2.
Tobacco-attributable cancer burden in the UK in 2010

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In 2004, the International Agency for Research on Cancer (IARC) judged that there was sufficient evidence in humans that tobacco smoking causes cancers of the lung, larynx, oral cavity and pharynx, paranasal sinuses, oesophagus, stomach, pancreas, liver, kidney, ureter, bladder, uterine cervix and bone marrow (myeloid leukaemia; IARC, 2004). At a recent expert review (to be published as IARC Monograph 100E), the list of cancers for which the evidence for tobacco smoking being causative was considered to be 'sufficient' was updated to include cancers of the colon and rectum, and mucinous tumours of the ovary (Secretan *et al*, 2009).

In the 2004 evaluation, the IARC judged that there was sufficient evidence that involuntary smoking – that is, exposure to second-hand or 'environmental' tobacco smoke (ETS) – causes lung cancer in humans (IARC, 2004). In this monograph, the results of meta-analyses were reported, showing a statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to second-hand tobacco smoke from the spouse who smokes. The relative risk was 1.24 in women and 1.37 in men after controlling for some potential sources of bias and confounding. The excess risk increases with increasing exposure. For lung cancer in never smokers exposed to ETS at the workplace, the relative risks were 1.19 in women and 1.12 in men. For children exposed to smoke from their parents smoking, the evidence for an increased risk of lung cancer was less consistent.

The reported increases in risk of lung cancer from ETS exposure pertain to non-smokers (indeed, usually to persons who have never smoked). It would be impossible to directly quantify the tiny increment in risk that a smoker might suffer from exposure to another person's smoke (as well as his own). Thus, calculation of attributable fractions will be undertaken only for lung cancer cases in never smokers. This makes sense in that the ultimate aim is to estimate how much cancer is caused by smoking, and this comprises the cases caused by direct smoking and those caused by involuntary smoking in never smokers. Even if a theoretical estimate of the total effect of other persons' smoking was made (including the incremental risk to current and past smokers), this latter component would have to be deducted from the total tobacco-attributable fraction, as involuntary smoking cannot occur without active smoking by others.

TOBACCO SMOKING

Methods

The numbers and percentage of cancers caused by tobacco smoking are estimated using the method developed by Peto *et al* (1992). This is based on the assumption that tobacco smoking is overwhelmingly the most important cause of lung cancer, and that the incidence of this disease in the absence of smoking would be more or less the same in all populations, so that contemporary incidence (or mortality) rates from lung cancer simply reflect the cumulative exposure of a particular population to tobacco smoking. A set of data is required for the calculation, comprising, from the same population, incidence rates of lung cancer in persons who have never smoked and relative risks of different cancers in smokers relative to never smokers. Similar to Peto *et al* (1992), we use the data from the follow-up during 1982–1988 of the American Cancer Society's second 'Cancer Prevention Study' (CPS II; Thun *et al*, 1997), the largest cohort study carried out until now, involving more than a million volunteers aged ⩾30 years at the time of enrolment in 1982 (Garfinkel, 1980; Burns *et al*, 1997). Lung cancer incidence in never smokers has been estimated from the death rates in the CPS II study, for a slightly longer period of follow-up (1982–2002; Thun *et al*, 2006; Figure 1).

The relative risks of death from different cancers during the follow-up period (1984–1988); and the sources are shown in Table 1. Most values listed here were those published in Ezzati *et al* (2005). For cancers of the colon and rectum, the values were those from the follow-up of the CPS II Nutrition Cohort to June 2005 (Hannan *et al*, 2009), in which the multivariate hazard ratios in current smokers were 1.24 in men and 1.30 in women. No data for the risk of mucinous carcinomas of the ovary in smokers have been published based on the CPS II cohort; the value used (2.1) was that from a meta-analysis published by Jordan *et al* (2006).

The first step is to calculate the number of lung cancer cases expected in the UK in the absence of smoking, by applying the age- and sex-specific never-smoker rates (in Figure 1) to the population of the UK in 2010. The number of cases attributable to smoking (and the attributable fraction) is then derived by subtracting the expected cases from the number actually observed in 2010. The results are shown in Table 2.

