HEALTH INEQUALITIES IN THE LIFE COURSE PERSPECTIVE

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Abstract—Life history approaches to the study of inequalities in health provide evidence that the biological and the social beginnings of life carry important aspects of the child's potential for adult health. Biological programming may set the operational parameters for certain organs and processes. Social factors in childhood influence the processes of biological development, and are the beginnings of socially determined pathways to health in adult life. Life history studies of health are beginning to show the important factors associated with the development of these pathways, and the life stages at which intervention to reduce adult health inequalities may be most effective. © 1997 Elsevier Science Ltd. All rights reserved

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INTRODUCTION

Although improvements in health since the Second World War have been considerable, particularly in terms of survival, social class differences in chances of good health have persisted [1–3]. The Black Report, referring to the period which ended in 1980, noted that "... perhaps the most important general finding is the lack of improvement, and in some respects deterioration, of the health experience of both class V and IV relative to class I" [1]. Social class differences in use of services, particularly preventive services, were persistently large during that same period [1]. Since then, according to many indicators, the extent of poverty has also increased in Britain over the last decade [4], and inequalities in health have continued to be found [2]. Since mortality risk and other indicators of health problems are apparently associated with income distribution [5] and with disadvantaged social circumstances [6–10], it is tempting to think about causes of health inequalities largely in terms of demographic scale factors and allocation of care resources. But the problem remains of how to begin to reduce inequalities. In recent years life history studies in health have begun to show the possibility that the roots of health inequalities lie in biological and social experience at the earliest times of life. This paper outlines the life history work, considering separately biological and social factors, and begins with an overview of the variety of methods used to study long periods of individuals' lives.

METHODS

Life history methods offer opportunities for bringing together in a fruitful way recent epidemiological work on the natural history of illness, with life history work from the social sciences on the development of adult life opportunities, and behaviour. The range of methods already in use is now outlined.

The method of linking census data collected at different time points from a sample of a national population has proved valuable in Britain, for example in the study of the relationship of mortality with unemployment [11]. This method offers the advantage of very large numbers, which is valuable in studying rare disorders or events, and the further value of complete national and age coverage, which together with the large numbers makes it possible to study regional and age associated trends. Its disadvantage is the limitation imposed by the necessarily restricted nature of the data collected at censuses. Response bias in censuses is also a potential problem.

Like census linked data, oral history studies offer a rapid return of results in comparison with study methods which involve waiting for time and age to pass [12]. Whilst invaluable for gaining perspective on perceptions of influences on lives, oral history studies have the problems that only survivor populations can be studied, that systematic distortion and reordering of sequence, as well as forgetting, may occur [13, 14], and most importantly that some things from the long-term past, such as measures of childhood respiratory function, may well not be known to the subject and very difficult, if not impossible, to find from records.

One way to contend with many of these difficulties without a long period of waiting for subjects to age, is to use a catch-up or follow-back design.
Catch-up designs begin, for instance, with clinic records and then trace a study population identified in that way to explore long-term outcomes of treatment or risk as, for instance, Robins [15] did in a child guidance clinic population. Barker [16, 17] has used this method extensively, with the help of birth records from the 1920s and 1930s. This method not only avoids some aspects of the problems of recollection, but can sometimes be used to take advantage of the information that a past national or social crisis can provide in order to explore an effect which would otherwise, ethically, be impossible to investigate. Obstetric records kept in the Dutch Hunger Winter of 1944–1945 have been used in catch-up studies to investigate the effects on the fetus and subsequently on the adult, of very poor nutrition at different times in pregnancy [18–20].

Noteable social science catch-up studies include the Cambridge–Somerville study of adult behaviour style and problems [21], and Elder's studies of the population originally selected for the Berkely and Oakland child development studies [22, 23]. Follow-back designs use current information, for instance on a population of those with a particular illness, and controls, to seek earlier life information from records. This method has been used, particularly, in the British national birth cohort studies, for example to investigate early life development behaviour differences between controls and those who had developed schizophrenia [24].

