The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010

Introduction

The overall objective of the study is to estimate the percentage of cancers (excluding non-melanoma skin cancer) in the UK in 2010 that were the result of exposure to 14 major lifestyle, dietary and environmental risk factors: tobacco, alcohol, four elements of diet (consumption of meat, fruit and vegetables, fibre and salt), overweight, lack of physical exercise, occupation, infections, radiation (ionising and solar), use of hormones and reproductive history (breast feeding). The number of new cases attributable to suboptimal exposure levels in the past, relative to a theoretical optimum exposure distribution, is evaluated. For most of the exposures, the attributable fraction was calculated based on the distribution of exposure prevalence (around 2000), the difference from the theoretical optimum (by age group and sex) and the relative risk per unit difference. For tobacco smoking, the method developed by Peto et al (1992) was used, which relies on the ratio between observed incidence of lung cancer in smokers and that in non-smokers, to calibrate the risk. This article outlines the structure of the supplement – a section for each of the 14 exposures, followed by a Summary chapter, which considers the relative contributions of each factor to the total number of cancers diagnosed in the UK in 2010 that were, in theory, avoidable.

SELECTION OF RISK FACTORS

Among dietary, lifestyle and environmental factors, those that fulfilled the following criteria were selected:

(i) There was sufficient evidence on the presence and magnitude of likely causal associations with cancer risk from high-quality epidemiological studies.
(ii) Data on risk factor exposure were available from nationally representative surveys.
(iii) There were achievable alternative exposure levels that would modify the risk.

Several other risk factors were considered but were not included because the evidence on causal effects was less convincing, or because their effects on national cancer incidence were likely to have been small and estimates of relevant past exposures difficult to obtain. This is discussed further below.

SOURCES OF DATA

(1) The risks of exposure (aetiological effect sizes) were taken from published systematic reviews and meta-analyses of epidemiological studies.
(2) Risk factor exposure distributions were obtained from nationally representative health examination and interview surveys. Data on prevalence of risk factors from epidemiological studies (cohort or case–control) were not used, as such...
Table 2 compares the numbers of cases diagnosed in 2007 with the projected numbers for 2010.

AETIOLOGICAL EFFECTS OF RISK FACTORS ON DISEASE-SPECIFIC INCIDENCE

The relative risk (RR) per unit of exposure or for each exposure category (for risks measured in categories) was obtained for cancers with probable or convincing causal associations with each risk factor. The studies used for aetiological effect sizes were observational studies (prospective cohort studies whenever possible) that estimated the effects relative to baseline exposure. The RRs used in the analyses represent the best evidence for the impact of risk factor exposure on cancer risk in the UK population, based on the current causes and determinants of the population distribution of exposure. Relative risks adjusted for major potential confounders were used to estimate the causal components of risk factor–disease associations. With respect to diet, for example, the relative risks for specific components – for example, meat – have generally been adjusted for intake of other components with which they may be confounded, as well as for total energy intake. However, if there is also a correlation between exposure and risk of a specific cancer, due to correlations of exposure with other risks or other unobserved factors, the above equations may result in under- (when there is positive correlation) or over-estimation (negative correlation) of the true PAF when used with adjusted RRs (Bruzzi et al, 1985).

The cancers that occur in a particular year, related to specific risk factors, are presumably related to cumulative exposures to the factor concerned over a period of many years. For tobacco smoking, for example, the risk of lung cancer relates to the duration and intensity of exposure with other risks or other unobserved factors, the above equations may result in under- (when there is positive correlation) or over-estimation (negative correlation) of the true PAF when used with adjusted RRs (Bruzzi et al, 1985).

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cumulative exposure to tobacco smoke (duration and dose), including the time since quitting in ex-smokers. Similarly, the total lifetime exposure to ionising radiation for individuals in each age group in 2010 was estimated on the basis of known or estimated levels of exposure in the past. Such detailed quantification of risk is not available for most exposures, and, even if it was, it would be impossible to partition the 2010 UK population according to the appropriate categories of past exposure. Therefore, for several exposures, an arbitrary latent period was included, which is the average interval between ‘exposure’ and the appropriate increase in risk of the cancers concerned. The most appropriate period was deemed to be the mean interval between measurement of exposure and cancer outcome in the prospective studies that were used as the source of data on relative risks. For most exposures, this was around 10 years, and thus the effects on cancers occurring in 2010 of suboptimal levels of exposure in 2000 were examined. When there was evidence about the duration between exposure and change in risk (for example, exposure to radiation, or exogenous and endogenous sex hormones), the appropriate interval was used to select the year for which exposure data were obtained. The method used for estimating the attributable fraction of the most important exposure – smoking – does not require estimation on the basis of past exposure, and so no such assumptions are needed (although, in fact, the latency between exposure to cigarette smoking and lung cancer risk (at least) is well documented).

