Synthesis of results

Section C1: Attributable fractions : summary and sources of uncertainty

1. Summary of attributable fractions

Tables C1.1 and C1.2 display the overall numbers of incident cancer cases and deaths attributable to risk factors evaluated in this report. It is tempting to sum the figures in these tables to obtain the total proportions of cancer cases and deaths that could be attributed to established risk factors. The percentages presented in Tables C1.1 and C1.2 reflect the effect of removing one cause of cancer independently of other causes. But because cancers have multiple causes, the same cancers can be attributed to more than one cause, so summing the figures in these tables would overestimate the global burden of cancer attributable to the established risk factors. Section C2 on interactions between risk factors provides a more adequate interpretation of the proportions of cancer attributable to each risk factor taking into account the joint effect of two or more of them.

Tobacco smoking and alcohol drinking are by far the main risk factors for cancer in France. The role of infectious agents as causal agents for cancer may be greater than suggested by our estimates because it is likely that many infectious agents involved in cancer remain unknown and the available data on exposure to infectious agents known to be associated with cancer remain imprecise (see Sections B3, E1 and E2). Current scientific knowledge suggests that all other factors would account for a relatively small proportion of all cancers cases and death, but it needs to be stressed that some factors like diet and air pollution deserve further studies for establishing their exact role in cancer occurrence (see Section D3 for detailed discussion of these aspects).

Because of the importance of tobacco smoking, we estimated the specific attributable fraction, separating ever-smokers (current smokers and former smokers) from never-smokers (Table C1.3). The method used was the following:

(i) We first distributed the observed number of cancers in 2000 by cancer site using the attributable fractions calculated in Section B1. For example, among the 3250 deaths in men from bladder cancer, we attributed 1715 to tobacco. We therefore considered these cases as coming from the population of ever-smokers.

(ii) The remaining deaths were distributed according to the prevalence of tobacco smoking, for example, 76% of the remaining 1535 bladder cancers were allocated to the ever-smokers (1165 deaths) and 24% were allocated to the never-smokers (370 deaths).

(iii) The attributable fractions associated with other causes of cancers (calculated in Sections B2 to B10) were applied to these denominators sorted by smoking status to estimate the number of cases attributable to each cause. Then the numbers of deaths according to smoking status were summed across cancer sites.

Applying the method further developed in Section C2 on interactions, we estimated that 50.6% of cancers in ever-smoker men were attributable to a

known cause. In male never-smokers, only 14.0% of cancers could be attributed to a known cause. For female ever-smokers, 31.8% of cancers were associated with a known cause, compared with 15.6% among female never-smokers. Among ever-smokers, cancers associated with tobacco smoking in men represent 67.3% of cancers for which a cause of cancer was attributed and in women 53.8%.

In this analysis, we grouped together current and former smokers. However, because of the lower attributable fraction associated with tobacco in former smokers, the attributable fractions for current smokers should be higher than shown in Table C1.3.

Moreover, no attempt was made to take into account potential interactions with other factors. As mentioned in the next section on interactions (Section C2), causes such as alcohol and occupation have interactions with tobacco smoking, and hence, for full appreciation of the burden of tobacco smoking, a factor of interaction should be included to increase the percentage of cancer associated with tobacco.

It is also worth noting that breast cancer and prostate cancer are included in the denominators, although tobacco smoking is not associated with their occurrence. If these cancers were not included in the denominators, the result would be that more than 60% of cancer in ever-smokers would be attributable to an established risk factor.

2. Sources of uncertainty

We have provided our best estimates of the proportions of specific cancers attributable to specific causes in French men and women in 2000. The uncertainty surrounding these estimates is substantial, and arises from several sources (Table C1.4). In some cases, it would be possible to quantify the uncertainty (e.g., confidence intervals of relative risks and exposure frequencies; alternative scenarios of exposures), while in other cases quantification would be either very difficult (e.g., modelling lag time to provide a biologically-driven estimate of cumulative exposure) or practically impossible (e.g., RR and exposure frequency data from non-comparable populations).