For the other cancers, the rates in non-smokers are not known, and thus the usual formula for calculating the population

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attributable fraction (PAF) is used:

$$PAF = \frac{P_e(r-1)}{1 + P_e(r-1)}$$

where P_e is the prevalence of exposure and r is the relative risk in smokers.

Using the attributable fractions of lung cancer, already estimated (Table 2) by age group and sex, and the relative risks for lung cancer in smokers from the American cohort (Table 1), the above formula enables calculation of P_e for each age/sex group. This may be thought of as the 'notional' prevalence of smoking (ever vs never) in the UK population – more specifically, the prevalence that would have been necessary in the UK population to produce the observed incidence rates if the relative risks of the CPS II study had pertained.
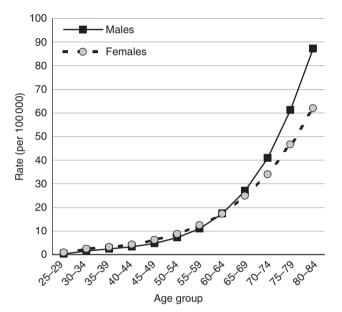
Finally, we use the same formula, the values of prevalence (P_e) and the relative risks for the other cancers (Table 1) to estimate their PAF and, consequently, the numbers of cases attributable to smoking.

'Notional prevalence' (P_e) is an artificial concept that may be quite different from the true prevalence, depending on how different the past experience of tobacco smoking in the population under study was from that in the volunteers of the CPS II study. It can, in fact, even be >1 if a particular age/sex/population cohort has a higher prevalence of smoking and/or a higher relative risk of lung cancer than the CPS II subjects.

Results

For lung cancer (Table 2), the results suggest that about 85% of the lung cancer cases in men are attributable to smoking, and in women the percentage is 80%.

Table 3 shows the estimated numbers of cancer cases at sites other than the lung, and the fractions due to tobacco smoking. (No estimate is made for cancers of the paranasal sinuses, owing to the lack of relevant data on the risk of tobacco smoking; the number of cases concerned would be very few: the total number of cases registered in England in 2008 was 125.)

Figure 1 Age-specific incidence rates of lung cancer in the lifelong never smokers (CPS-II) in the US.

Table 1 Estimated relative risks (RR) for current smokers aged $\geqslant 35$ compared with never-smokers

Cancer	Male	Female
Lung[a]	21.3	12.5
Oral cavity and pharynx[b]	10.9	5.1
Oesophagus[b]	6.8	7.8
Stomach[a]	2.2	1.5
Liver[a]	2.3	1.5
Pancreas[a]	2.2	2.2
Colon−rectum[c]	1.24	1.30
Larynx[b]	14.6	13.0
Cervix[a]	—	1.5
Ovary (mucinous)[d]	—	2.1
Urinary bladder[a]	3.0	2.4
Kidney and renal pelvis[a]	2.5	1.5
Acute myeloid leukaemia[a]	1.9	1.2

[a]From Ezzati et al (2005). [b]From US Department of Health and Human Services (2004). [c]From Hannan et al (2009). [d]From Jordan et al (2006).

Table 2 Cases of lung cancer attributable to smoking, by sex and age group (UK, 2010)

Age group (years)	Population (thousands)	Rates observed	Cases observed	Rates expected[a]	Cases expected[a]	Excess attributable cases	PAF (%)
Males							
0−14	5548	0.0	1	0.0	0	1	0
15−34	8365	0.5	38	0.5	38	0	0
35−44	4387	4.9	215	2.9	128	87	40
45−54	4202	27.1	1138	6.0	252	886	78
55−64	3580	116.9	4184	14.3	513	3705	89
$\geqslant 65$	4526	368.9	16 697	51.5	2331	14 366	86
Total	30 609	72.8	22 273	—	3262	19 011	85
Females							
0−14	5292	0.0	2	0.0	0	2	0
15−34	8048	0.4	42	0.5	42	0	0
35−44	4452	4.5	224	3.8	168	56	25
45−54	4331	26.7	1088	7.4	320	768	71
55−64	3731	85.3	3441	14.9	556	2885	84
$\geqslant 65$	5759	218.0	13 335	42.9	2471	10 864	81
Total	31 614	53.1	18 132	—	3557	14 575	80

Abbreviations: PAF = population-attributable fraction. [a]Expected in a population that had never smoked.