Many calculations of PAFs are based on current levels of exposure to risk factors; for example, the work of the Global Burden of Disease/Comparative Risk Assessment Group (Ezzati et al, 2002; Danaei et al, 2005) or the World Cancer Research Fund (WCRF/AICR, 2009). Although this simplifies the business of obtaining data on prevalence of the different exposures, the effect being imputed must relate to cancers that will be caused by these exposures at some variable, and undefined, period in the future.

To measure the effects of non-optimal levels of exposure, one must define, for each exposure, an optimal exposure distribution, sometimes referred to as the theoretical-minimum-risk exposure distribution (TMRED), against which the excess risk due to actual exposure is evaluated. The optimal exposure may be zero for risk factors for which zero exposure is imaginable, and results in minimum risk (e.g., no tobacco smoking, alcohol drinking or consumption of red meat). For some exposures (e.g., BMI, solar radiation, salt consumption), zero exposure is physiologically impossible. For these risks, we used optimal exposure levels corresponding to accepted recommendations for the UK population, or, for UV radiation, corresponding to those observed in a population with an attainable low level of exposure (Table 1). The ‘optimum’ exposure levels for factors with protective effects (physical activity, and dietary fruit and vegetable and fibre intake) were selected as the intake and activity levels recommended for the UK population (Table 1). Strictly speaking, these baselines should be called ‘recommended levels’, as benefits may continue to accrue at higher (for preventive exposures) or lower (for carcinogenic exposures) levels, but the terminology of ‘optimum’ is retained for consistency. The optimum exposure levels (TMREDs) should obviously be identical in calculations for the effect of the same exposure on different cancers.

The fraction of cancer cases considered to be attributable to a given exposure is based on estimating the effect of bringing all those individuals at suboptimal levels to the exact level of the optimum baseline, without changing (improving) the exposure (and risk) of those individuals who already exceed it. This approach is a conservative one. In other studies, for example, that of the WCRF (2009), attributable fractions are based on the estimated effect of moving all those in suboptimal exposure categories to the most favourable one (in which the mean exposure is considerably higher than the optimum baseline).

The analyses use data on the fraction of the UK population at different levels of exposure, and estimates of the risk associated with each, relative to the optimum exposure. The PAF is given by the following equation:

$$PAF = \frac{\sum_{i=1}^{n} (p_i \times ERR_i)}{1 + \sum_{i=1}^{n} (p_i \times ERR_i)}$$

where $p_i$ is the proportion of the population in exposure level $i$ and $ERR_i$ the excess relative risk (relative risk – 1) at exposure level $i$.

The calculation is carried out separately by sex and age group (the choice of which depended on availability of exposure data).

The method of estimation of PAF follows the same principle for the different exposures, although some variations to the formula above are necessary depending on the type of exposure and the availability of pertinent data; they are presented in detail in each chapter. For tobacco smoking, the method developed by Peto et al (1992) was used, which relies on the ratio between observed incidences of lung cancer in smokers and that in non-smokers, to calibrate the risk.

Because the current (2010) cancer risk is, for most of the factors considered, related to past exposures that occur only in adulthood (age 15+), or for which data are available only for adults, PAFs can be calculated only for ages ≥ 25, when the latency between exposure and outcome is 10 years. Even where a fraction of cases occurring at ages < 25 are related to childhood exposure, the effect of ignoring these on the estimate of the total PAF (at all ages) will be very small, owing to the rarity of cancer in the age group of 15–24 years.

A separate section is devoted to each lifestyle/environmental factor, for which the number of cases of different cancers attributable to suboptimal levels exposure is estimated. This is expressed also as a percentage of the observed number of cases in 2010. The total number of cancer cases (all sites) attributable to each risk factor was obtained by summing the numbers at the individual sites. Cases of different cancers attributable to a single risk factor are additive because each cancer case is assigned to a single ICD category.

In a summary chapter, the estimates for the 14 different exposures are listed together, and the numbers of cancer cases caused by all of them functioning individually, or in combination, are estimated. See acknowledgements on page Si.

Conflict of interest

The author declares no conflict of interest.
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