PAPERS AND SHORT REPORTS

Analysis of trends in cancer mortality in England and Wales during 1951-80 separating changes associated with period of birth and period of death

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Abstract

Cancer mortality rates in England and Wales were analysed so to describe simultaneously changes affecting successive generations—that is, associated with period of birth—as well as changes associated with the period in which the deaths took place. When mortality from all cancers was considered the analysis implied that, contrary to a widely held view, the rate of death from cancer had been declining in each sex in successive generations. For men the decline had occurred in generations born since 1900, whereas for women the peak came in the 1925 birth group. On the other hand, there had been little decline in the rates associated with period of death. Five examples of cancers of specific organs for which the trends contrasted are shown.

Introduction

Changes in published death rates over a period of time may be due to alterations in the incidence or fatality of the disease being considered or may merely reflect changes in classification or diagnosis. Though mortality data have limitations, so far as cancer is concerned they usually provide more reliable information than morbidity data in the interpretation of time trends.[1] Changes in mortality should therefore be recognised promptly and interpreted correctly.

In diseases like cancer, where many aetiological factors act over an extended period, incidence (as reflected in the death rate) is the result of various influences. Some will act long before the appearance of the neoplasm, while others act later. Cancer in the aged may result from exposure to a hazard which began long before (and continued or stopped). Hence current high rates (crude or standardised) do not necessarily reflect current exposure to new generations. Incorrect interpretations have led to misplaced alarm.[2] [3] The best indicator of the future is the study of mortality among the youngest generations.

We have analysed trends in age-specific rates by separating the variations attributable to age at death, period of birth (or cohort), and period of death. Three curves, one for each of these factors, were fitted to the mortality rates. We characterised each cohort by its central year of birth and each period of death by its date of death. It is possible to distinguish changes of rates which are identified with particular generations from those related to a period. Diseases with long periods between cause and effect (say, death), such as lung cancer, often show changes related to generations, while diseases with more immediate causes, such as an epidemic infection, may show period effects unrelated to generation. Changes in diagnosis, classification, and treatment are likely to show period changes. Our statistical analysis was designed to assess the contributions of these two types of influence.

Methods

The basic data were the death rates for England and Wales in five-year calendar periods for five-year age groups and according to sex and site of disease as published by the Office of Population Censuses and Surveys.[4-7] To limit inaccuracies in diagnosis as far as possible we restricted our analysis to deaths occurring under 70 and to deaths since 1950 and up to the end of 1980.[1] The lower limit of the range of age considered for different cancers was varied to ensure that no age-specific death rate was based on fewer than 20 deaths.

The statistical technique was modified from one described by Barrett[8] and followed a concept described by Kermack et al in 1934.[9] Each age-specific death rate was regarded as the product of three numbers—an age value, a period of birth (or cohort) value, and a period of death value. Cohort and period of death values were given an "average" value of unity, making the age values similar to the age-specific death rates themselves. Since knowledge of any two of age at death, period of birth, and period of death necessarily implies knowledge of the third, these three factors are not statistically independent.

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The methods that we used to surmount this difficulty are described briefly below and in detail elsewhere (Osmond and Gardner, paper in preparation).

The dependence implies that there is no unique set of age, cohort, and period of death values which are solutions to the model, though the alternatives are related. This difficulty does not arise when the age-specific death rates are regarded as the product of only any two of the three factors, and we used this to make progress. Each of the three possible two-factor models (age and cohort, age and period of death, cohort and period of death) was solved separately, and then an overall solution to the three-factor case was obtained by a suitably weighted technique. This was related both to how well the two-factor results fitted the original data—that is, the age-specific death rates—and to how close they were to the possible solutions of the three-factor model.

It was a feature of the available data that cohort values relating to the earliest and latest years of birth were based on fewer age-specific death rates than the central cohorts. Thus in figs 1 and 2 the cohort values shown as points on the extreme left and right were derived from one five-year age group, those adjacent from two, and so on. This means that the extreme points are statistically less reliable than the central ones. Since the extreme right ones were based on recent death rates in the young, however, they may also yield important, early clues to future trends for all ages. Such clues would probably be lost in any age standardisation.

Results

Figures 1 and 2 give the results in standard form. On the left section of each figure the age value (similar to the age-specific death rate) is plotted against age at death on a log-log scale. A straight line would indicate a power relation of the rate with age.[10 11] On the right section of each figure the cohort and period of death values are plotted against time with a horizontal dotted line indicating the average value. Cohort values are plotted against central year of birth and period values against central year of death.