Some authors of systematic reviews of the contributions of different causes to human cancer have provided 'acceptable ranges' around their point estimates. In particular, this was done by Doll and Peto in their 1981 and 2005 publications (Doll and Peto, 1981, 2005). The authors, however, did not provided a rationale for deriving such ranges or intervals, although one appreciates that they intended to reflect the global degree of uncertainty for a particular cancer or risk factor (Table C1.5). For example, Doll and Peto (2005) provided range widths of ± 10% in the case of tobacco and ± 40% in the case of diet: this clearly reflects the stronger evidence available for the former as compared to the latter risk factor, which we have also discussed elsewhere in this report.

To be consistent with our strictly quantitative approach, however, we decided not to provide such ranges, which would necessarily be subjective. We outline below the difficulties in quantifying uncertainty levels of AFs.

First, uncertainty can proceed from known statistical considerations. Most prevalence data and relative risks used in this report were presented with their respective confidence interval or an indication of variability such as population size in surveys. We used a Delta method (Klein, 1953) to estimate uncertainty intervals for the AF estimates in Tables C1.1 and C1.2. Based on Levin's formula, the estimated variance of the AF is of the form:

$$V(AF) = \frac{(e^{\beta} - 1)^2 V(P) + (Pe^{\beta})^2 V(\beta)}{\left[P(e^{\beta} - 1) + 1\right]}$$

where *P* is the prevalence of exposure and ß defined as ln(RR).

When prevalence data were available for the whole population (such as for alcohol consumption or average indoor radon exposure), we considered that the variance of the prevalence data was null.

For EBV infection, HPV infection (for cervix uteri cancer) and asbestos exposure, we directly used an estimate of AF from the literature. No uncertainty interval was available for these causes. Estimation of uncertainty intervals for summary numbers of cases and deaths attributable to infection and to occupational exposure was performed under the hypothesis of no variability for the AF for EBV infection, HPV infection (for cervix uteri cancer) and asbestos exposure.

Table C1.6 presents the number of deaths attributed to each cause with the corresponding uncertainty interval calculated by the Delta method.

Second, various sources of errors in relative risks could have influenced our estimates. Even if a

cause of cancer is clearly established by the IARC, the relative risks available in the literature could be biased towards greater or lower values due to misclassification or selection biases. The use of relative risk estimates from meta-analyses dilutes the effects of biases from a single study. Prevalence data are also highly susceptible to biases, since it is well established that any population-based survey tries to infer values for the whole population, although some populations can hardly be included in survey campaigns. These populations are also known to be more highly exposed to various risk factors such as tobacco or alcohol than the groups included in the surveys. Selection biases (in epidemiological studies or in surveys) cannot be adjusted for by statistical methods. Combining biases in relative risk with biases in exposure prevalence would contribute to increasing the bias in the estimate of AF.

For these reasons, as far as the available data allowed, we used RRs from the most appropriate meta-analyses or epidemiological studies and exposure prevalence data from studies specifically designed to assess exposures. Hence, because we used the "best" estimate of relative risk and prevalence measured with the most suitable methodology, our estimates of AFs were the best that could currently be calculated.

Third, the exposure prevalence data and relative risks were extracted independently. The estimation of AFs requires the use of similar definitions and units of exposure. A small shift in the measurement between the two independent sources could produce a bias in the estimation of AFs. This is especially true if there is misclassification of subjects who should have been classified as unexposed (Wacholder et al., 1994). This could have affected the estimate of the AF for infection, because detection tests for infection may be less sensitive when used on wide populations than tests used in studies designed for accrual of a maximum of infected persons (such as case–control studies). Underestimation of AFs for physical inactivity could also result if the prevalence of inactivity is underestimated; studies on physical activity detail the various types of physical activity and are therefore less susceptible to underreporting, while in surveys it is highly probable that individuals will tend to give a "politically correct" answer. For similar reasons, our occupational prevalence estimates might be higher than the true levels because we used prevalence data from identifiable populations rather than from less exposed populations (e.g., the difference between populations surveyed by the different SUMER surveys in France; see Section B4).

