LETTERS TO THE EDITOR

Life expectancy and relation to smoking

EDITOR,—I was interested to see the short report by Dr Eva Prescott and colleagues, presenting life table analysis of the pooled data of three smoking cohorts.1 I agree wholeheartedly with one conclusion: that life expectancy, presented as survival graphs, shows the ultimate consequences of smoking in a way that is more easily comprehended than the regular relative risk method, which is why I suggested that the British doctor study be presented in that manner.2 It is perhaps not strictly true to suggest that the present Danish pooling of three cohort studies is the first to compare life shortening of men and women in this way.

Data of the British doctors showed eight and five years of life shortening for men and women respectively, compared with heavy smokers (25+ cigarettes/day) with heavier smokers (fig 1). The 40 year follow up showed the gap to have increased over time.3 While the Denmark study may have included a larger proportion of women heavy smokers, their definition of heavy equates with moderate and heavy in the British study. This would suggest that the Danish women experienced considerably more life shortening (nine years) than British women (five years).

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Reply

EDITOR,—I thank Dr Robert West for his interest in our paper1 and apologise for having missed the paper by Dr West on survival in the British doctor study3 although I did refer to survival among male British doctors as illustrated in the paper by Doll and Peto.1 However, although Dr West has taken daily tobacco consumption into account, I still think he may overestimate survival in women smokers. As shown in table VI in another paper by Doll and Peto,4 women smokers inhale much less than their male counterparts, and their mean age at starting to smoke differs considerably from that of the men. Indeed, in the paper it is stated that “Evidently, therefore, over the period of our study men and women who had reached ages at which death was likely to occur had had smoking histories that differed in ways that would not be allowed for adequately by standardisation according to the amount stated to have been smoked daily.” In our study population we also had many women smokers, who stated that they did not inhale the smoke. We have found inhalation to be a very strong predictor of morbidity and mortality, and, consequently, we excluded non-inhalers from our survival tables. This may explain the difference in years of life shortening between our study and that of Dr West.

One point we wished to make in our study is that young women today start smoking at a much younger age, they smoke more and almost all of them inhale the smoke: therefore these studies based on older birth cohorts of women with different smoking habits may still underestimate the impact smoking has on the health of women today.

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Relation between high temperature and mortality

EDITOR,—Rooney et al showed that exceptionally high temperatures in England and Wales, though rare, do cause increases in daily mortality. They also draw attention to “harvesting”, the forward displacement of mortality as possibly present in the series of excess deaths. However, “harvesting” is clearly evident in figure 1 in their paper.1 In figure 1 the excess deaths attributed to the exceptional temperatures are visually evident in the area lying below the moving average for deaths between late June (when the moving average for temperature in 1995 first exceeded the equivalent moving average for 1993–1994) and above the equivalent moving average for deaths in 1993–1994 until mid-August. From then onwards this excess is succeeded by a deficit whose area for the weeks to the end of September is at least as large as that of the initial excess. This amount of “harvesting” from the weeks immediately after the excess deaths surely dramatically reduces the significance of these excess deaths attributed to excess heat.

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BOOK REVIEWS


This book edited by Diana Kuh and Yoav Ben-Shlomo with contributions from a variety of authors from the United Kingdom, Canada and the USA, is a review of the scientific literature on the association of a wide range of social determinants during pregnancy and early childhood and the development afterwards of chronic diseases or risk factors in the adult life. The book discusses and gives evidence in five different sections about the hypothesis supported by the authors: There are social and biological interactions acting during pregnancy that influence adult life in the occurrence of chronic diseases. In some ways this hypothesis challenges important mainstream assumptions of the so called “risk factor” epidemiology.

The few pieces of research published about this topic, though rare, are used as the information base for this book. It is mainly based on long cohorts from registers made early in this century for other purposes.

The book describes the well documented and most important associations found between coronary heart disease, cardiovascular diseases, cancer, diabetes and respiratory and allergic diseases as well as risk factors like blood pressure and hypertension with exposures during pregnancy and early life.
The best contributions are two in my opinion: one is the possibility of opening a new field of interest in epidemiology so focused recently in reiterating information on the “holy four” (smoking, eating fat, drinking alcohol and lack of physical exercise). The other emphasises the importance of social processes during the whole life course.

Although it is not implicit, the main message of the book could be interpreted in a simple way as a call to shift the focus from the behaviours in adult life to behaviours of women during pregnancy and also during early childhood.

Care should be taken that this provocative work does not introduce a new danger: adding “mother blaming” to the burden of suffering of the people in the same way as associations of behaviours with diseases produced “victim blaming” forces 20 years ago.

However, this book is a “must” for epidemiologist involved in chronic diseases aetiology. The authors are to be congratulated on this thought provoking book.

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There is increasing interest within epidemiology regarding the potential early life origins of disease in adulthood. This 37th symposium volume of the Society for the Study of Human Biology concentrates on this issue, with contributions from a wide range of disciplines. David Barker, whose group in Southampton has carried out the most influential epidemiological work in this area, contributes a chapter documenting the inverse associations between birth weight and cardiovascular disease risk in later life. He concludes that “programming” of the fetus through intrauterine supply of nutrients and oxygen changes several aspects of structural and functional capacity of the body, which reveal themselves in disease in later life. In a chapter on the long term consequences of early environmental influences for human growth Stanley Ulijaszek distinguishes between two forms of programming: “distorted morphology” caused by the induction, deletion or impaired development of a permanent somatic structure caused by an environmental factor operating in a critical time; and “metabolic imprinting” in which physiological functions are set in train in response to early life environmental factors. Ulijaszek concludes that the available evidence suggests that distorted morphology is a better characterised, and less speculative, process than metabolic imprinting.

Several detailed examples of other long term health consequences of early exposures are considered in considerable detail in other chapters. For example the possible origins of amyotrophic lateral sclerosis (ALS) and Parkinsonism-dementia (PD) in Guam may reflect secondary hyperparathyroidism provoked by calcium deficiency leading to enhanced absorption of aluminium and the deposition of calcium and aluminium in neurofibrillary tangles. Early life influences on age at menarche, human taste and smell preferences, the development of sexuality and obesity is also discussed, among other examples. The relative contribution of early life and later life influences are given relatively little attention, although Michael Golden presents evidence that catch up growth can occur in people stunted at an early age through poor nutrition and Ralph Guaruto demonstrates how ALS and PD can be induced through long term exposures both in early life and in later life. The future development of the fascinating research discussed in this volume will depend upon a full consideration of how early life influences accumulate and interact with exposures acting later in the life course.

The only disappointing aspect of the book is the rather limited focus on methodological issues. The difficulty of studying exposures acting in early life in relation to outcomes that may occur many decades later is not discussed. This gives no way into understanding why different research groups have come up with different findings in relation to some of the issues discussed in this book (for example, the importance of catch up growth) and does not provide a stimulus for designing the kinds of studies that can answer the complex questions in an entirely adequate way.

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