Follow-up studies of many kinds have the important advantage over the designs previously described of being able to collect information during the intervening years between an outcome and presumed causes or risks. On the other hand they take longer to carry out, although they usually produce findings throughout their times of data collection. Beginning times of follow-up studies are usually related to the age at which risks for the problem or illnesses under investigation are thought to begin. So, for example, the Whitehall study of cardiovascular health began in the middle life of its subjects [25]. The British birth cohort studies began as investigations of perinatal mortality risk [13, 26–29], and the concern of the Fels study [30] and of the Oakland and Berkely growth studies [22, 23] was with the effect of the American Great Depression on children's development. The Glasgow based 2007 study of the origins and social distribution of health inequalities avoids the restrictions associated with a single age at commencement and the time of waiting for age to increase, by beginning three follow-up studies at ages (15, 35 and 55 years) at which and soon after which life stage transitions occur [31, 32]. The Framingham epidemiological study of heart disease used an adult sample of a range of ages based in a single town [33]. Reasons for follow-up in the studies that began at birth or in early childhood vary, and may be summarised as concerned with the development of risk, vulnerability and protective factors. New objectives, unforeseen at the outset of such studies, may be added to take advantage of the data already collected [30, 34]. Reasons for follow-up can include assessment of selective intervention made at an earlier time in the same study [35]. Some long-term studies, such as the British 1946 birth cohort [13], recruit only once and then follow up the same population, whilst others recruit gradually over many years [30]. In prospective studies the difficulty of collecting, in the early stage, what will turn out to be useful precursor data for the study of outcomes many years later is unavoidable, and can be exacerbated by a change of study aims. It is reduced in severity in focussed studies, and by the collection of adequate detail of data not unduly restricted to current notions of risk source.

Many of these study designs, with the exception in particular of the census studies, the Glasgow 2007 studies, and the Fels study, have the problem of being tied, through the age of the population, to a particular historical period, so that it is impossible to separate the effects of risk specific to that period from risks that are not time bound, or not bound to that particular time. This is not necessarily a problem, if it is recognised, and comparative studies are undertaken. Examples of such specific period bound risks include, in the 1946 cohort for example, the experience of postwar food rationing from birth to age 8 years, and of childhood spent in a time of heavy atmospheric pollution from parental smoking and from general atmospheric pollution from coal burning; in the 1958 birth cohort study the experience of the years of high unemployment risk in Britain occurred early in the cohort's years of employment. Studies specifically concerned with the effects of social change use a panel design and short-term follow-up of individuals, as in the British Household Panel Study [36].

The overwhelming strengths of prospective studies are their information on sequence and timing of events, and the opportunities they offer to study not only, in the conventional way, the relative predictive strengths of precursors of an outcome, but also the range of outcomes associated with a presumed risk factor or combination of risk factors which may either co-occur or be related sequentially or synergistically [37, 38].

AGE RELATED VULNERABILITY

Biological factors

During the last 10 years there has been an increasing tendency to take a lifetime view of the natural history of some common serious illnesses which usually begin in middle or later life. Before this new approach, conditions such as raised blood pressure and chronic obstructive airways disease (COAD) were commonly believed to be associated with sources of risk which began only in middle
adulthood, and most research was concentrated on that stage in life. This change and progression in perspective can be illustrated with the example of research into COAD.