Fourth, our estimates are based on an *a priori* lag time of 15 years, which allows only a crude estimate of AFs. Cancer occurring in 2000 could be caused by exposure that occurred over any period from 1900 to 2000. For example, lung cancer occurring in older age-groups can be attributed to exposure to tobacco starting before 1950, when the prevalence was totally different from what it is now. This arbitrary lag-time is currently the most conservative and plausible value and it produces an average estimate of AFs based on the assumption of no major change in prevalence before or after this time. For most causes such as tobacco, alcohol and infection, of which prevalence in the population tends to change only slowly, the effect of choice of lag time on the AF estimate is expected to be low.

3. Conclusion

In summary, about 35% of all cancer deaths are potentially avoidable because they are due to tobacco, excess in alcohol intake, infectious agents, obesity, lack of physical activity, taking of hormones and excessive sun exposure. Better implementation of preventive regulations at the workplace could also further decrease cancer deaths due to occupational factors. The contribution of the fight against pollutants in cancer control may much smaller, but there is a need for further research on this topic.

References

Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst 1981;66:1191–308.

Doll R, Peto R. Epidemiology of cancer. In: Oxford Textbook of Medicine, 4th edition, Oxford, Oxford University Press, 2005.

Klein LR. A Textbook of Econometrics. New York, Row, Peterson and Company, 1953.

Wacholder S, Benichou J, Heineman EF, et al. Attributable risk: advantages of a broad definition of exposure. Am J Epidemiol 1994;140:303-309

Table C1.1 – Numbers of cancer cases and proportions attributed to various factors in France in the year 2000

	Males		Females		Both sexes	
Risk factors*	**Number**	**% of all cancers**	**Number**	**% of all cancers**	**Number**	**% of all cancers**
Tobacco	43 466	27.0	7095	6.1	50 561	18.2
Alcohol	17 398	10.8	5272	4.5	22 670	8.1
Infectious agents	4206	2.6	4871	4.2	9077	3.3
Physical inactivity	780	0.5	5541	4.7	6321	2.3
Obesity and overweight	2249	1.4	3899	3.3	6148	2.2
Ultraviolet light	2380	1.5	3234	2.8	5614	2.0
HRT-OC	–	–	5828	5.0	5828	2.1
Occupation	4013	2.5	314	0.3	4327	1.6
Reproductive factors †	–	–	2260	1.9	2260	0.8
Pollutants ‡	119	0.07	179	0.15	298	0.1

HRT-OC: Hormone replacement therapy and oral contraceptive use

* Ranked according to number of cancer cases in both sexes

† Change in reproductive factors between 1980 and 2000

‡ Several factors such as air particulate matter were not taken into account (see Section D3). If 50% of French population was exposed to air particulate matter concentrations associated with an increase in lung cancer risk of 7%, then in this table, 0.83% of all cancers in men and 0.4% of all cancers in women would be attributable to pollutants

Table C1.2–Numbers of cancer deaths and proportions attributed to various factors in France in the year 2000

	Males		Females		Both sexes	
Risk factors*	**Number**	**% of all cancers**	**Number**	**% of all cancers**	**Number**	**% of all cancers**
Tobacco	28 934	33.4	5449	9.6	34 383	23.9
Alcohol	8188	9.4	1692	3.0	9880	6.9
Infectious agents	2867	3.3	2511	4.4	5378	3.7
Occupation	3183	3.7	256	0.5	3439	2.4
Obesity and overweight	995	1.1	1321	2.3	2316	1.6
Physical inactivity	427	0.5	1812	3.2	2239	1.6
HRT-OC	–	–	1239	2.2	1239	0.9
Ultraviolet light	548	0.6	499	0.9	1047	0.7
Reproductive factors †	–	–	606	1.1	606	0.4
Pollutants ‡	107	0.12	165	0.3	272	0.2

HRT-OC: Hormone replacement therapy and oral contraceptive use

* Ranked according to number of cancer deaths in both sexes

† Change in reproductive factors between 1980 and 2000

‡ Several factors such as air particulate matter were not taken into account (see Section D3). If 50% of French population was exposed to air particulate matter concentrations associated with an increase in lung cancer risk of 7%, then in this table, 0.83% of all cancer deaths in men and 0.4% of all cancer deaths in women would be attributable to pollutants

