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Assessing the risks of Cancer

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Introduction

In recent years it has been suggested by Epstein and others (Epstein, 1978; ASTMS, 1980) that we are having a cancer "epidemic". This "epidemic", they suggest, is overwhelmingly caused by chemical and physical agents in the environment, some of which are self-inflicted, as in smoking, but the majority of which are external and beyond the individual's control.

Is this really so? The objective of this paper is to examine the truth of these claims by

(a) quantifying the loss of life due to cancer

(b) assessing whether or not cancer rates are increasing, and,

(c) looking at the relative importance of the different causes of cancer.

The magnitude of the problem

In England and Wales, about 20% of male and 20% of female deaths are due to cancer (table 1). Though the percentage of deaths due to cancer is higher in those dying before age 65 than in those dying after age 65, especially in women, the majority, almost two-thirds, of cancer deaths still occur in the over 65 (table 2) group.

It might be thought by the statistically uninitiated that, because life expectation is around 70 years and because 20% of all deaths are caused by cancer, removal of cancer as a cause of death would add about 14 years (= 20% of 70) to our life expectation. In fact this is not so at all. Because most of cancer deaths occur

in the old, and because death rates from other causes are also high then, life expectation for the population at large would only go up by slightly less than 3 years if the whole of cancer deaths were suddenly to disappear overnight. This compares with an increase of almost 30 years in life expectation since the beginning of this century due predominantly to the conquering of infectious diseases.

I am not, of course, trying to argue that great efforts should not be made to prevent or to cure cancer. After all, the average loss of life expectation of someone who dies of cancer is quite substantial, almost 15 years. Rather by using the figure of less than 3 years I am trying to illustrate the point that we should not overestimate the loss of life due to cancer in the population as a whole, nor become overly hopeful of the magnitude of the benefits to be gained from preventing it.

Is the problem increasing?

It is certainly easy to produce figures that suggest cancer incidence and mortality have been rising sharply - for instance cancers were responsible for only 7% of male deaths and 9% of female deaths at the time of the first World War, whereas they are responsible for 20% now. However, such comparisons are misleading for two major reasons. Firstly, the age structure of the population at risk has changed dramatically over this century, such that far more people nowadays survive to ages at which cancer is common. Secondly, diagnosis of cancer for some sites has improved considerably since the beginning of the century. This is particularly clear for lung cancer where, at the turn of the century, it was estimated by Sweany (Rigdon and Kirchoff, 1958) that the percentage of correct diagnosis was only 5%. Recently, the Royal College of Physicians (1977) presented estimates suggesting that though recorded male lung cancer death rates rose by a factor of 40 between 1916 and 1951, actual death rates probably only rose by a factor of 3 or 4. In other words improvements in diagnostic standards have resulted in a detection rate of 10 or more times better.

Of course even an increase in the true lung cancer rate by a factor of 3 or 4 is a large one, but there are clear indications that this increase, which seems to has been be mainly attributable to the mass adoption of the cigarette smoking habit around the attributed time of the first World War by men and some 30 years later by women, is now coming to an end. Indeed, as Table 3 shows, lung cancer rates are now falling in men at all ages under 70 and in women at ages under 45, probably due in part to the prevailing switch to filter cigarettes with reduced tar deliveries. The continuing rise in the older groups is probably due to the fact that in these age-groups, but not in the younger ones, proportions of people who have smoked for a relatively long part of their life are still on the increase. By considering a hypothetical population, all of whom took up smoking in 1915, or at age 15 if born after 1900, it can be seen that the rises are likely to die out soon for men (table 3). Indeed during very recent years overall male lung cancer rates in England and Wales appear to have been static and should fall soon. It has been estimated that female rates will flatten out eventually at about a third of those for males. (OPCS, 1978).

What are the recent trends in age-specific cancer rates generally? Table 5 (men) and table 6 (women) list percentage changes in overall cancer rates between 1966-70 and 1971-75 by site, presenting the data in terms of the relative frequency of the different types of cancer. Longer term trends, over the period 1951-55 to 1971-75 are summarised in table 7.