Before the Second World War there was an urgent need for a solution to the problem of lower respiratory illness in working men; this was a time of heavy industry and a predominantly male workforce. Research concentrated then on the stage at which the problem began, in middle life, and where it was most trouble, in the labour force [39]. The effect of atmospheric pollution on the whole population was also acknowledged, but not resolved until well after the Second World War, in the Clean Air Act of 1956 [40]. Although the mass of research on working men in middle life brought great improvements in working conditions and health in midlife [41], and eventually a reduction in atmospheric pollution from coal burning, it did not greatly reduce the mortality rate from chronic bronchitis [16]. Reid [42], and later Barker [16], took a long-term view, and using data on migration and on time and regional trends in mortality, concluded that biological risk of COAD was likely to be established in early life. Studies of long time periods in the lives of the same individuals added confirmation of this view by showing three things. First that respiratory function tracks during childhood, so that those who have poor function in early life have a strong tendency to continue to have poor function [43]. Second that those who have lower respiratory illness in early childhood are at greatest risk of respiratory illness later in childhood [44-46], and in adult life [47, 48]. Third that low birth weight, an indicator of poor growth before birth, was associated with a significantly increased risk of COAD and poor respiratory function in adult life [16, 48]. Barker [17] concluded that poor maternal nutrition during pregnancy, and other adverse influences on the developing foetus, such as mothers' smoking, reduced the opportunity for optimal development of organs associated with respiratory function, and the developmental process was still also at risk in the final stages of the baby's respiratory development during the first year of life. Thereafter, it is hypothesised, the individual can function only within the parameters set during this unique developmental opportunity. Barker [16, 17] describes this process as biological programming.

Comparable hypotheses on the development of the kidney, the cardiovascular system, and glucose tolerance in adult life have been developed by Barker et al. [16, 17], and similar suggestions of factors affecting children in early life, during a uniquely occurring period of development, have been put forward in neurological studies [49]. However, since early life effects on biological development are hypothesised to determine the operational parameters of a biological system, then when demands on the system become too great problems would be expected. It is suggested, for instance, that kidney development that is less than optimal may not become a problem unless "the system is stressed, for example by high salt intake and thereby becomes unable to maintain the volume and composition of bodily fluids" [17]. Hales and others argued that diabetes is a consequence of poor nutrition during critical periods of foetal life and infancy with consequent impaired development of beta cell function. If poor nutrition continues reduced ability to produce insulin is not a disadvantage. It becomes so only if nutrition becomes abundant, when increased demand for insulin outstrips the capacity for production. [50]

Much work remains to be done to validate the proposed biological programming hypotheses. It is not yet known whether such biological risk, apparently established early in life, is a source of vulnerability which necessarily requires a later life trigger, other than ageing, to activate its effects. Nor is it known how such early life effects interact with processes of ageing, nor with genetically defined risk. It may be that it is possible to escape from the apparent determinism of biological programming, through environmental or genetic means. Current work in a number of centres is exploring such questions, and biological and clinical researchers are investigating the processes by which the proposed programming is established, so that it may be better measured. In terms of COAD the programming hypothesis continues to receive support from laboratory studies in humans and from animal research [51].

Age related social factors

Socio-economic circumstance is a collective term for a wide range of factors which include not only occupational status and security, and at an earlier age educational attainment, but also housing environment and tenure, and family circumstances. Each of these factors seems likely to act differently to affect health at each stage in life. In this section evidence is presented of the range of social factors found to be associated with current health and future health potential in early life, infancy, childhood, adolescence and adulthood. Then the possible processes that may account for such associations are discussed.

Poor social circumstances at birth have long been shown to be associated with an increased risk of perinatal death. Originally this was thought to be the result of variation in quality of medical care, and time at which such care in pregnancy first began [52]. But risk of perinatal death was associated also with social class of family of origin, so that in risk terms "each mobile group carried with it, into marriage, the imprint of its class of origin" [52], and it was therefore hypothesised that this showed the effect of poor growth of the mother during her childhood. Short maternal stature was...
another powerful risk factor for perinatal death [52] and this effect, which was much greater in lower social classes, was thought likely to have been the result of poor maternal and child nutrition in the previous generation, and associated probably also with parental smoking [53]. Perinatal risk was much higher among single mothers, and this was thought to be because, at that time, the social unacceptability of single parenting tended to delay attendance at antenatal clinics [52]. More recently, the Barker hypotheses [16, 17], described in the previous section, implicate poor socio-economic circumstances before birth as risks to intrauterine development that were incurred in the mother’s own generation. Here again parental smoking, maternal alcohol consumption and levels of nutrition are all known risk factors, and each varies with socio-economic circumstances.

