII of the impact assessment[1] pp 202-204) from Eurobarometer Surveys conducted in 2006 in all 27 member states, with about 1,000 responses in most of the countries. Subjects were asked about ETS exposure at indoor workplaces/offices and in restaurants/pubs/bars and in each case were counted as positive if they reported exposure for an hour or more. Questions were also asked about where subjects worked and, to be counted as exposed to ETS in indoor workplaces and offices, for example, subjects had to both work and be exposed there. Unlike the 2002 estimates shown in Tables 3 and 4 in section 1 of these comments, which related to the overall effects of ETS exposure at home and at work in the general population, the 2008 estimates only concern effects of ETS exposure at work. Someone may have worked in a smoke-free office and spent his evenings in smoke-filled bars, but would not have been counted as exposed.

Subjects were also asked about their smoking habits, with non smokers including former smokers as well as never smokers, and smokers (actually smoker/tobacco users) not including smokers of packed cigarettes, roll-up cigarettes, cigars or pipe, but also those who chewed tobacco or took snuff.

As the survey was conducted in 2006 and the intent was to provide estimates of deaths for 2008, the exposure prevalences were adjusted downwards to take account of legislation implanted after 2006. It was assumed that for countries introducing full smoke-free legislation after 2006, ETS prevalence rates would fall to the average prevalence of those countries (Ireland, Italy and Sweden) that had already implemented smoke-free legislation prior to 2006, while partial bans were assumed to have half the effect of a full ban.

While the accounting for legislation has merit, there still remains a major problem. Is the average level of exposure for an exposed person equivalent to the average level for a person considered to be exposed in the epidemiological studies? The relative risks used in the calculations come from studies with various definitions of ETS exposure, many conducted years ago when exposure levels were much higher than now.

It seems extremely doubtful whether, even if the epidemiological studies provide an unbiased assessment of the risks of ETS exposure, the actual average level of exposure for a person considered to be exposed based on the Eurobarometer Surveys is in fact equivalent to those in the epidemiological studies.

It is assumed that the extent of exposure for pub/bar/nightclub workers is three times higher than average workplace exposure. This same assumption was used by Jamrozik[7] and is based on one study which relates to nonsmokers and not to the whole population. Given that smokers no doubt get most of their ETS exposure from their own cigarettes, it is rather difficult to imagine that this factor can possibly be true for smokers.

4. Summary

It is clear from my comments above that the estimates of deaths in the impact assessment[1] and the Rand report[2] have little or no scientific foundation. Even if one assumes (which I do not) that meta-analysis estimates of relative risk associated with ETS exposure represent an approximate indication of the increase in risk due to ETS exposure, the estimates of deaths are subject to a number of criticisms:

(i) failure to base relative risk estimates on up to date and comprehensive literature synthesis, so somewhat overstating the strength of the association.

(ii) unjustifiably assuming that relative risk estimates derived from studies of lifelong never smokers also apply equally to former smokers and current smokers.

(iii) failure to take age and sex into account in the calculations, a *sine qua non* in epidemiology, especially when as here death rates are relatively low and employment in bars and clubs relatively high in the young.

(iv) use of a technically incorrect method of calculating attributable deaths from multiple exposures, and

(v) failure to take the "healthy worker effect" into account.

There is also concern that average levels of exposure in subjects classified as exposed in the Eurobarometer Survey which formed the basis of the prevalence estimates used may be different from the average levels of exposure in the studies (often conducted many years ago) on which the relative risk estimates were based.

Another major limitation of the impact assessment[1] is that the methods employed are not properly described and that it provides no useful discussion of the strengths and weaknesses of its approach. A proper discussion would have revealed the glaring weaknesses.

The report ends with a table (reproduced below as Table of this report) comparing the estimated number of deaths from ETS exposure to other health hazards, both in the workplace and in the general population. Given the likely huge inaccuracy of the ETS estimates, one cannot regard such comparisons as particularly meaningful.

Table 5 Benchmarking of deaths attributable to ETS against other risks

Workplace		**General population**	
Exposure to ETS	**6,007**	Road traffic accidents (TREN)	42,953
Accidents at work (OSHA)	7,460	**Exposure to ETS**	**77,449**
Exposure to hazardous substances (OSHA)	73,989	Air pollution (RTD)	>300,000 310,00

The large inaccuracy of the estimates of deaths due to ETS also means that there is little or no point in attempting to evaluate estimates of economic costs and of reductions in deaths and costs due to policy measures, as these depend on the original estimates of deaths being accurate.

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