Table C1.3–Proportions of cancer deaths attributed to various factors according to smoking status in the absence of interaction between tobacco and other factors

	Males		Females	
	Ever-smokers*	Never-smokers	Ever-smokers*	Never-smokers
Risk factors	**AF (%)**	**AF (%)**	**AF (%)**	**AF (%)**
Tobacco	39.7	–	19.3	–
Alcohol	10.0	6.7	2.9	3.0
Infection	3.1	3.0	4.8	3.9
Obesity and overweight	1.1	1.4	2.1	2.5
Inactivity	0.4	0.7	2.8	3.5
Ultraviolet light	0.5	0.9	0.7	0.9
HRT-OC	–	–	1.9	2.4
Occupation	4.0	1.9	0.7	0.3
Pollutants	0.1	0.05	0.5	0.1
Total §	**50.6**	**14.0**	**31.8**	**15.6**

HRT-OC: Hormone replacement therapy and oral contraceptive use
* Current or former smokers
§ The overall AF was estimated considering multiplicative interaction as described in Section C2

Table C1.4 - Sources of uncertainty in the estimation of attributable cancers

Component of AF	Source of uncertainty	Explanation, examples	Quantitative aspects
Relative risk	Random error	Relative risks (both in individual studies and in meta-analyses) are subject to random variability that depends mainly on the size of the study populations	Quantifiable (confidence interval)
	Bias	Relative risks may be biased because of residual confounding and lack of proper control of bias in the original studies	Qualitative assessment possible; quantification difficult
Exposure frequency	Random error	Exposure frequency data are subject to random variability, that depends on the size of the study populations	Quantifiable (confidence interval)
	Bias	Surveys and other studies on exposure frequency may be subject to selection and information bias	Qualitative assessment possible; quantification difficult
Correspondence of relative risk and exposure data	Geographic correspondence	Relative risks and/or exposure frequency data derived from different populations and/or from populations other than that under study	Quantification difficult
	Temporal correspondence	Relative risks and/or exposure frequency data refer to different time periods and/or populations other than that under study; exposure data refer to a time period irrelevant for the carcinogenic effect of the risk factor	Modelling and alternative exposure scenarios feasible
	Substantive correspondence	Relative risks and exposure frequency data refer to different entities (even partially so)	Quantification difficult

Table C1.5. - Factors applied by Doll and Peto (2005) to calculate 'acceptable ranges' of estimates of attributable factors in United Kingdom

Risk factor	Uncertainty factor
Tobacco	1.1
Alcohol	1.33
Ionizing radiation	1.2
Ultraviolet light	1
Infection	3
Medical drugs	NA*
Occupation	2.5
Pollution	2.5
Diet	1.4
Reproduction	1.33
Physical inactivity	NA*

NA: Not available

* In the case of medical drugs and physical inactivity, the best estimate is < 1% and the acceptable range 0–1%

Table C1.6 – Uncertainty intervals (UI) of number and proportion of deaths associated with various factors based on a 95% CI of RRs and exposure frequency estimates

	Males					Females			
Cause	**No.**	**95% UI**	**%**	**95% UI**		**No.**	**95% UI**	**%**	**95% UI**
Tobacco	28 934	[27 219–30 649]	33.4	[31.4–35.3]		5449	[4930–5968]	9.6	[8.7–10.5]
Alcohol	8188	[7578–8797]	9.4	[8.7–10.1]		1692	[1469–1914]	3.0	[2.6–3.4]
Infection	2867	[2252–3482]	3.3	[2.6–4]		2511	[2310–2712]	4.4	[4.1–4.8]
Occupation	3183	[2753–3612]	3.7	[3.2–4.2]		258	[224–291]	0.5	[0.4–0.5]
Obesity and overweight	995	[801–1189]	1.1	[0.9–1.4]		1321	[1212–1429]	2.3	[2.1–2.5]
Physical inactivity	427	[152–702]	0.5	[0.2–0.8]		1812	[808–2816]	3.2	[1.4–4.9]
HRT-OC	–	–	–	–		1239	[1089–1390]	2.2	[1.9–2.4]
Ultraviolet light	548	[469–627]	0.6	[0.5–0.7]		499	[427–571]	0.9	[0.8–1]
Pollutants	107	[0–269]	0.12	[0–0.3]		165	[117–214]	0.3	[0.2–0.4]