In infancy, socio-economically differentiated factors continue to act on children’s health. Parental smoking, poor housing, and nutrition habits continue to have direct effects on the child’s risk of current illness [54], and on future respiratory capacity [17, 48]. Rates of growth are faster in those whose families are in higher socio-economic circumstances [55]. Poor nutrition at this time has, like parental smoking, both a current and a long-term effect. Another long-term effect begins at this time, which is also strongly differentiated by current socio-economic circumstances, namely opportunities for educational attainment. Children in poorer home circumstances, and with parents with low educational attainment, have reduced chances of preschool experience [27, 56], which provides a disadvantaged beginning to the school years [56, 57], and a consequent long-term health disadvantage, since, in adult life, little or no educational attainment is associated with poor health related habits and health [7, 58]. Findings on family circumstances, in terms of parental relationships and physical health in infancy, have shown associations between quality of maternal care and health [59], that infants and children in poor socio-economic circumstances are at greater risk of injury [60, 61], and that those in families experiencing chronic parental emotional disruption are at greater risk of disturbed behaviour [62], and intentional injury [61].

During childhood the most consistent socio-economic difference in health is the variation in height growth [63], which in due course becomes associated with adult health risks. This continuation of socio-economic differences in speed of growth and in achieved height seems to be the result of continuing socio-economic differences in nutrition and possibly also exercise [64, 65]. Data from a longitudinal study showed that poor home physical environment, low levels of parental education, and large family size were also associated with height growth [63, 66]. Since upward paternal (in the past, and probably now parental) social mobility is associated with accelerated growth [63], this general indicator of improved circumstances within the family emphasises the importance of socio-economic circumstances for growth. Also in childhood there are significant social class differences in morbidity, with a greater illness burden falling on children in the lower socio-economic groups [67]. Poor health during childhood is associated with reduced educational attainment [57]. Health associated habits of parents and children also have long-term implications in that, for example, children who begin to smoke, and those who live with parents who smoke, have significantly reduced development of respiratory capacity [68].

In adolescence, whilst there is little apparent socio-economic variation in many aspects of health [69], there is such variation still in the differences in growth already evident at earlier ages [63], and therefore in potential for health in adult life. For example, although there is little social patterning in respiratory function at this stage, the potential for the significant adult social variation in respiratory health is already present in terms of the effects of smoking by individuals and by their parents on adolescent respiratory capacity [68], and in the influential early life factors of birth weight, socioeconomic circumstances and lower respiratory illness [17, 48]. Similarly the ground work for socio-economic variation in adult height has already been prepared [63], and the social variation in educational attainment is continued at this stage in the socio-economic differences in age at school leaving, and in going on to further and higher education [70]. Within the family, during adolescence, parental concern for educational attainment continues to be a powerful predictor of attainment and of occupational status [13], and family disruption caused by parental divorce and separation increases the risk of the child not continuing into further and higher education [71, 72]. Sweeting and West [73] have shown family conflict to be associated with a somewhat raised risk of physical health problems in young women adolescents, and confirm the association of family functioning with later occupational opportunity.

By early adulthood growth is completed, not only in height but also in all other aspects of development [17]. The patterns and extent of growth carry the imprint of the social environment of the previous generation, via the mother, as well as of the time in utero, and in childhood. Educational attainment makes its mark in adulthood in a number of ways, most evidently in occupational and socio-economic status; and these in turn are strongly associated with health. Health related habits of smoking [74, 75], nutrition [58], exercise [75, 76], and alcohol consumption [58, 75], all have a strong socio-economic bias; they are also each associated with education, which generally has the effect of reducing adverse health habit practices.
Consequently, it is unsurprising to find that biological risk factors associated with these health related habits, in particular overweight and obesity, are also significantly differentiated by social class [75, 77, 78]. Findings from a longitudinal study showed that women who came from manual social class families and held non-manual social class occupations in adulthood (at 36 years) tended significantly to show the prevalence of low obesity, as in the class they had joined, compared with the class they had left [77]. Unemployment is also commonly found to be associated with ill health [79-81] and premature death [82], and a life history approach to this subject shows the importance of pre-existing vulnerability [83]. Longitudinal studies have shown that unemployment is a greater risk for young adults from less favourable family circumstances, with low or no educational attainment, and with early signs of vulnerability to ill health in adulthood [7, 83]. Experience of divorce and separation is associated with raised risk of self-reported ill health [75] and with psychiatric morbidity [80], but little work has been reported on its association with physical health. It seems an important area for new work [73], particularly since it is an experience which in childhood affects mental health, and increases vulnerability to adult mental health problems [84-86], and since parental divorce or separation is associated with raised risk of offspring’s divorce or separation in the following generation [87].

