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Commentary: William Ogilvy Kermack and the childhood origins of adult health and disease

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The 1934 Lancet paper by Kermack, McKendrick and McKinlay was a landmark in the discussion of birth cohort influences on adult disease risk.¹ It pre-dated Wade Hampton Frost's paper on cohort influences on tuberculosis mortality,² and, whilst it was pre-dated by several demographic analyses highlighting birth cohort influences,^{3,4} was prescient in using these analyses to inform hypotheses regarding early-life exposures and their influence on later disease. As Kermack, McKendrick and McKinlay concluded, the data behaved as if 'the expectation of life was determined by the conditions which existed during the child's early years', and concluded, 'the health of the child is determined by the environmental conditions existing during the years 0–15, and ... the health of the man is determined preponderantly by the physical constitution which the child has built up'.¹

The idea that adult disease is determined in early-life is currently most influentially reflected in the 'fetal origins' work of Professor David Barker and his colleagues. In this formulation

the critical period of early-life development which is influenced by environmental exposures and has long-term effects on later health is the intra-uterine period.⁵ Interestingly Kermack *et al.* noted that one exception to the general pattern they observed—of mortality rates initially declining in the young, and only declining at older ages when the cohorts experiencing the initial mortality declines had entered these older age groups—was mortality under one year of age, which started to fall only after mortality at later ages had done so. They suggested that infant mortality was dependent upon the health of the mother, and thus improvement in infant mortality followed the generational improvement in the vitality of women of childbearing age. Kermack *et al.* suggested that the fall in the death rate at ages 1–4 was also somewhat delayed, but to a lesser extent than infant mortality. They did not provide data to support this assertion, however, and as Harris shows, at least in the case of Scotland, the fall in 1–4 year-old mortality fitted in well with the cohort-specific falls, occurring 10 years before the fall in 5–14 year-old mortality.⁶ The data suggest that very early-life development—before or within the first year of life—could not determine the mortality risk of birth cohorts as they aged. Other recent research—taking a lead from the seminal work of Anders Forsdahl^{7,8}—has investigated the possibility that

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exposures acting right across the growth and developmental phase—from pre-natal growth through to the end of adolescence—may importantly influence later health.^{9,10} Some epidemiologists maintain that there is nothing special about the early-life period in terms of embodied (i.e. irreversible) outcomes of early-life exposure, with early-life conditions only influencing adult health through the social trajectories into which they place people.¹¹ Clearly the issues raised by Kermack *et al.* nearly 70 years ago still resonate within current epidemiology.

Who was William Ogilvy Kermack?

WO Kermack was born on 26 April 1898 in Kirriemuir Angus, Scotland, the only child of William Kermack, a postman, and Helen Ogilvy.¹² His pre-university education was in rural Scottish schools, during which he studied advanced geometry and mathematics. He graduated in Mathematics and Natural Philosophy from Aberdeen University in 1918, and at the end of his university period was involved in the statistical analysis of milk yields of dairy cattle, and was introduced to the work of Karl Pearson, 'Student', and the journal *Biometrika*. After a brief period of service in the Royal Air Force he worked in Oxford in industrial chemistry (during which time he had his first scientific publication) and then went on to the Chemical Section



Figure 1 William Ogilvy Kermack. Photograph courtesy of Godfrey Argent Studios

of the Royal College of Physicians Laboratory, Edinburgh, where he was for 28 years. He worked largely on chemical questions (chemistry being a major component of his university course) until on the 2nd June 1924 he was blinded while working alone in a laboratory, an explosion causing caustic alkali to enter his eyes. With help from colleagues at the Royal College of Physicians Laboratory he continued his scientific career, working in particular on colloidal chemistry and the search for new anti-malarial drugs. He also started a fruitful collaboration with Lieutenant-Colonel AG McKendrick, the superintendent of the Royal College of Physicians Laboratory from 1920 to 1941, on mathematical approaches to human disease. This resulted in what is certainly the most cited and best known of Kermack's papers in the field of epidemiology, a seminal work on the mathematical theory of epidemics.¹³ A series of further papers on this topic from these authors appeared over the subsequent decade. Their work started with the formal treatment of the course of epidemics in closed populations, investigating the influence of population density, infectivity of affected individuals, and probability of death or recovery after infection. From initial considerations of closed populations further publications investigated the effect of the continued introduction of susceptible individuals into a population, of death not due to the epidemic disease under investigation, and tests of these models against the experimental animal studies of Major Greenwood.¹⁴ With McKendrick, Kermack also published on statistical tests of the randomness of number series and the design and analysis of experiments, together with the derivation of simple formulae for calculating 95% confidence intervals. In the field of mathematical theory Kermack worked with Sir Edmund Whittaker and WH McCrea on the solution of differential equations, geometry and the theory of the 'expanding universe'. Writing after Kermack's death, McCrea remarked that Kermack had an 'altogether exceptional sense of algebraic form, in addition to [a] penetrating sense of mathematical significance', with the blind Kermack 'doing all the working in his head'.¹²