HRT-OC: Hormone replacement therapy and oral contraceptive use

Section C2: Interactions between cancer risk factors

Cancer arises through inherited or acquired genetic alterations in multiple pathways involved in cell replication, proliferation and growth (Hanahan and Weinberg, 2000). As a first approximation, each such alteration can be caused by inherited conditions, endogenous factors or exogenous carcinogens, including the risk factors reviewed in this report. Cancer can therefore be described as the result of a multistep process and as a multifactorial disease; this view not only helps in understanding the molecular and cellular mechanisms of carcinogenesis, but offers a framework to interpret the results of observational studies which suggest an 'interaction' between different risk factors.

1. Biological interaction

Although the precise role played at the molecular and cellular level by known carcinogens is in most cases unknown, it is plausible that certain carcinogens, in particular those consisting of complex mixtures such as tobacco smoke, act on more than one step of the carcinogenesis pathway. This is consistent with the epidemiological evidence of tobacco acting both as an 'early-stage' (e.g., as a mutagen) and a 'late-stage' (e.g., as a promoter) carcinogen (Tubiana, 1999, Hazelton et al., 2005).

A practical consequence of the multifactorial nature of cancer and of interactions between carcinogens is that the same cases of cancer can be attributed to more than one risk factor. This notion has far-reaching implications in the interpretation of estimates of attributable cancers such as those presented in this report. First, we should aim at identifying risk factors that explain more than 100% of a specific cancer when their individual effects are summed. Second, any estimate of the 'global' burden of cancer attributable to multiple causes should take into account the overlap between the effects of different carcinogens. As a consequence, for a specific cancer, the attributable fraction for all risk factors considered together should be smaller than the mere sum of the AFs associated with each risk factor.

The independence of the effects of risk factors, leading to multiplicative effects of relative risks, as outlined in Table C2.1, is the default assumption in most calculations of attributable fractions. It is based on the hypothesis that different risk factors act on different carcinogenic pathways. This choice is justified by the lack of detailed quantitative data on the risks resulting from combined exposure to several risk factors. Indeed, the statistical power needed to demonstrate an interaction is lacking in the vast majority of epidemiological studies. The hypothesis of the multiplicative effect of relative risks can be considered as reasonable since it has already been described at least for the two main risk factors, tobacco smoking and alcohol drinking, as risk factors for laryngeal cancer (Figure C2.1). This multiplicative effect has been further confirmed in relative risk models (Roy and Estève, 1998). However, a model with less than multiplicative interaction seems to best fit the data on combined exposure to asbestos and tobacco smoke with respect to lung cancer risk (Vainio and Boffetta, 1994).

A detailed quantitative review of all combinations of risk factors goes beyond the scope of this report, but the reader should be aware of the following conclusions:

a) the number of attributable cancers due to a combination of risk factors is less than the sum of the number attributable to each of the risk factors;

b) prevention of the same cancers can take place through multiple interventions; in other

words, prevention of one cause of cancer may also reduce the number of cancers due to another cause;

c) estimates of attributable cancers adding up to a total of 100% are not biologically or statistically correct.

2. Interaction between risk factors considering independence of risk factors

Although the available epidemiological data support the notion of interaction between risk factors, in most instances they fall short of conclusively demonstrating its precise nature. To assess the importance of interactions for AFs of cancer, we estimated the AF for the combination of exposures under the hypothesis of independent exposures and effect. This hypothesis implies the multiplication of relative risks in the case of combined exposures. For two risk factors, A and B, the AF of exposure to either factor is given by:

$$AF = \frac{p_A p_B (RR_A RR_B - 1) + p_A (1 - p_B)(RR_A - 1) + p_B (1 - p_A)(RR_B - 1)}{p_A p_B (RR_A RR_B - 1) + p_A (1 - p_B)(RR_A - 1) + p_B (1 - p_A)(RR_B - 1) + 1}$$

where P_A and P_B are the prevalences of exposure to factors A and B, and RR_A and RR_B are the corresponding relative risks. This formula can be written as:

$$AF = AF_A + AF_B - (AF_A \times AF_B)$$

This formula can be generalized to more than two risk factors. This approach allowed us to estimate the fraction attributable to established risk factors for all cancers in 2000.