Figure 1 shows the results for all cancers in men and women separately. In each sex cohort values rose to a maximum and then declined. The peaks, however, related to different generations—the maximum for men occurring in the cohort born around 1900, and for
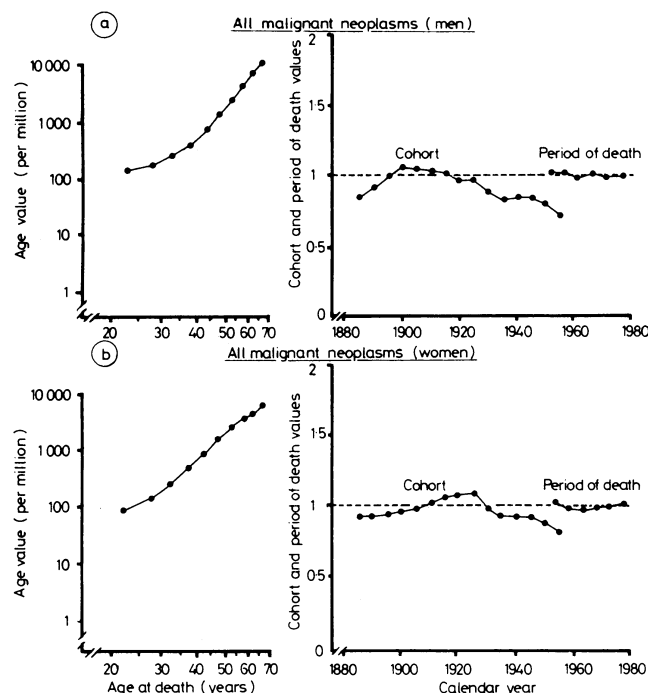

FIG 1—Age, cohort, and period of death values (a) for all malignant neoplasms in men and (b) for all malignant neoplasms in women. (Mortality in England and Wales during 1951-80 among persons aged 20-69 certified to ICD codes 140-239 (8th revision).)

women in the 1925 cohort. This suggests that one or more influences which affected successive generations rose and then declined. There was little variation in the period of death values, though a small gradual decline was apparent for men but not for women.

Figure 2 gives the results for five cancers, each of which showed a clear pattern in relation to either cohort or period of death values. Figure 2(a) describes the mortality from cancer of the bladder in men. There was little variation in period of death values, but cohort values rose and then fell. The largest cohort value (about 1·09) occurred in relation to men born around 1900, which implies that that generation experienced a 9% increase in death rates from bladder cancer over the average for all cohorts considered. A similar graph (not shown) was constructed for lung cancer in men, the same birth cohort showing the largest value of 1·09 in that case also (based on ages 25-69, periods 1951-80). Thus those two cancers, responsible for over 40% of cancer deaths in men during recent years, showed similar trends.

Figure 2(b), for testicular cancer, shows a contrasting picture with successive increases in mortality in generations born after 1920 following an earlier decrease. By comparison period of death values again showed little variation. A pronounced rise in men born after 1940 related to men who died before reaching 40. The graph understates the rise in incidence in so far as the treatment of this condition has recently improved. The final point in this cohort graph is based, as explained above, on only one age-specific rate (based on 58 deaths) and should therefore be treated with caution. The pattern of mortality from cancer of this site was unusual in that there was a decline in middle age.

In fig 2(c), for cancer of the oesophagus in men, there is a distinctly different pattern with fluctuations of similar size in cohort and period of death values. Mortality increased in successive generations up to the group of men born around 1925, since when it stabilised. In contrast, period of death values increased after the 1956-60 period.

The final sections of fig 2 show two of the main sites of cancer in women. For cancer of the lung (fig 2(d)) there were pronounced changes in mortality in successive generations with an increase up to a peak (1·69) for women born in the mid-1920s, followed by a downward trend. A rise and fall were also found in the corresponding curve for men, though the peak occurred some 25 years earlier. All period of death values were close to unity.

Figure 2(e), for cancer of the cervix, shows a more complex trend in successive generations than was shown for the other sites. If the cohort values are interpreted in terms of year of birth, there was a decline at the end of the last century which continued until the 1905 cohort. After this there was a rise until the 1920 cohort, followed by a further decline to the generation of women born in the mid-1930s. Subsequently there was a pronounced upward movement with successive birth groups. Again, as shown in each section of fig 2 except 2(c), the degree of variation in cohort values was substantially greater than that found in the period of death values, though the latter showed a gradual decrease. Cervical cancer is generally regarded as associated with the number of different sex partners, so that it is relevant to relate these cohort values to the times when the generations of women reached maturity. The peak shown then relates to women who were 20-24 during the second world war, and the nadir to the middle 1950s before the period of sexual liberation.