Most of the work for which Kermack is still remembered was carried out during his period at the laboratory of the Royal College of Physicians of Edinburgh. In 1949, having been elected a Fellow of the Royal Society in 1944, Kermack became the foundation MacLoud-Smith Professor of Biological Chemistry at the University of Aberdeen (the chair was not to become known by the more familiar term of 'Biochemistry' until 1969). Here he concentrated on teaching and university administration. Although better known for his mathematical, chemical and epidemiological interests, Kermack had co-authored a popular book on biochemistry in 1938, 'The stuff we're made of', with Phillip Eagleton; a second edition of this was published in 1948. He published the occasional biochemical paper (for example on the non-protein constituents of lobster and locust tissues), and continued work on anti-malarials.

Kermack's scientific work was mainly carried out while he was totally blind. He kept up with the scientific literature through colleagues and research students reading papers from scientific journals to him, which they would subsequently discuss. He apparently had a remarkable memory for the content of documents that were read to him. He was keenly interested in debate on religion and politics, being a humanist and rationalist in the former and a leftist in the latter, although a trip to Moscow in

1961 to attend the Fifth International Congress of Biochemistry modified his views somewhat.¹² After his retirement in 1968 he continued active involvement in the University of Aberdeen and undertook a course in computer science. He died (while working at his desk) 2 years later, in 1970.

Kermack on the determinants of population health

Kermack's work on the determinants of mortality levels was, clearly, only a minor part of his scientific research. The context in which it was carried out was one of considerable concern with fertility levels of populations, the health of children as an indicator of the biological potential of a country, and the search for 'laws of mortality'.^{15,16} In the latter regard, in 1927 two papers appeared from actuaries suggesting that mortality rates at any particular age depended upon year of birth.^{3,4} Figures 2 and 3, from the work of VPA Derrick, show how mortality rates plotted against year of birth—rather than the more familiar year of death—were strikingly parallel. Death rates for younger age groups had been falling from the middle of the nineteenth-

century, whereas the death rate for those in early middle-age did not start to decline until the end of the century, and for the late middle-aged and the elderly no fall was seen until after the turn of the century. The paper by Kermack, McKendrick and McKinlay that we reprint in this issue of the IJE,¹ repeated these earlier analyses and extended them to Scotland and Sweden, where similar year-of-birth dependent falls in mortality were seen, but from different starting points. In the case of Sweden, the decline started for cohorts born around the beginning of the 19th century, but an interruption was seen mid-century where period specific relative increases in mortality occurred. Working around the same time as Kermack *et al.* the Swedish demographers, Cramer and Wold¹⁷ also noted that the generational declines in mortality were seriously distorted by period effects, a finding which has been more recently confirmed.¹⁸