Table 3 Other cases: cases attributable to smoking in 2010

Cases attributable to smoking for specific cancers

Age (years)	Oral cavity and pharynx			Oesophagus			Stomach			Liver			Pancreas			Colon–rectum			Larynx			Cervix			Ovary			Bladder			Kidney and ureter			Myeloid leukaemia		
	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)	Obs.	Exp.	PAF (%)
Males																																				
0–14	5	5	0	0	0	0	0	0	0	15	15	0	0	0	0	1	—	—	0	0	0	—	—	—	—	—	—	—	—	0	54	54	0	41	41	0
15–34	77	77	0	12	12	0	14	14	0	23	23	0	9	9	0	163	163	0	2	2	0	—	—	—	—	—	—	8	8	0	37	37	0	97	97	0
35–44	244	183	25	86	72	16	82	79	4	43	41	4	67	64	4	397	394	1	35	24	31	—	—	—	—	—	—	58	54	6	206	196	5	89	86	3
45–54	902	332	63	469	234	50	251	208	17	166	136	18	278	230	17	1436	1379	4	195	58	70	—	—	—	—	—	—	235	175	26	612	486	21	150	130	13
55–64	1458	325	78	1308	430	67	677	476	30	493	338	31	818	575	30	4129	3807	8	535	92	83	—	—	—	—	—	—	961	564	41	1295	847	35	348	264	24
>65	1887	471	75	3836	1389	64	3443	2524	27	1530	1097	28	2910	2133	27	15999	14912	7	1035	202	81	—	—	—	—	—	—	5448	3390	38	3493	2400	31	1321	1038	21
Total	4573	1393	70	5711	2137	63	4467	3300	26	2270	1650	27	4082	3011	26	22125	20656	7	1802	378	79	—	—	—	—	—	—	6711	4191	38	5697	4020	29	2046	1656	19
Females																																				
0–14	6	6	0	0	0	0	0	0	0	18	18	0	0	0	0	2	—	—	0	0	0	0	0	0	12	12	0	—	—	0	57	57	0	43	43	0
15–34	75	75	0	5	5	0	29	29	0	12	12	0	12	12	0	164	164	0	2	2	0	739	739	0	273	273	0	6	6	0	37	37	0	86	86	0
35–44	131	117	11	27	23	16	64	63	1	29	29	0	53	51	3	402	399	1	12	9	26	679	669	1	438	435	1	31	30	4	110	108	9	90	89	4
45–54	354	191	46	147	61	59	129	117	9	70	64	9	221	177	20	1230	1158	6	53	15	71	411	372	9	926	901	3	104	80	23	298	270	18	142	136	8
55–64	593	208	65	398	98	75	287	234	18	199	163	18	633	411	35	2755	2427	12	105	16	84	317	259	18	1506	1451	4	303	186	39	639	521	16	210	193	7
≥65	1196	466	61	1663	466	72	2067	1735	16	969	813	16	3361	2304	31	13233	11871	10	210	38	82	547	459	16	3665	3571	3	2126	1385	35	2223	1866	15	1025	952	7
Total	2355	1063	55	2240	652	71	2576	2178	15	1298	1099	15	4280	2955	31	17786	16020	10	382	80	79	2693	2498	7	6820	6643	3	2571	1688	34	3364	2860	15	1596	1500	6

Abbreviations: Exp. = cases expected; Obs. = cases observed; PAF (%) = population-attributable fraction.

Table 4 Relative risks of lung cancer from exposure to ETS (IARC, 2004)

Source of exposure to ETS	RR by sex	
	Males	Females
At home (spouse)	1.37	1.24
At work (occupational)	1.12	1.19

Abbreviations: ETS = environmental tobacco smoke; RR = relative risk.

Taking these figures together, we can estimate that, in total, 36 102 (22.8% of the total) cancers in men and 23 722 (15.2% of the total) in women are attributable to smoking tobacco (currently, or in the past).