The tables illustrate that, when all sites except the bronchus are considered together, the short-term picture is of no overall change in the age-standardised death rate while looking longer-term there is a small decrease. Some sites show a clear decrease (stomach, large intestine, rectum and uterus) and some show a clear increase (bladder, pancreas, leukaemia, oesophagus, breast and ovary). When one considers that, there have been marked increases in the ability to diagnose pancreatic cancer and leukaemia accurately and when one also notes that bladder and oesophageal cancer have been linked to smoking, (Doll and Peto, 1976) and that the rise in breast

cancer is consistent with the hypothesis that it is related to low fertility, it is clear that the grounds for believing there to be an explosion of occupationally linked cancers from this evidence is weak. Of course the fact that some cancers may be related to smoking does not exclude them being also related to occupational factors. Therefore, in the next section we will look at recent scientific estimates of the role of different factors in cancer aetiology.

Before leaving this section, however, we will note, parenthetically, that improvements in cancer cure rates have only been marked for some rarer forms of cancer (such as Hodgkin's disease, choriocarcinoma or Wilm's tumour). Those arguing in favour of a growing cancer epidemic cannot therefore argue that we have a situation where improving cancer cure is being counterbalanced by increasing cancer incidence.

Role Played by Different Factors in Cancer

Last year the American Health Foundation organized a conference on the primary prevention of cancer, a specific aim of which was to assess the role played by different risk factors in cancer. Their conclusions, which accord quite well with earlier estimates given by Higginson (1979) are summarised in table 8.

In looking at this table one should bear in mind that it is unrealistic to assume that cancer is always caused by a single agent. Not only is there evidence that smoking and alcohol have a synergistic effect in relation to cancer of the upper aerodigestive tract (Rothman 1980) and that smoking similarly reacts synergistically with asbestos and other occupational hazards to cause lung cancer (Higginson 1980), but also it is clear that in a number of cases genetic, endocrine, nutritional and immunological factors substantially modify the response to chemical or physical carcinogens. For this reason, and also because the smoking estimate by Hammond and

Seidman (1980) on which the figure in Table 8 is based ignores recent changes in the type of cigarette smoked, the percentages attributed to smoking in the table are likely to be on the high side, a view which is reinforced by the observation by other workers in the UK (Adelstein 1966, Dean et al 1978) that general air pollution may play a rather larger role in lung cancer aetiology than indicated by the Hammond and Garfinkel (1980) estimates used in table 8.

The most striking differences between the conclusions of the AHF conference and those of Epstein (1978) lie in their estimates of the relative contribution of occupationally related factors and of nutritional factors to the total.

The major contribution to Epstein's estimate of occupationally related cancer came from a document that was widely circulated in the USA but was never published (Bridbord 1978). This document encouraged Califano, the former Health, Education and Welfare Secretary in Washington, to make the incredible assertion that as many as half of all workers exposed to asbestos, numbering 8 to 11 million, would develop serious diseases related to asbestos exposures. As has been noted previously (AIHC, 1978; Peto 1980) the method of calculation was open to two major flaws both of which had the effect of greatly increasing the estimates of the numbers of cancers attributable to occupational factors. The first was that risk ratios of exposed to non-exposed workers used were taken from studies of workers exposed in the 1920's to the 1950's whereas workplace conditions for known carcinogens and for many other chemicals also, have improved considerably since then. The second is that the document used exposed population figures based on the National Occupational Hazard Survey, a survey which did not measure levels of exposure and in fact included actual, potential or inferred exposures as well as part-time exposures.

The gross weakness of their estimation procedures is highlighted by the fact that detailed studies of excess deaths related to asbestos exposure at work (Advisory Committee on Asbestos 1979) shows that 13% at these are due to mesothelioma, thus implying that almost a million deaths from mesothelioma are likely to occur in the asbestos esposed US population according to the estimates used by Califano and Epstein. In fact, total annual deaths from mesothelioma are less than 1,000 in the US and less than 300 in the UK. It is clear that the estimates used by Califano and Epstein are at least an order of magnitude too high.