In a companion paper published in the *Journal of Hygiene* Kermack *et al.* gave a more technical and mathematical presentation of these data.¹⁹ In the latter paper mortality was decomposed into two factors, one of which, β , depended only on age and the other, α , only on the year of birth. In this way β values for a country together with α values according to the year of birth could be plotted. In the paper we reprint, Kermack *et al.* summarized their data in tables in which relative mortality was tabulated according to period of death and age of death (see Tables 2 to 4 in the reprinted paper). These relative mortalities followed diagonal trajectories, as would be expected given a determining role of birth cohort. In a later paper Kermack, McKendrick and Barclay performed similar analyses to investigate whether mortality within both town and country districts of Scotland also followed what they called 'the diagonal law'.²⁰ They showed both with simple tabulations by age at death and period of death, and by computations of α and β values, that the β curves were similar for town and country, but that the α values were considerably lower in the country areas across the nineteenth-century. The town value started to decline from around 1831 and declined rapidly; the decline started later in the country regions and was less rapid, converging with the town values by year of birth 1921. The β curve was interpreted as reflecting the physiological constitution of the population as a function of age, whilst the α curve was taken to be a measure of the influence of the environment; more specifically the ' α value is a measure of the general level of environment during childhood, the period during which the general constitution of the individual is being built up'.²⁰ Since there was considerable mixing of the town and country population—indeed the town population had within recent generations largely been derived from the surrounding country—the genetic constitution of the two groups could not greatly differ, the authors thought. Thus the α value 'which may be called the "generation mortality coefficient", is a measure of the social conditions which existed during the childhood of the generation'.²⁰ The convergence of the α curves between the town and country led these authors to conclude that the real healthiness of the town environment had, by 1921, become approximately equal to that of the countryside, 'a verdict rather more favourable to the town at the present day than the mere inspection of the crude figures would suggest'. The fact that the town still had higher overall mortality rates than country districts reflected environmental conditions many decades before, not the current environmental conditions.