While most life history research on social factors and health has concentrated on the association of childhood factors with adult health, there has been less work, although some hypothesising in retrospect from studies of the elderly, on the relationship of adult midlife experience with later life health, and even less on the relationship of childhood factors with old age. Research on these areas will be increasingly reported, as current studies of cohorts begun in mid-life and at birth continue into later life, for example the 1946 and 1958 national birth cohort studies [13, 88], and the British Regional Heart Study [82]. There are a number of psychological studies of middle life [89] and of ageing and cognitive change with age [90], which include new concern with social factors and ageing; of particular interest in life history research is the measurement in longitudinal studies of ageing of biological and psychological latent reserves, and adaptability or plasticity of individuals [90]. Some medical studies of ageing have generated hypotheses about associations between mid-life health related habits and, for instance, later life skeletal state and risk of damage [91], in which social habits play a part, through their influence on health related habits [58].

Such a range of social factors is implicated in the life history studies of health, that it is difficult to categorise their possible processes of operation. Unlike the processes suggested by the biological programming hypotheses, the social hypotheses do not propose that their effects operate solely or mainly at critical developmental periods. From the life history studies of childhood and adolescence it may be concluded that social factors probably operate in a cumulative fashion. Children of families in favourable socio-economic circumstances have what is, in effect, a stock of social capital, which is enhanced if one or both parents have further or higher educational attainment [7, 13, 92], and still further by strong parental concern for their child’s education [57, 93]. These beginnings predispose children to higher educational attainment which is, in turn, associated with better health in adulthood [94]. Conversely, vulnerability to physical ill health in childhood and later adult life is associated with poor parental socio-economic circumstances, and low levels of parental education and concern [7, 13, 48], and consequent lower levels of educational attainment with chances of lower occupational status, greater vulnerability to unemployment, risk of more adverse health related behaviour in adulthood, and poorer health.

**BUT HOW MAY THESE SOCIAL FACTORS HAVE THEIR EFFECTS?**

In childhood, for instance, do poor home circumstances affect health in a bio-social fashion, because of the concomitant increased chances of poor nutrition, the greater likelihood of parental smoking, the slower rate of growth, and the greater risk of childhood infection and other illnesses, which strike the child at biologically vulnerable times, particularly since children in these poor circumstances are those who are likely to have had a less than optimal development before birth? Are there also psychosocial processes of importance? Do the higher levels and chronic persistence of anxiety about money in poor families, and the poor nutritional, smoking, and exercise habits provide the growing child with poor examples in coping styles and health associated habits which may be difficult to change later? It is possible to argue for a dynamic model of a childhood acquisition of a stock of socially related potentiating factors for later health, susceptible to change from parental social mobility and from education, which seem to make it possible to escape from the adverse health related aspects of early life social factors since, for example, upward parental social mobility is associated with raised chances in education [57] and in height growth [63]. The psychosocial processes seem less easy to escape [95], although here too education is likely to be a key factor. It is worth noting, in terms of assessment of pre-crises psychological functioning, that life history studies in psychophysiology and deviant behaviour suggest that early acquisition of styles of stress management and of behaviour seem to be remarkably stable in the long term [95, 96].
Much of the work on how social factors may operate in adult life comes from studies of social factors and health in relation to crises experienced by individuals, such as divorce and unemployment. This work is beginning to ask how far earlier, precodes, social, psychological, and biological states mediate the effect on health of such crises [37, 79, 84]. Conclusions for physical health are most often in terms of psychological stress leading to biochemical and immunological change [79], but such processes have yet to be fully explored.