Nutritional factors are scarcely considered at all by Epstein but are well documented as having a major association with cancer incidence (eg Miller, 198C). There is not time here to consider these factors in detail but two associations are worthy of mention. The first is the striking correlation between fat consumption in different countries and their breast and colon cancer rates (figure 1). This is not explicable in terms of dietary fat being a vehicle for fat-soluble pesticides and industrial chemicals, as Epstein (1978) suggests, as it ignores the fact that countries with high chemical use do not stand out from the general cancer-fat relationship (Peto 1980)

The other nutrition-cancer relationship demanding particular attention is the dramatic relationship seen in animal experiments between total calorie intake and tumour rates. One example of this can be seen in the experiment of Conybeare (1979) where a 25%reduction in total dietary intake caused a dramatic reduction in incidence of a whole range of tumours in untreated control mice (table 9). This, and similar findings by Tucker (1979) have been reviewed by Roe (1979) who notes that it is certain that this effect could not be due to a 25% lower intake in one or more toxicants or carcinogens in the diet, for this would assume an incredibly steep dose response curve. Roe suggests that a possible explanation is that rats or mice seeing an empty food-hopper wonder

anthropomorphism.

where their next meal is coming from and suffer stress with a consequent rise in plasma corticosteroid levels, He postulates that a daily rise in plasma corticosteroid levels is perhaps protective against the development of many kinds of tumour.

Summary

Our look at the evidence does not all suggest the existence of any rampaging epidemic of cancer related to exposure to new physical and chemical agents in the environment. In fact, though one cannot be encouraged at the slow progress made in conquering cancer, there is no evidence of any overall rise in non-smoking-related cancer at all in recent years. The look at causes of cancer suggests that too much attention is being given to occupational carcinogens and not enough to cancers associated with nutrition.

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Table 1

Percentage of Deaths due to Cancer by Age

(England and Wales, 1974)

	Age							
	0-14	15-34	35-64	0-64	65-74	75+	65+	All Ages
Men	4	19	28	25	26	16	21	22.3% = 66,000 deaths.
Women	5	23	41	36	25	11	15	19.3% = 56,000 deaths.

Table 2

Percentage of all Cancer Deaths occurring at different ages

(England and Wales,1974)

	Age							
	0-14	15-34	35-64	0-64	65-74	75+	65+	All Ages
Men	0.6	1.4	34	36	39	25	64	100% = 66,000 deaths.
Women	0.5	1.3	35	37	30	34	63	100% = 56,000 deaths.

Table 3

Recent Changes in Lung Cancer Death Rates in England and Wales

Age Group	Percentage Change in Rate; 1966-70 to 1971-75	
	Men	Women
35-39	-22%	-16%
40-44	-17%	-17%
45-49	- 4%	+16%
50-54	- 7%	+17%
55-59	- 6%	+24%
60-64	- 5%	+30%
65-69	- 2%	+23%
70-74	+ 9%	+23%
75-79	+21%	+24%
80-84	+26%	+31%
85+	+26%	+26%
All Ages	+ 7%	+27%

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Table 4

Trends in lung cancer risk for a hypothetical population all of whom took up smoking in 1915, or at age 15 for those born after 1900.

	Duration of Smoking at Year						Risk increases up to year
Age	1930	1940	1950	1960	1970	1980	
40	15	25	25	25	25	25	1940
50	15	25	35	35	35	35	1950
60	15	25	35	45	45	45	1960
70	15	25	35	45	55	55	1970
80	15	25	35	45	55	65	1980
90	15	25	35	45	55	65	1990

Assumptions:

1) Risk increases with increased duration of smoking

2) No change in cigarettes

3) Non-smoking related factors ignored

Table 5

Recent trends in age-specific cancer death rates in men

(England and Wales)