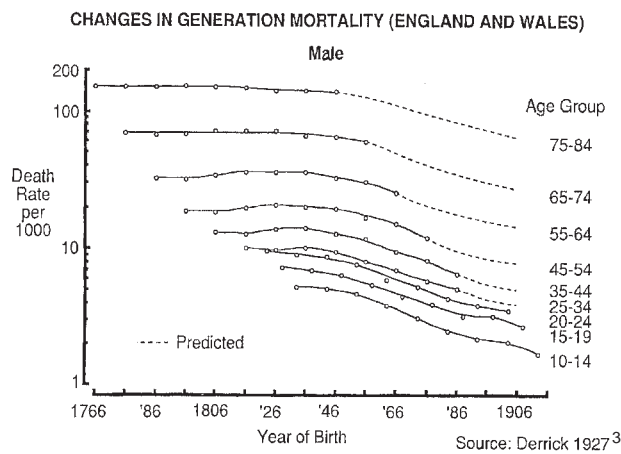


Figure 2 Generational mortality for males. Dotted lines indicate projections

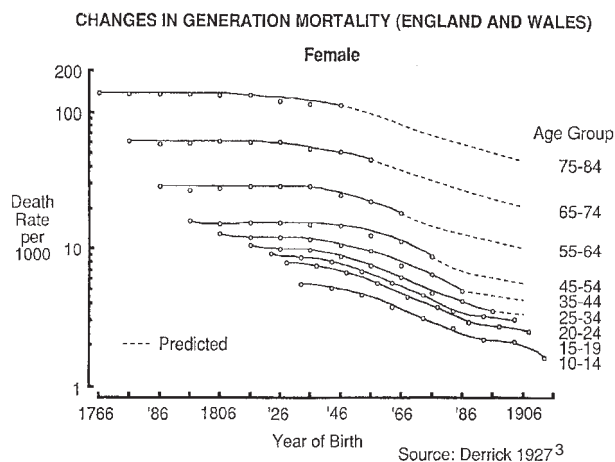


Figure 3 Generational mortality for females. Dotted lines indicate projections

In the paper we reprint, Kermack *et al.* state that an important way of determining what is the real explanation of the phenomenon they observe is ‘the detailed examination of the causes of death at different periods of different age-groups in any one country. Analysis of the Scottish records from the latter point of view is proceeding’.¹ Unfortunately no systematic analysis of generation effects by cause of death appeared. When Major Greenwood reviewed English mortality trends, in a valedictory address for Karl Pearson,²¹ he was more cautious than Kermack *et al.* with regard to the overwhelming importance of the childhood years: ‘I do not think persons over the age of 40 need abandon hope that social and hygienic betterments introduced after their school days may increase their expectations of life’. He thought that their approach may, however, be of importance for certain causes of death, in particular tuberculosis. The only cause-specific analyses performed by Kermack and colleagues related to short-term trends in cancer mortality.²² For some sites, (e.g. buccal cancer) declines were detected in earlier age bands and it was ‘perhaps, not too optimistic to hope that these generations will carry their lowered cancer death-rate, along with them, as they have done their general death-rate, and that subsequent generations will progress to a still lower level of mortality’.²² The trend for many cancers went against this pattern, however, in particular respiratory tract cancer (in retrospect due to the rise in cigarette smoking), and Kermack and colleagues thought that the rise in lung cancer might at least partly indicate improved diagnosis, but breast cancer was also increasing, and improved diagnosis was unlikely in this case. No comprehensive analysis of the importance of the generation effect for different causes of morbidity and mortality was presented by Kermack *et al.* however, and the topic was not returned to after the 1940 paper.

Kermack and his collaborators did, however, apply their ‘diagonal law’ to one specific issue: fertility. Analysing data from Sweden, Denmark, Finland, Norway and France, Barclay and Kermack suggested that these conformed to the ‘diagonal law’,²³ and later showed that predictions based on the law appeared to apply to Scotland.²⁴ The fertility and mortality data showed a striking difference, however: in the former case, declines were first seen at older ages and then at younger ages. Barclay and Kermack remarked that ‘In the case of the death-rates the wave of reduced mortality swept in the opposite direction, from the young to the old, and it was pointed out that here the existence of the diagonal law meant that each generation carried its own characteristic reduction in mortality along with it. In the case of the birth-rates there does not seem to be any corresponding simple interpretation. At present we can only regard the fact

that the rate at which the wave progresses is one year of age per year of time as a curious accident’.²³ While the application of the ‘diagonal law’ could only be seen as curious in the case of reproductive outcomes, there was certainly a link between considerations of the determinants of mortality and determinants of fertility. Working in Aberdeen at the same time as Kermack, the pioneering obstetrician and epidemiologist Dugald Baird was greatly interested in the proposition that early-life development strongly influenced the reproductive success of women,^{25,26} and he referenced the paper we reprint here as indicating the importance of childhood circumstances in general to health outcomes.²⁷

Birth cohort influences on adult mortality: what happened after 1934?

Kermack *et al.* used their data to predict death rates in Scotland as a whole and in the town and country regions in Scotland up to 1951,^{1,20} and Derrick was even more ambitious, predicting mortality rates up until the 1980s (see Figures 2 and 3). Generational influences on mortality were also given official recognition. A 1942 governmental report on population trends forecast populations in Great Britain up until 1971, utilizing the birth cohort approach²⁸ and the epidemiologist and vital statistician, Percy Stocks, remarked in 1941 that ‘Derrick’s happy observation in 1927 that each generation tends to carry with it throughout its life a characteristic mortality, confirmed as it has been by other statisticians, has opened up interesting fields of study. The time seems to be coming, indeed, when the Registrar-General’s annual survey will not be complete without some record of the progress of particular vintages of children’.²⁹ This dominance of the generational approach in official population forecasting was short lived, however. When the Royal Commission on Population report was published in 1949, it considered but rejected such an approach (Box 1). Inspecting the agreement between Derrick’s predictions illustrated in Figures 2 and 3, and the observed pattern in Figures 4 and 5, and our updating of the table for England and Wales from the Kermack paper, given in Table 1 makes clear why this was the case: there are dramatic differences between predicted and observed mortality. At younger ages, mortality rates fell faster than predicted on the basis of birth cohort regularities, whereas at older ages, mortality declined at a much slower rate than predicted. Considering the change in the cause of death profile between the middle of the 19th and latter part of the 20th century, this failure to maintain earlier regularities is not unexpected. In the middle of the 19th century, respiratory tuberculosis accounted for over a third of deaths in the 15–44 year age group and 15% of 45–64 year-old

Box 1

Reference was made to a tendency for the death rates at the older age-groups to begin to fall now that these groups are being recruited from generations of men and women who at earlier periods of life experienced lighter mortality than their predecessors and who may therefore represent progressively healthier and longer-lived stocks There appeared to be some evidence of a progressive weakening in the generation effect with advancing age, and several experiments were made in modifying the rather extreme results of the strict generation approach at the older ages. Tests showed that the results obtained depended in considerable degree upon the extent of the data used, in particular according to whether ages 0–4, generations born before 1836 and the experience of the calendar years 1941–45 were included or excluded.⁵¹