We calculated the combined AF for selected risk factor–cancer mortality associations in men and women (Tables C2.2 and C2.3), as well as in both sexes combined (Table C2.4). These tables show that, in the case of a risk factor with high relative risk, the contribution of additional risk factors to the combined AF is small. For instance, for lung cancer in men, the AF is 83% for tobacco only, and adding the effect of occupation and pollutants only increases the overall percentage of lung cancer attributed to one of these causes to 85%. However, given the uncertainties in current knowledge of the biological

interactions between different cancer risk factors, the figures presented in Tables C2.2–C2.4 should be interpreted with caution.

References

Hanahan D, Weinberg RA. The hallmarks of cancer. Cell 2000;100:57–70.

Hazelton WD, Clements MS, Moolgavkar SH. Multistage carcinogenesis and lung cancer mortality in three cohorts. Cancer Epidemiol Biomarkers Prev 2005;14:1171–1181.

Roy P, Estève J. Using relative risk models for estimating synergy between two risk factors. Stat Med 1998;17:1357–1373.

Tubiana M. Contribution of human data to the analysis of human carcinogenesis. Comptes Rendus Acad Sc, Sciences de la vie / Life sciences 1999;322:215-224.

Tuyns A, Estève J, Raymond L, et al. Cancer of the lartnx/hypopharynx, tobacco and alcohol : IARC international case-control study in Turin and Varese (Italy), Zaragosa and Navarra (Spain), Geneva (Switzerland) and Calvados (France). Int J Cancer 1988; 41: 483-491.

Vainio H, Boffetta P. Mechanisms of the combined effect of asbestos and smoking in the etiology of lung cancer. Scand J Work Environ Health. 1994;20:235–242.

Table C2.1–Interaction between two risk factors A and B

		Risk factor A	
		–	+
Risk factor B	–	RR=1	RR_A
	+	RR_B	RR_{AB}

– Multiplicative model of interaction: $RR_{AB} = RR_A \times RR_B$
– Presence of positive interaction: $RR_{AB} > RR_A \times RR_B$

Table C2.2. Summary of attributable fractions of cancer deaths (%) and estimate of the overall attributable fraction to an established risk factor by cancer site for men in France in 2000

Cancer site	Tobacco	Alcohol	Infection	Obesity and overweight	Inactivity	UV light	Occupation	Pollutants	Total
Bladder	52.8						5.1		55.2
Central nervous system									0.0
Colon-rectum		11.2		6.6	5.1				21.3
Gallbladder									0.0
Hodgkin lymphoma			40.0						40.0
Kidney	26.4			14.6					37.2
Larynx	75.9	57.3					3.1		90.0
Leukaemia							4.1		4.1
Liver	37.5	31.8	32.4						71.2
Lung	83.0						11.3	0.4	85.0
Melanoma						71.1			71.1
Mesothelioma							83.2	2.4	83.6
Multiple myeloma									0.0
Non-Hodgkin lymphoma			8.0						8.0
Oesophagus	51.1	55.2		5.0					79.2
Oral cavity and pharynx	71.5	70.7	6.7						92.2
Pancreas	24.9								24.9
Prostate									0.0
Sinonasal							27.0		27.0
Stomach	31.1		18.1						43.6
Thyroid									0.0
All cancer	33.4	9.4	3.1	1.1	0.5	0.6	3.7	0.1	42.5

The total for each cancer represents the overall percentage of cancers attributed to an established risk factor

Table C2.3. Summary of attributable fractions of cancer deaths (%) and estimate of the overall attributable fraction to an established risk factor by cancer sites for women in France in 2000 *