Studies of the association of social factors with adult health, not associated with crises, have been greatly concerned with poverty. Findings about the effects of poor housing, chronic anxiety about money, and adverse health related habits need now to be supported with studies of how such social factors have their effects on the health of individuals. Life history approaches will be necessary in such work on processes, in order to take account of earlier life established vulnerability and protective factors, already known, such as parental social class and own educational attainment [7, 48, 58, 77] as well as more recent attributes and experiences.

**TIME ASSOCIATED VULNERABILITY**

To understand more fully the effects of the development of vulnerability and protective factors in a social context, and to take account of how they may affect public health in future, it is necessary also to appreciate their changing effects over historical time. This is particularly important in the light of the biological programming hypotheses. If they prove to be correct in proposing a short and unique developmental time early in life for many biological aspects of health capital, then amelioration of social factors that adversely affect biological development has only one brief time of opportunity in the childhood generation, although much greater time in the maternal generation. And if the biological programming proposals are correct then the imprint of the social time of fetal and infant biological development will remain. In that case, examination of social effects in past times should provide clues about the later life health of populations now in middle life.

The notion of time associated vulnerability is now examined in terms of biological and social risk factors.

**Time associated biological vulnerability**

The biological factors associated with the programming hypotheses fluctuate with time. Despite the fact that the case is not yet proven, if we take the balance of probabilities to be that biological programming does determine the parameters within which biological systems operate, then the effects of three kinds of time associated biological factors which affect biological vulnerability must be taken into account. Each, whilst not challenging the idea that later life biological parameters may be set in early life, nevertheless disputes the notion that such programming would be invariably deterministic.

The first of these three factors is the biological action on health of health associated habits. These habits have their effects throughout the life course, and their prevalence varies greatly over historical time. Those born, for example, at a time of low rates of maternal smoking will, if these hypotheses are correct, be significantly less likely to carry that aspect of raised risk forward in life to affect blood pressure and respiratory function in adulthood. Cigarette smoking in women increased in popularity some years after it first became common among men, so that those who were children in the earlier time will have been exposed to less risk than those who were in utero or infancy at the time when smoking popularity was at its peak in both men and women [97]. The effects of gender differences in the time of first popularity of smoking are seen now in gender differences in lung cancer rates [98]. In middle life health is vulnerable to the effects of overweight, smoking, little exercise, and poor eating habits. Because the life course of those born, say 50 years ago, crossed periods of great change in such factors, it is possible to be born into a period of high prevalence of parental smoking, and lived middle life in a time of much reduced likelihood of smoking. One life history study showed how risk to health from smoking was greatest in those born into manual social class families at a time when parental smoking prevalence was high, who then became smokers in adulthood [48]. Similarly many people now in middle life in Britain were born into a period of food shortages and a higher likelihood of physical activity, but now in adult life experience raised risk of overweight through poor food choice and low exercise patterns.

The second aspect of biological vulnerability that changes with historical time is that of periodic epidemic instances of illness. A number of conditions have been hypothesised to affect the later life health of those who experienced them in childhood. Some viral infections including mumps, have been shown to increase the risks of insulin dependent diabetes mellitus in later life, together with a range of other autoimmune destructive processes [99]. Measles has been implicated in the aetiology of inflammatory bowel disease [100], and the experience of living as a child during poliomyelitis epidemics has been found to be associated with a later raised risk of motor neurone disease in population studies [101]. These are period effects, in that all are now greatly reduced.