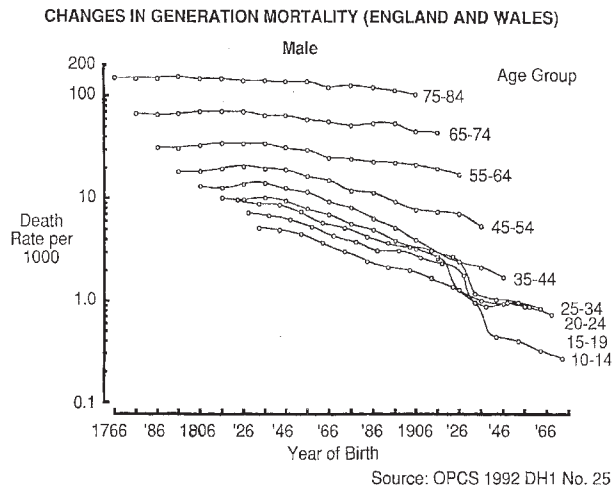


Figure 4 Changes in generation mortality (England and Wales): male

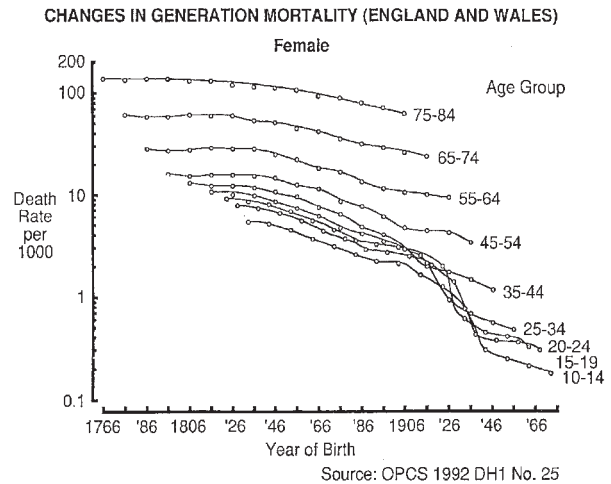


Figure 5 Changes in generation mortality (England and Wales): female

Table 1

Age at death	Period of death														
	1845	1855	1865	1875	1885	1895	1905	1915	1925	1935	1945	1955	1965	1975	1985
5-14	100	94	87	72	59	48	39	41	28	23	14	6	5	4	3
15-24	100	93	87	75	60	50	41	45	35	31	23	9	8	8	6
25-34	100	95	95	87	74	62	50	53	36	29	23	11	9	7	7
35-44	100	95	99	99	90	81	64	57	43	36	26	18	16	14	11
45-54	100	97	102	105	101	99	84	74	59	55	43	36	34	33	25
55-64	100	97	102	106	105	105	94	84	71	68	59	54	52	48	42
65-74	100	97	99	102	102	102	92	82	82	77	67	64	61	57	50-50

Source: OPCS 1992 DH1 No. 25

deaths.^{30,31} Respiratory tuberculosis is a disease in which birth cohort influences are well established, by John Brownlee in 1916,³² by the British Registrar General in his 1921 report²¹ (Box 2), through Frost's better known and more frequently referenced 1939 paper² to Springett's analyses of the British situation in 1952.³³ TB in late adulthood is believed often to reflect the recrudescence of infection acquired in earlier life, and improvement in earlier life circumstances should therefore be reflected (as they are) in reductions in TB at a later age. Other important contributors to adult mortality, which declined between the mid-19th century and the 1990s, include stroke (around 10% of deaths of those aged over 45 in the mid-19th century), stomach cancer and bronchitis, for all of which there is evidence of important early-life influences, or birth cohort