Discussion

The method of estimation developed by Peto *et al* (1992, 2000) is based on the assumption that the excess mortality (or incidence) from lung cancer, above that which would have been observed in persons who have never smoked, is the result of smoking (past and current). Thus, the attributable fraction of lung cancer can be estimated as

$$(\text{cases observed} - \text{cases expected})/\text{cases observed}$$

and used to estimate the attributable fractions of other cancers. It should be noted that it is of no consequence that the data set used for estimates of PAF in 2010 is derived from study results pertaining to the period 1984–1988, so long as the two components (mortality/incidence of lung cancer in non-smokers, and relative risks of different cancers in smokers *vs* never-smokers) derive from the same population. On the other hand, it is important that the non-smoker rates observed in the US volunteers in 1984–1988 are appropriate to the UK population in 2010. The only large cohort study in the UK was for British Doctors – almost all of them being men. The US CPS II non-smoker rates predicted 19.03 lung cancer deaths in 40 years of follow-up, *vs* 19 actually observed (Peto *et al*, 2000), confirming that non-smoker rates in the UK are likely to be very similar to those in the US CPS II cohort.

The main advantage of the Peto method is that it does not require detailed information of the current relative risks of different cancers in relation to smoking history in the UK population. The risk of tobacco smoking depends on cumulative exposure to carcinogens in tobacco smoke, and therefore varies with the amount smoked, duration of smoking and time since cessation (in ex-smokers), as well as with the type of cigarette smoked. Factors such as these differ between countries, and over time, and thus one cannot be sure that relative risks taken from studies in different populations (geographic or temporal) would be appropriate for the UK in 2010. In the USA, the relative risk of lung cancer in current smokers (relative to never smokers) was 11.5 in men and 2.7 in women in the Cancer Prevention Study I (CPS I) conducted by the American Cancer Society during 1959–1965, whereas it was 23.3 in men and 12.7 in women in CPS II (US Department of Health and Human Services, 2004). In the British Doctors study, the relative risk in current smokers rose from 15.5 during 1951–1971 to 18.5 during 1971–1991 (Doll *et al*, 1994). In fact, one might have expected the switch to cigarettes delivering low tar to have reduced the hazard of lung cancer, but this effect is being offset by the 'maturing' of the smoking epidemic, and thus smokers still alive in more recent years have had a longer history of regular consumption of cigarettes than men of the same ages would have had during the 1950s and 1960s. Another factor that may be important in the maturing of the epidemic (but which is impossible to quantify) is a change in the way cigarettes have been smoked in recent decades. The minority of doctors who continued

to smoke cigarettes in the latter half of the study may have tended to be those who smoked them in a way different from that of the greater number who had stopped smoking them earlier.

Using the ratio of mortality rates from lung cancer in never, former and current smokers after the 50-year follow-up of British doctors (Doll *et al*, 2005), and the prevalence of smoking among British men in 2008 (22% current smokers, 30% ex-smokers; General Lifestyle Survey 2008/ONS 2010, 2010), the estimate of the PAF of lung cancer is 80%. This is somewhat lower than the 85% estimate of the current analysis, and that of Peto *et al* (2006), who, using essentially the same methodology, estimated that 88% of lung cancer deaths in men in the UK in the year 2007 were due to smoking, and 84% of deaths in women. The reason, as noted above, is that the relative risks observed in British doctors are unlikely to be the same as the averages for the UK population in 2010.

ENVIRONMENTAL TOBACCO SMOKE (ETS)

Methods

Estimation of the fraction of cancer caused by exposure to ETS in lifelong non-smokers uses the traditional method for attributable fractions, incorporating estimates of relative risk (of exposure to tobacco smoke) and the prevalence of such exposure among never smokers. The formula for calculating PAF is as follows:

$$\text{PAF} = \frac{P_e(r-1)}{1 + P_e(r-1)}$$

where P_e is the prevalence of exposure and r is the relative risk of lung cancer in those exposed to ETS. The attributable fraction is applied to the number of lung cancer cases estimated to occur among never smokers. From the section on tobacco smoking, this

was estimated to be 6819 (3262 in men and 3557 in women) in the UK in 2010 (Table 2).

We may estimate two components:

(1) Cases of lung cancer (in never smokers) caused by domestic exposure to ETS.
(2) Cases of lung cancer (in never smokers) caused by exposure to ETS in the workplace.