ICD (8th rev)	Site	% of all neoplasms rate, 1966 - 70	Age-Standardized death rates per million 1966-70	1971-74	% Change
162,163	Bronchus	38.8	953	983	+ 3%
151	Stomach	11.9	293	270	- 8%
153	Large Intestine	6.5	159	163	+ 3%
185	Prostate	6.4	156	156	Nil
154	Rectum	5.o	122	119	- 2%
188	Bladder	4.2	103	105	+ 2%
157	Pancreas	4.2	103	106	+ 3%
204-207	Leukaemia	2.7	66	65	- 2%
150	Oesophagus	2.6	63	68	+ 8%
	All Others	17.8	436	444	+ 2%
140-239	Total	100.0	2454	2479	+1.0%
	Total (excluding bronchus)	61.2	1501	1496	-0.3%

Table 6

Recent trends in age-specific cancer death rates in women

(England and Wales)

ICD (8th rev)	Site	% of all neoplasms rate, 1966 - 70	Age-Standardized death rates per million 1966-70	1971-74	% Change
174	Breast	20.5	385	409	+ 6%
153	Large Intestine	10.5	197	196	- 1%
151	Stomach	10.0	187	163	- 13%
162,163	Bronchus	9.7	182	217	+ 19%
180-182	Uterus	7.8	147	137	- 7%
183	Ovary	5.3	131	132	+ 1%
154	Rectum	4.9	92	88	- 4%
157	Pancreas	4.2	79	83	+ 5%
204-207	Leukaemia	2.7	50	49	- 2%
150	Oesophagus	2.3	43	45	+ 5%
188	Bladder	1.9	36	37	+ 3%
	All Others	18.6	349	362	+ 4%
140-239	Total	100.0	1878	1918	+2.1%
	Total (excluding bronchus)	90.3	1696	1701	+0.3%

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Table 7

Percentage changes in age-standardized death rates 1951-55 to 1971-74

(England and Wales)

Site	Males	Females
Bronchus etc	+ 49%	+ 115%
Stomach	- 29%	- 41%
Large Intestine	- 16%	- 19%
Rectum	- 25%	- 17%
Bladder	+ 19%	+ 6%
Pancreas	+ 29%	+ 26%
Leukaemia	+ 23%	+ 14%
Oesophagus	+ 3%	+ 15%
Prostate	+ 4%	----
Breast	----	+ 13%
Uterus	----	- 20%
Ovary	----	+ 16%
All Others	+ 2%	+ 8%
Total	+ 12%	+ 2%
Total excluding bronchus	- 7%	- 5%

Table 8

Assessment of risk factors for cancer (conclusions from AHF conference, 1979)

Major Factors	Risk attributable
Nutrition	Up to 50% of common cancer
Smoking	25 to 35% of male cancers in USA; 5 to 10% of females.
Minor Factors	
Alcohol	3% of all USA cancers
Occupation	6% of male and 2% of female cancers in UK
Ionizing radiation	Less than 3% of cancers in USA (half background radiation and 40% or more diagnostic X-rays)
Ultraviolet radiation	Less than 2% of cancer deaths in USA.

Factors contributing little to overall risk	Other Factors
Food additives and contaminants	Genetic factors - may affect susceptibility in 10 to 25% of cases.
Drugs	
General air pollution	Viruses - no common cancer shown to be virus associated.
Water pollution	
Immunological factors.	