Cancer site	Tobacco	Alcohol	Infection	Obesity and overweight	Inactivity	UV light	HRT-OC	Occupation	Pollutants	Total
Bladder	39.3							0.6		39.6
Breast		9.4		4.8	10.1		10.7			30.8
Central nervous system										0.0
Cervix uteri*	22.9		100							100.0
Colon-rectum		2.7		4.9	9.2					16.0
Corpus uteri				17.8						17.8
Gallbladder										0.0
Hodgkin lymphoma			40.0							40.0
Kidney	11.5			11.3						21.5
Larynx	64.8	17.8						0.3		71.2
Leukaemia								0.4		0.4
Liver	17.1	8.4	25.1							43.1
Lung	69.2							4.5	3.8	71.7

Table C2.3. cont'd - Summary of attributable fractions of cancer deaths (%) and estimate of the overall attributable fraction to an established risk factor by cancer sites for women in France in 2000 *

Cancer site	Tobacco	Alcohol	Infection	Obesity and overweight	Inactivity	UV light	HRT-OC	Occupation	Pollutants	Total
Melanoma						71.1				71.1
Mesothelioma								38.4	2.4	39.9
Multiple myeloma										0.0
Non-Hodgkin lymphoma			8.0							8.0
Oesophagus	34.4	16.9		7.3						49.4
Oral cavity and pharynx	28.5	24.6	6.7							49.7
Ovary							1.9			1.9
Pancreas	17.0									17.0
Sinonasal								6.5		6.5
Stomach	14.3		18.1							29.9
Thyroid										0.0
All cancer	9.6	3.0	4.4	2.3	3.2	0.8	2.2	0.5	0.3	23.6

The total for each cancer represents the overall percentage of cancers attributed to an established risk factor
HRT-OC: Hormone replacement therapy and oral contraceptive use
* For cervix uteri all cancers are attributed to infection

Table C2.4. Summary of attributable fractions of cancer deaths (%) and estimate of the overall attributable fraction to an established risk factor by cancer sites for both sexes combined in France in 2000 *

Cancer site	Tobacco	Alcohol	Infection	Obesity and overweight	Inactivity	UV light	HRT-OC	Occupation	Pollutants	Total
Bladder	49.6							4.0		51.6
Breast		9.4		4.8	10.1		10.7			30.8
Central nervous system										0.0
Cervix uteri*	22.9		100.0							100.0
Colon-rectum		7.2		5.8	7.1					18.7
Corpus uteri				17.8						17.8
Gallbladder										0.0
Hodgkin lymphoma			40.0							40.0
Kidney	20.9			13.4						31.5
Larynx	75.0	54.1						2.9		88.8
Leukaemia								2.3		2.3
Liver	32.6	26.1	30.6							65.5
Lung	80.6							10.1	1.0	82.8

Table C2.4. cont'd - Summary of attributable fractions of cancer deaths (%) and estimate of the overall attributable fraction to an established risk factor by cancer sites for both sex in France in 2000 *

Cancer site	Tobacco	Alcohol	Infection	Obesity and overweight	Inactivity	UV light	HRT-OC	Occupation	Pollutants	Total
Melanoma						71.1				71.1
Mesothelioma								73.8	2.4	74.4
Multiple myeloma										0.0
Non-Hodgkin lymphoma			8.0							8.0
Oesophagus	48.3	48.8		5.3						74.9
Oral cavity and pharynx	64.7	63.4	6.7							88.0
Ovary							1.9			1.9
Pancreas	21.2									21.2
Prostate										0.0
Sinonasal								20.9		20.9
Stomach	24.6		18.1							38.2
Thyroid										0.0
All cancer	23.9	6.9	3.6	1.6	1.6	0.7	0.9	2.4	0.2	35.2

The total for each cancer represents the overall percentage of cancers attributed to an established risk factor
HRT-OC: Hormone replacement therapy and oral contraceptive use
* For cervix uteri all cancers are attributed to infection

Figure C2.1 - Relative risk of laryngeal cancer for tobacco smoking and alcohol drinking in a study from Southern Europe (Tuyns et al., 1988)