The relative risks from the IARC (2004) meta-analyses, described in the Introduction, are used (Table 4).

Exposure to ETS at home   Most studies investigate the risk of lung cancer in lifelong non-smokers (never-smokers) living with a smoking spouse, and it was on a meta-analysis of such studies that the estimated relative risks in the IARC monograph were based. There appear to be no survey data upon which one can estimate the prevalence of such exposures in the UK. A range of approaches have been used by others, from using the exposure prevalence of control subjects in case–control studies (IARC, 2007) to extrapolation from exposure of children to ETS at home (Jamrozik, 2005). Trédaniel *et al* (1997) estimate the exposure from spouse smoking based on the prevalence of smoking in men and women, and the probability that couples would be discordant for their smoking status. This seems to be the method most likely to yield exposures equivalent to those for which relative risks have been estimated, as well as allowing estimates specific to the UK (which controls from case–control studies cannot). Using data from the General Household Survey for 2008, we may obtain the prevalence of current, ever or never smokers by age group, as well as the probability of being married or cohabiting currently or ever in the past. We use the 'aggregation factor' of 3.0 proposed by Wald *et al* (1986) to express the relative probability of couples being concordant for smoking status.

Table 5 shows the percentage of the UK population who are currently married or cohabiting (column 1), and the percentage

Table 5 Prevalence estimates of cohabitation with smoking partner among non-smokers in UK, and fraction of lung cancer cases attributable to cohabitation with a smoking partner

Age group (years)	Cohabitation status of never-smokers (%)[a]		Population smoking status (%)[a]		Estimated prevalence of never-smokers cohabiting with smoking partner and lung cancer cases attributable to cohabitation with smoking partner (%)[b]					
	1	2	3	4	5	6	7	8	9	10
	Living with a partner	Ever had a partner	Current smokers	Never smokers	Never-smokers living with current smoking partner	PAF	Never-smokers living with ever smoking partner	PAF	Never-smokers ever living with smoking partner	PAF
Men										
16–24	8	8	24	69	1	0.5	2	0.6	2	0.6
25–34	61	63	30	53	10	3.5	19	6.4	19	6.6
35–44	77	82	24	51	11	3.8	25	8.3	26	8.8
45–54	77	88	24	48	11	3.8	24	8.1	27	9.1
55–64	79	93	18	41	7	2.5	16	5.6	19	6.5
65–74	77	94	13	37	4	1.3	20	6.9	25	8.3
⩾75	60	93	13	37	8	2.8	14	5.0	22	7.5
Total	63	72	22	49	6	2.0	17	6.0	20	6.8
Women										
16–24	18	19	26	71	3	1.0	4	1.5	4	1.5
25–34	68	72	29	57	12	4.1	24	8.1	25	8.6
35–44	75	87	25	56	8	3.0	29	9.8	34	11.2
45–54	75	93	24	57	10	3.4	31	10.3	38	12.4
55–64	72	96	22	55	4	1.6	29	9.7	39	12.5
65–74	63	96	13	56	7	2.5	32	10.7	50	15.5
⩾75	29	94	13	56	4	1.6	14	5.1	47	14.8
Total	59	79	21	57	7	2.5	23	7.8	31	10.1

Abbreviations: PAF = population-attributable fraction. [a]Cohabitation status and population smoking status from General Lifestyle Survey 2008/ONS 2010 (2010). [b]Estimates are based on cohabitation status and population smoking status, and assume couples are in the same broad age groups as those in the table and the relative probability of couples being concordant for smoking status is 3.0 (Wald *et al*, 1986).

who have ever been married (or cohabiting; column 2), by age group. Column 3 shows the prevalence of current smokers, and column 4 the percentage of persons who have never smoked.

Under the assumption that couples are in the same broad age groups as those in the table, and that the 'aggregation factor' described above is 3.0, we can estimate the percentage of never smokers who belong to the following categories:

- Currently living with a smoking partner (column 5)
- Currently living with a partner who has ever smoked (column 7)
- Has ever lived with a partner who was a smoker at some point of time (column 9).