patterns in their decline, or both.³⁴⁻³⁷ In the second half of the 20th century, adult mortality was dominated by coronary heart disease—which initially shows period-specific rather than cohort-specific increases³⁸ and lung cancer, which showed birth cohort-specific increases,³⁵ reflecting the uptake of cigarette smoking by successive groups of young adults.³⁹ In addition to this the treatment of infectious disease with antibiotics, increasingly effective from the end of the 1930s, influenced mortality in a period rather than cohort-specific way. In the paper we reprint, Kermack *et al.* pointed out that as 'in the case of most empirical statistical results, it is assumed that no abnormal agency of great magnitude suddenly appears'. Factors such as the rapid uptake of cigarette smoking and the improvement in medical therapeutics clearly represent 'abnormal agencies of great magnitude'. Taken together the change in cause-of-death profile (and thus of particular aetiological agents) and development of medical therapeutics led to change in the relative importance of birth cohort and period effects on all-cause mortality. This change coincided with a shift in focus of public health and epidemiological thinking, from the importance of childhood environment (in particular childhood nutrition) to the importance of adult lifestyles, as major determinants of population health.^{15,16,40}

The meaning of birth cohort effects

The interpretations of birth cohort influences on adult mortality rates—or the absence of such influences—are clearly influenced by the dominant current explanatory modes. Derrick, for example,

Box 2

'This change [in mortality from respiratory TB] continues a tendency to which attention was drawn in the 'Supplement' for 1901-10, towards postponement to a higher age of the period of greatest decline of adult mortality. (Supplement to the 75th Annual Report of the Registrar-General, Part III, p.lxxi.). For males this has moved from 20-25 in 1881-90 to 45-55 in 1911-20, and for females from 20-25 in 1881-90 to 55-65 in 1911-20. Thus throughout these four decades a single generation of both males and females—those born somewhere about 1860—appears to have contributed more than any other to the reduction of mortality which has occurred.'²¹

considered that the patterns he observed showed ‘evidence that, in the comparatively stable conditions of this country during the past century, inherited characteristics have played a dominant part in the determination of our life history’. The influence of eugenics and the hereditarian thought of social biologists such as Karl Pearson is reflected here. Pearson thought that ‘a heavy death-rate does mean the elimination of the weaklings’.⁴¹ He supported this assertion by demonstrating that between 1838 and 1900 the infant mortality rates correlated negatively with mortality rates for those aged 1 to 5 years. He concluded that the improvement in mortality rates for those aged 1 to 5 years had been dependent on the rising infant mortality rate, because of its assumed selective winnowing effect.⁴¹ The actuaries Davidson and Reid, who reported their findings in 1927, spent much of their discussion down-playing this interpretation of mortality trends (Box 3).

The difference of opinion on this issue was the stimulus for an intense debate. Writing some years after the event, Kyd remembered that ‘We had on our Committee Mr Derrick, the great exponent of [the generational] theory, and we had on the other side Professor Kuczynski, who was the exponent of the year of observation theory, and the arguments were so furious at some of these meetings that I sometimes had considerable doubt whether the rate of mortality among the Committee might not be unduly high!’⁴² Kermack and colleagues countered the eugenic argument, both in the paper we reprint and elsewhere,^{1,20} on the grounds that inherited constitution would be reflected in infant mortality (which, as discussed above, did not demonstrate the regularities seen for mortality in other age groups), and that population movement between town and country did not allow for constitutional differences to determine different mortality rates and trends in mortality rates within these areas. They allowed for the possibility of the survival of

less fit individuals, due to improved environmental conditions in early life, if this was combined with a less harsh environment in adult life. However, their main interpretation—reflecting considerable interest in the 1930s in the long-term impact of poor nutrition in childhood (which included the work of PL McKinlay, one of the co-authors of the paper reprinted here)^{43,44}—was in terms of long-term consequences of childhood environment.