Discussion

There are several reasons for the often held view that cancer mortality is on the increase. For instance, the yearly number of deaths from cancer has been increasing over the past 20 years because the population is aging; cancer is more widely and openly discussed in relation to individual victims as well as in the media; improved diagnosis has probably led to recognition of more cases; and there are various other components.[1] Conclusions based on these statements, however, ignore the absolute risks of dying from cancer among people alive today. Figure 1 shows that the risk of dying from cancer of all sites is decreasing so far as recent successive generations are concerned.

The sites of cancer listed in fig 2 were chosen because they showed widely varying patterns. The male generations with highest rates from bladder cancer were probably those who suffered highest industrial exposure to aromatic amines—an important cause of this tumour[12]—and, judging by the similarities that this curve shared with that for male lung cancer, those who smoked most. Improved working conditions contributed to
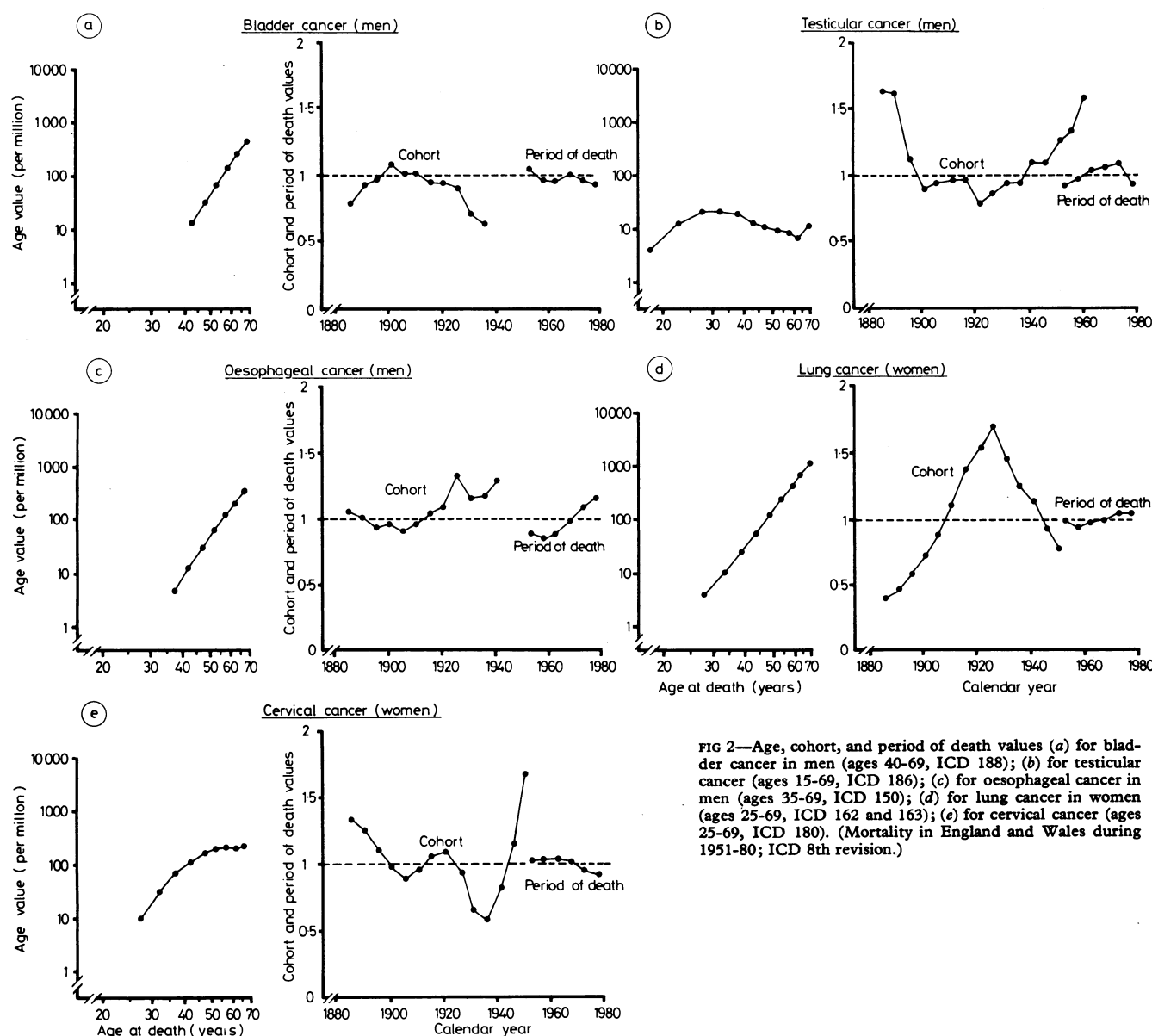
FIG 2—Age, cohort, and period of death values (a) for bladder cancer in men (ages 40-69, ICD 188); (b) for testicular cancer (ages 15-69, ICD 186); (c) for oesophageal cancer in men (ages 35-69, ICD 150); (d) for lung cancer in women (ages 25-69, ICD 162 and 163); (e) for cervical cancer (ages 25-69, ICD 180). (Mortality in England and Wales during 1951-80; ICD 8th revision.)