The corresponding attributable fractions of lung cancers among never smokers are shown in columns 6, 8 and 10. They range from 2% of lung cancer cases in non-smoking men (due to their current partner's smoke) to 10.1% of lung cancers in non-smoking women, as a consequence of ever having had a partner who was a smoker at some point of time.

Although the relative risks derive from studies of non-smokers with current partners who smoked, the corresponding estimates of PAF in Table 5 (column 6) are probably an underestimate, because of the following factors:

- They take account only of current partnerships, and it is likely that past partnerships with a smoker would have had some

adverse effects, particularly when separation had occurred only recently

- Some non-smoking partners may have quit relatively recently, and their past smoking would have had an adverse effect
- There may be other members of the household smoking, even though the partner does not.

For these reasons, the attributable fractions in column 8 (based on non-smokers with a current partner who was ever a smoker) are taken as the relevant estimate for the UK population.

Exposure to ETS at work   Jamrozik (2005) gives the prevalence of passive smoking at work as 11%, an estimate that probably derives from the survey commissioned by ASH in April 1999, which revealed that approximately 3 million people in the UK are regularly exposed to ETS at work (ASH, 2004). There are otherwise very few data on workplace exposure to ETS in the UK. Chen *et al* (2001), in a small sample derived from participants in the fourth Scottish MONICA survey of 1995, found that any (regular) exposure of adults aged 25–64 years to environmental tobacco smoke at work was 68.1% for men and 57.5% for women (of which 21.5% of men and 17.4% of women classified such exposure as 'some' or 'a lot'). The EPIC study collected data on exposure to ETS at the time of recruitment among 123 000 non-smokers from 11 centres (none of them in UK) during 1993–1998, 78% of whom were women; 67% reported exposure at work (Vineis *et al*, 2005). The proportion of non-smoker controls in the multi-centre

Table 6 Lung cancer cases attributable to exposure of non-smokers to ETS in UK in 2010

| Age group (years) | Spouse | | | Workplace | | | Both | | | | |
| | | | | | | | Independent ETS exposure | | | Correlated ETS exposure | |
	PAF	Obs.	Excess attributable cases	PAF	Obs.	Excess attributable cases	Obs.	Excess attributable cases	PAF (%)	Excess attributable cases	PAF (%)
Men											
16–24	0.01	0	0	0.08	0	0	0	0	—	0	—
25–34	0.06	38	2	0.08	38	3	38	5	13.7	5	12.9
35–44	0.08	128	11	0.08	128	10	128	20	15.5	19	14.6
45–54	0.08	252	20	0.08	252	20	252	38	15.3	36	14.4
55–64	0.06	513	28	0.08	513	40	513	66	12.9	62	12.0
65–74	0.07	854	59	0.08	854	67	854	121	14.2	114	13.3
⩾75	0.05	1478	74	0.08	1478	115	1478	183	12.4	170	11.5
All ages		3262	195		3262	255	3262	434	—	406	—
% of total (all ages)			6.0			7.8		13.3		12.4	
Women											
16–24	0.01	0	0	0.09	0	0	0	0		0	
25–34	0.08	42	3	0.09	42	4	42	7	16.5	7	15.6
35–44	0.10	168	16	0.09	168	15	168	30	18.0	29	17.1
45–54	0.10	320	33	0.09	320	29	320	59	18.5	56	17.6
55–64	0.10	556	54	0.09	556	51	556	100	18.0	95	17.1
65–74	0.11	822	88	0.09	822	75	822	155	18.9	148	18.0
⩾75	0.05	1649	84	0.09	1649	151	1649	227	13.8	212	12.8
All ages		3557	279		3557	325	3557	578	—	547	—
% of total (all ages)			7.8			9.1		16.3		15.4	
Persons											
16–24		0	0		0	0	0	0		0	
25–34		80	6		80	7	80	12	15.2	6	7.3
35–44		296	27		296	25	296	50	16.9	27	9.1
45–54		572	53		572	49	572	98	17.1	53	9.3
55–64		1069	82		1069	91	1069	166	15.5	82	7.7
65–74		1676	147		1676	142	1676	276	16.5	147	8.8
⩾75		3127	158		3127	266	3127	410	13.1	158	5.0
All ages		6820	474		6820	580	6820	1013	—	952	—
% of total (all ages)			6.9			8.5		14.8		14.0	

Abbreviations: ETS = environmental tobacco smoke; Obs. = observed cases; PAF = population-attributable fraction.