The third aspect of time associated biological vulnerability comprises the changing availability of medical care and the changing effectiveness of treatment. Each of these factors has changed greatly over the last 50 years, affecting the health of individuals at all ages, and enabling demographic
change. The latter has occurred, for instance, through the introduction of reliable contraception, and reduction of the burden for mothers of infectious illness in children, which has helped to enable women to take a full role in the labour market. The ageing structure of the British population is also associated, in part, with improvements in medical care of the older generation, and the control of pregnancy and the postponement of first pregnancy in younger generations [102]. In terms of early life health and the biological programming hypotheses, improvements in effectiveness and availability of care cannot be straightforwardly interpreted as providing the bases for a healthier future adult population, since these beneficial changes take place in a social context, and have to compete for effectiveness with other factors. Such retrospective interpretations may not be straightforward. In Britain, for example, the beneficial effects of cost free access to medical care and the introduction of antibiotic therapy, which both occurred in the late 1940s, should have had a beneficial effect on one of the known long-term risks to respiratory health, namely the early childhood experience of lower respiratory illness [16, 48]. But at the same time maternal smoking, another known risk factor, increased considerably as fashions in smoking changed and is now a class biased risk factor [97].

Time associated social vulnerability

Vulnerability to ill health is affected by the social environment, both through social policy and through attitudes, behaviour and taste. Each of these sources varies across historical time, and affects health differently at different times in the life course. In this section examples are first given of policy, attitudes, behaviour and tastes that affect health. Then processes of such effects in different stages of the life course are discussed.

Since the end of the Second World War two kinds of social change in Britain are likely to have influenced health, namely change brought about by policy, and change arising from attitudes, behaviour and fashions. Policy associated change that has affected health is of three kinds. The first comprises the postwar policy changes introduced to alter the national wealth generating base from dependence on manual labour to dependence on non-manual, from muscle to brain. This great social change was achieved not only by technological means, but also by education. Since the Second World War rising numbers have stayed on in education for greater periods, and attained higher levels of qualifications, but until the mid 1970s the proportion of women gaining higher qualifications was much lower than that of men [70].

The second policy change that has affected health is that concerned with income distribution and the opportunity for employment. Whilst home circumstances have improved greatly in terms of such things as home heating, and availability of washing machines and cars [103], indicators of poverty have, in both Britain and the United States, shown a rising problem over the last 20 years [4, 104]. In Britain before housing costs the real median income of the bottom fifth increased by just 3% between 1979 and 1990–1991, while the corresponding figure for the top fifth is 49%. After housing costs the real median income of the bottom fifth actually fell between 1978 and 1990–1991, but increased for all other groups. [103]

These changes in income are associated also with changes in economic policy which brought rising levels of unemployment to Britain, particularly during the 1980s [103].

Changes in such health associated social factors as behaviour, fashions and tastes have also been considerable. Divorce rates have risen steeply in Britain since the early 1970s [103], fashions for exercise appear to have increased in some sections of the population [76], tastes in food have changed [105], and smoking and alcohol consumption habits have varied considerably over time [97, 102, 103].

The possible effects on health of such changes, in education, income, employment and divorce are now discussed, with particular reference to variation in effect according to life stage.

Trends in education have tended to be favourable to health, in that increasing numbers of young people are gaining qualifications at levels which have so far been associated with favourable changes in health related habits [13, 70]. In Britain the rising numbers of women going on to further and higher education is likely to benefit the future health of their children. Whereas proportions of women undergraduate students are now equal to those of men, this increase was not achieved until the 1970s [106]; before then the proportions of mothers with educational qualifications at the level associated with receptivity to new ideas in health related habits was as low as 18% [58]. The delay in improvements in women's educational chances will have affected the health of infants and children born before the increase in educational opportunity for women and, if the biological programming hypotheses are in some measure correct, the adult health of those children also. Rising opportunities in education have been instrumental in increasing upward social mobility, and this experience may bring a new source of stress to those already biologically vulnerable. Forsdahl hypothesised that the observed raised risk of premature death in adult life from arteriosclerotic heart disease in populations born into poor socioeconomic circumstances might be accounted for by their shift, in adult life, to relative affluence. He noted that raised cholesterol level caused by affluent living was possibly the process through which the observed association had its effect [107]. In terms of the biological programming hypotheses it may be that, as Barker et al. argue [17], early life circum-
stances set the biological parameters for adult blood pressure. Following either argument it seems possible that risks to adult life blood pressure will be incurred by those whose biologically programmed capacity is later challenged. In times of rising affluence challenges from, for example, adverse body shape and inappropriate nutrition, will bring damage especially to the biologically vulnerable. Biological vulnerability is strongly associated with poor socio-economic circumstances in early life [16], which are associated also with the likelihood of development of an adverse chain of social risk factors, which increase the risk of the low educational attainment associated with adverse adult body shape and nutrition [58, 77]. And so, although adult vulnerability may be increased in prevalence by contemporary historical social change, the origins of both biological and social vulnerability for the individual are argued to lie in early life. Although not strictly a health effect, it is worth noting that since greater educational attainment tends to be associated with raised chances of geographical mobility and reduced chances of adult offspring caring for elderly parents, there are important care policy effects also associated with the postwar educational improvements [108].