The interpretation of determinants of mortality trends—both general and for specific causes—will reflect the current dominant modes of explanation, and disputes between different modes of explanation. At the time Kermack *et al.* were writing, the difference of opinion over the consequences of declining infant and child mortality (and possible survival of those who would otherwise have died) was at the top of the agenda. Later in the 20th century, the main considerations related to the causes of chronic disease in adulthood and what was seen as the transformation of the epidemiological landscape from infection to non-infectious environmental and lifestyle exposures.^{16,40,45} In 1962 Susser and Stein examined the birth cohort and period influences on peptic ulcer mortality, which at the time was a key health issue.⁴⁶ They demonstrated clear year-of-birth influences on peptic ulcer mortality, and considered that the disease could be an outcome of an early phase of urbanization. However, the cohort phenomenon might ‘reflect upheavals which have had an uneven impact on past generations. The timing of the first world war, and the unemployment of the 1930s, roughly fit the fluctuations, and the cohorts with the highest peptic-ulcer death-rates were also the chief victims of the first world war. The immediate effects of war are evident in the rise in perforations and deaths from peptic ulcer which followed air-raids and the stress of war. Perhaps in a chronic condition such as this acute events might precipitate prolonged effects’. As Richard Asher said in 1951, it was fashionable to ‘put forward

Box 3

‘It is a common superstition—it does not seem to deserve a better name—that any step which is taken through medical science or otherwise to save life in the earlier stages is not a real saving, but is merely a wasteful deferring of mortality which reduces the vitality of the nation as a whole and keeps in existence a proportion of weaklings who are of no service to the State The foregoing discussion indicates that any steps which we take to improve the conditions of life give to the casual observer the idea that we have improved the mortality in early youth only at the expense of the mortality in early manhood; but the values of the two constants make it clear that we have not interfered with the mortality of early manhood, but have saved a certain amount from youth to a later point in life, and that ultimately, if we are enabled to carry out our work of improvement, we save mortality so much that at all stages we can show a decrease on the original rate of mortality Those persons who believe, of course, that any softening of conditions which enables a weak life to survive is a bad thing because they abhor physical weakness are presumably concentrating their attention on the desire to produce a race of physically strong persons who can withstand any amount of physical hardship. The method of achieving this end which follows logically from the idea that the amelioration of conditions is undesirable is not mere inaction and *laissez faire*, but a vigorous spreading of disease and bad conditions which might have the result of reducing the strong to weaklings as well as weeding out the latter. It is true that if we base our reasoning on the habits of life and on the circumstances of the primitive animals we find that the race adapts itself to its surrounding by a wasteful and unreasoning process, which is what is probably in the minds of those who object to the savings of early life; but in spite of this there does not seem to be any reason why man should not rise above this process and make use of the obviously superior alternative of adapting the surroundings to his own requirements. It seems very reasonable to remove the causes which create weaklings rather than to allow the weaklings to die of brutal neglect. In this connection it must be borne in mind that it is longevity and health which we seek to promote and not mere muscle strength; many of the men whose names stand highest in our national records would have cut no great figure in tests of endurance. As an excuse for what is perhaps a digression, we may point to the fact that the values of α and β which we have obtained seem to illustrate the manner in which social enterprise affects mortality in a very clear and striking manner.’⁴

mental causes for those illnesses where physical causes have not yet been found—for instance, peptic ulcer ...'.⁴⁷ A later series of elegant analyses by Amnon Sonnenberg and colleagues demonstrated that birth cohort dependency of peptic ulcer could be seen in a large number of countries⁴⁸ and that causative factors became effective before age 15 years.⁴⁹ They suggested that changes in lifetime occupational work load occurred in a cohort-specific fashion and could be responsible for the time-trends in peptic ulcer.⁵⁰ Now, however, the probable interpretation of the cohort effects is *H Pylori* infection, occurring during childhood and influencing peptic ulcer many years later.⁵² The mode of thinking in the epidemiology of chronic disease did not generally consider infections to be candidate causes in the 1960s, however.

A potentially detrimental tendency to interpret evidence primarily in terms of dominant current paradigms may also be reflected in current research programs that focus on early life development, or on adult life behavioural or psychosocial factors, to the exclusion of other factors. As Major Greenwood put it in 1936:

'At present we are, I think, rather prone to over-emphasize any factor which happens to be a subject of general discussion. At one moment, perhaps, domestic housing, at another national nutrition, attracts almost universal attention. It is the duty of the young statistician not to be a prig and sneer at generous enthusiasms, but to keep a cool head, and always to seek measures of the importance of the several factors. Perhaps that will be easier if he can share the emotional belief I hope I shall never grow old enough to lose, that no honest attempts to make the lives of human beings more agreeable are made in vain, and that apparent failure often leads to ultimate triumph'.²¹

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