European case–control study of Boffetta et al (1998) who reported ever being exposed to ETS at work was 71% in men and 47% in women.

It is difficult, based on such incomplete data, and the varying definition of 'exposure', to decide an appropriate prevalence for the UK. On the basis of the average of the results from Boffetta et al (1998), Chen et al (2001) and Vineis et al (2005), 71% for men and 53% for women, 8% of lung cancers in never-smoking men and 9% in women would be due to workplace exposure to ETS. With the much lower exposure estimate of Jamrozik (11%), the attributable fractions would be 1.3% and 2.0% in men and women, respectively.

RESULTS

Estimate of attributable fraction in lifelong non-smokers

Table 6 shows the final estimates of lung cancer attributable to ETS from the spouse, and at work, with the assumptions described above. With respect to combined exposure, it is assumed that the relative risks are simply multiplicative (no interaction). The exposures are assumed to be either

- independent of each other or
- correlated, in that individuals exposed at home are more likely to be exposed at work. In fact, the concordance between exposures at the two sites is rather weak: on the basis of the results among the control subjects in the study by Boffetta et al (1998), the κ value is -0.005 for women and $+0.05$ for men.

In total, 14–15% of lung cancer cases among individuals who have never smoked are estimated to be due to exposure to ETS.

DISCUSSION

The estimate of the effect of exposure to spousal smoking is based on current (2008) data on the proportion of persons married or cohabiting, and an estimate of the likelihood that their current partner has ever smoked. The percentages are 17% for men (aged over 16) and 23% for women. Self-reported exposure to spousal smoke among controls in the multi-centre European case–control study of Boffetta et al (1998) was reported as 12.8% for men and 62.7% for women – but these are values for those ever exposed, which were used in estimating the PAF in France (IARC, 2007). In the EPIC study, 28.5% of non-smokers (78% women) from 11 centres in Europe (not UK) reported ETS exposure (probably at the time of recruitment) at home (Vineis et al, 2005). The estimates of Jamrozik (2005) − 37% of adults under 65 exposed at home – are clearly inappropriate, as they relate to exposure of children to smoke at home from either parent. In the UK, Jarvis et al (2003), in a sample of adults from the general population of England in 1994 and 1996, found that among 9556 married or cohabiting non-smokers 14.5% had a partner who was a current cigarette smoker. This is similar to the indirect estimate of 17% (men) and 23% (women) who would be expected to have a smoking partner, based on the current prevalence in 2008, and an aggregation factor of 3, on which the result in Table 5 is based. Smoking prevalence has declined over time, and exposure to smoke from a smoking spouse would have been greater in the past (among individuals developing lung cancer in 2010), especially for women, as smoking has declined among men much more than among women. However, as the estimate is based on the probability of the current partner ever having been a smoker, any bias will be small.

The estimate of the role of exposure to ETS in the workplace uses the relative risks from the meta-analysis of case–control studies conducted by IARC (2004). A somewhat more recent meta-analysis of 22 studies (Stayner et al, 2007) suggested a similar magnitude of relative risk (1.24). The definition of 'exposure' in the studies included in these analyses varies, and, in any case, estimates of the PAF depend on the prevalence of workplace exposure to ETS in the UK population, for which there are no representative data.

A previous estimate for deaths attributable to passive smoking in the UK was made by Jamrozik (2005). The results are rather different from those obtained here – 1372 deaths from lung cancer due to exposure at home and 160 due to exposure at work. The

Table 7 Cases of lung cancer attributable to tobacco, by sex and age group (UK 2010)

Age group (years)	Observed cases	Smoking attributable cases	ETS attributable cases	Total attributable cases	
				Excess attributable cases	PAF (%)
Males					
0–34	38	0	5	5	14
35–44	215	87	20	107	50
45–54	1138	886	38	924	81
55–64	4184	3671	66	3737	89
⩾65	16 697	14 366	305	14 671	88
Total	22 273	19 011	434	19 445	87
Females					
0–34	42	0	7	7	17
35–44	224	56	30	86	39
45–54	1088	768	59	827	76
55–64	3441	2885	100	2985	87
⩾65	13 335	10 864	382	11 246	84
Total	18 132	14 575	578	15 153	84
Persons					
0–34	80	0	12	12	15
35–44	439	143	50	193	44
45–54	2226	1654	98	1752	79
55–64	7625	6556	166	6722	88
⩾65	30 032	25 230	687	25 917	86
Total	40 405	33 586	1013	34 599	86

Abbreviations: ETS = environmental tobacco smoke; PAF = population-attributable fraction.