the decline in mortality in later cohorts, and it has been suggested that the occupationally induced epidemic may be over (J M Davies, paper presented to the Society for Social Medicine meeting, Stockton-on-Tees, 9 October 1981). Safer smoking habits may also have helped.

The rising period of death values in male oesophageal cancer corresponded to the increasing national level of consumption from about 1950 of beer, wine, and spirits.[13] For these two trends to be causally related would require that consumption had been increasing in most or all age groups and that the effect occurs over a short interval of time. This could be so if, for example, alcohol acts as a promoter.

Increases in the incidence of testicular cancer in recent generations of men have been recognised in various countries, and it is now the most commonly occurring tumour in men aged 25-34 in England and Wales. Davies pointed out that successive generations of boys and young men have been subjected to factors such as tight dress, work in the sitting position, and central heating which might have the effect of increasing temperature in the scrotum.[14]

For women the position of the peak cohort for lung cancer

corresponded to the later onset of smoking among women than men. In the absence of substantial decreases in the numbers of cigarettes smoked by successive generations of either sex,[15] the decline in mortality from lung cancer has been attributed to the reduction in the tar content of cigarettes[16] but is not unanimously accepted.[17] Another suggestion is that the reduction in air pollution after the Clean Air Act has played a part.[18] Despite the decline in cohort values the annual number of deaths from lung cancer in women has more than trebled since 1951 to reach over 8000 in 1980. This increase in numbers is likely to continue for some years as the worst affected cohorts age. Correspondingly, annual numbers of deaths from lung cancer in men have only recently stabilised, despite the 25-year difference in peak cohorts.

The remarkable cervical cancer cohort curve has been shown to be related closely to the incidence of gonorrhoea in related generations and, as mentioned above, probably reflects changes in the sexual behaviour of successive groups of women reaching maturity.[19] The decreasing period of death values might be related to an improved prognosis either by earlier detection or by more effective treatment or both.

As always when using mortality data, one must consider the quality of the diagnosis from which the death rates are obtained. Though it is known that there are errors, and that changes in accuracy take place over time, the effect of these was reduced in our analysis by limiting the data to mortality among persons under 70, dying since 1950. Mortality reflects the balance of incidence and case fatality, and trends should be interpreted with this in mind. The absence of any substantial period of death effects for three of the five cancers discussed should not therefore be taken as indicative necessarily of the lack of improvement in treatment over the years.

A complete description of trends for all sites of cancer for which suitable mortality rates are available is being prepared and will be published elsewhere.

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Short-term prophylaxis with cefotaxime for prostatic surgery

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Abstract

A randomised controlled trial of a new cephalosporin, cefotaxime, was carried out in men undergoing transurethral resection of the prostate. The purpose of the trial was to determine whether 48-hour prophylaxis with this new broad-spectrum, non-nephrotoxic cephalosporin would reduce postoperative bacteriuria and postoperative complications. The treated patients fared significantly better than the non-treated patients in having fewer febrile episodes, fewer episodes of tachycardia, a lower incidence of appreciable bacteriuria postoperatively, and fewer complications, and spending on average one day less in hospital. There was no difference in postoperative urea and creatinine concentrations between the groups, and no other side effects of cefotaxime occurred in this elderly population.

Prophylaxis with cefotaxime would appear to make prostatic surgery safer.

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Introduction

A 40-year-old man has been estimated to have a 10% chance of requiring prostatectomy by the age of 80.[1] Thus prostatectomy remains one of the most frequently performed operations. In recent years there has been a change in technique so that most prostatectomy operations are by transurethral resection. This has occurred because of the lower mortality and morbidity associated with transurethral surgery.[2] Nevertheless, complications still occur and any treatment or variation in technique that will make this operation safer is to be welcomed.

In a previous study a 48-hour regimen of antibiotic prophylaxis with cephradine resulted in a statistically significant reduction in postoperative bacteriuria and a possible but not statistically significant reduction in postoperative complications.[3] We undertook the present trial to determine whether by using the new parenteral cephalosporin cefotaxime, which has a greater