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Figure 1

Breast Cancer Mortality and Dietary Fat Consumption in 39 Countries

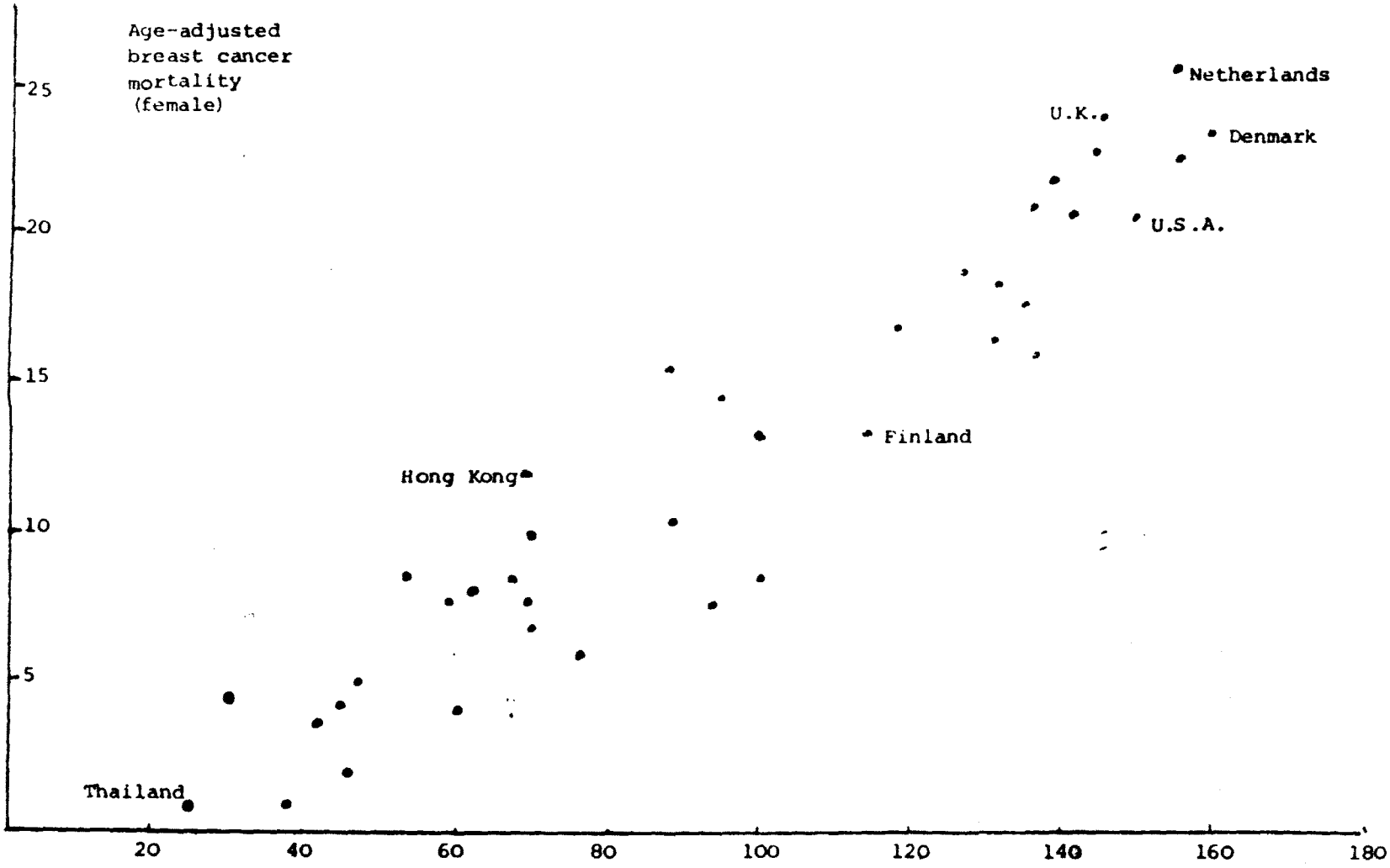

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Table 9

Effect of simple dietary restriction on tumour incidence in mice

Sex	Type of Tumour	% incidence		% reduction in incidence
		Ad Lib	Restricted to 75% of ad lib	
Male	Lung	30	19	- 37%
	Liver	47	12	- 74%
	Lymphoma	4	1	- 75%
	Other	8	4	- 50%
	Any tumour	71	36	- 49%
	Any malignant tumour	17	7	- 59%
Female	Lung	24	8	- 67%
	Liver	7	1	- 86%
	Lymphoma	11	4	- 64%
	Other	12	4	- 67%
	Any tumour	50	17	- 66%
	Any malignant tumour	23	7	- 70%

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