Table 8 Cancer cases caused by exposure to tobacco smoke (by smoking, or environmental), UK 2010

Cancer	Observed cases	Cases in UK, 2010	
		Excess attributable cases Number (% at this site)	Population-attributable fraction (% of all cancers)
Males			
Lung	22 273	19 445 (87)	12.3
Oral cavity and pharynx	4573	3180 (70)	2.0
Oesophagus	5711	3574 (63)	2.3
Stomach	4467	1167 (26)	0.7
Liver	2270	620 (27)	0.4
Pancreas	4082	1071 (26)	0.7
Colon−rectum	22 125	1469 (7)	0.9
Larynx	1802	1424 (79)	0.9
Cervix uteri	—	—	—
Ovary	—	—	—
Bladder	6711	2520 (38)	1.6
Kidney	5697	1677 (29)	1.1
Leukaemia	4639	390 (8)	0.2
All cancers	158 667	36 537 (23.0)	23.0
Females			
Lung	18 132	15 153 (84)	9.7
Oral cavity and pharynx	2355	1292 (55)	0.8
Oesophagus	2817	2009 (71)	1.3
Stomach	2576	398 (15)	0.3
Liver	1298	199 (15)	0.1
Pancreas	4280	1325 (31)	0.9
Colon−rectum	17 786	1766 (10)	1.1
Larynx	382	302 (79)	0.2
Cervix uteri	2693	195 (7)	0.1
Ovary	6820	177 (3)	0.1
Bladder	2571	883 (34)	0.6
Kidney	3364	504 (15)	0.3
Leukaemia	3201	96 (3)	0.1
All cancers	155 584	24 300 (15.6)	15.6
Persons			
Lung	40 405	34 599 (86)	11.0
Oral cavity and pharynx	6928	4472 (65)	1.4
Oesophagus	8528	5583 (65)	1.8
Stomach	7043	1565 (22)	0.5
Liver	3568	819 (23)	0.3
Pancreas	8362	2396 (29)	0.8
Colon−rectum	39 911	3235 (8)	1.0
Larynx	2184	1726 (79)	0.5
Cervix uteri	2693	195 (7)	0.1
Ovary	6820	177 (3)	0.1
Bladder	9282	3403 (37)	1.1
Kidney	9061	2181 (24)	0.7
Leukaemia	7840	487 (6)	0.2
All cancers	314 251	60 837 (19.4)	19.4

reasons for this are different assumptions concerning prevalence of exposure (as mentioned above) and relative risk, and the attribution of no lung cancer deaths after the age of 64 years to workplace exposures. What is more, Jamrozik estimates lung cancer deaths attributable to passive smoking in the whole population – including among current and past smokers; as noted in the introduction, this is illogical, as such deaths would not occur among non-smokers if no one smoked.

SUMMARY

Table 7 summarizes the findings with respect to lung cancer and exposure to tobacco smoke. In total, 34 599 cases of lung cancer in the UK (86% of the total) were due to exposure to tobacco smoke in 2010, the great majority of which (97.4%) are due to active smoking (current or in the past). The figures for men are 87% cases due to exposure to tobacco (of which 97.7% were due to smoking), and for women 84% cases due to exposure to tobacco (of which 96.2% were due to smoking).

Table 8 shows the final summary of the estimate of tobacco-attributable cancer in the UK. In total, the estimate is of 60 837 cancer cases (19.4% of all new cancer cases) attributable to tobacco: 36 537 (23.0%) of cancers in men and 24 300 (15.6%) of cancers in women.

See acknowledgements on page Si.

Conflict of interest

The author declares no conflict of interest.

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