Associations between income and health have been well documented for both children and adults [1, 109, 110]. Life history studies have yet to show whether those born into non-manual class families have better health chances in adulthood if their income is then low, compared with those born into manual class families. It has been shown, however, that low social class in childhood is associated with poorer adult health [94], unless combined with poor educational attainment. Rising paternal social mobility has been shown to increase educational attainment [57] and children’s height growth [55]. As in education, the question of how indicators of parental and own income have their apparent effect is not known, but in childhood poor nutrition, and cold and damp housing are likely to be implicated. In adulthood, anxiety about financial matters is judged likely to affect health.

The effects of unemployment on health and survival have been extensively explored [79, 81]. Because employment is in many important respects the basis of status, and therefore of identity, it is not surprising that loss of employment through redundancy or retirement is associated with raised risks to health. It may be that during unemployment the feelings of reduced control and disturbance of the pattern of life, and the reduction in psychological support that employment provides in terms of self-image, companionship, and the provision of roles additional to those of home life, may all be part of a pattern of accumulating risk to mental and physical health. It seems clear that unemployment is damaging to mental health, and has been shown to be damaging to prospects of future employment [79]. In due course changes in prevalence of mental illness in relation to the national rise in unemployment may be detected. Unemployment has been shown to be adversely associated with smoking and alcohol drinking habits, and with increased body weight [82], and it is unlikely that the excess of those with very serious illness among the unemployed explains the relationship between unemployment and mortality [79]. Studies that take the longer-term life history view of health risk associated with employment and unemployment show that health risk is raised in those with poor social capital. A forthcoming report using longitudinal data from the 1958 British birth cohort shows that childhood health potential, in terms of height at age 7 years and social adjustment at 11 years, was associated with raised risk of unemployment in early adult life [83]. It seems arguable that parental unemployment will present serious risks to the immediate and long-term health of children.

The effects of parental divorce or separation on health have been studied extensively in relation to adult mental health [37, 84–86]. Two studies [62, 111] have found that children who experienced parental divorce or separation achieved significantly lower educational qualifications than would have been expected. It was hypothesised that this was perhaps because of moving schools, as a result of moving home, or of the child’s perception of the need to leave school at the earliest opportunity in order to increase the family income or to begin an independent life. It may also be that changes in, and perhaps greater reliance on, age peer groups following parental separation or divorce play a part in this hypothesised process. Whatever the explanations for this finding, the effect on offspring of experiencing parental divorce or separation was found in one longitudinal study to be a reduction in income levels in subsequent adult life, an increased chance of lower occupational levels, and a raised risk of unemployment [62]. It was found, in the same study, that those whose parents had divorced or separated were themselves at significantly increased risk of divorce, of smoking, and among women, of higher than average alcohol intake, and in men higher than average smoking [62]. Another longitudinal study has shown lower self-esteem amongst offspring living in circumstances of family conflict, and significantly reduced reports of well-being and of increased numbers of physical symptoms were also made by female offspring living in such circumstances [73]. Protective factors during childhood, such as parental encouragement of the child in educational terms, may co-exist with the adverse effects of parental divorce and separation, and in adult life protection can come in the form of a supportive partner [37]. The effect of divorce or separation on adult health has been less extensively studied [112], but uninterrupted marriage has been shown to be associated with good health and chances of survival [113].
CONCLUSIONS

Life history studies offer new opportunities to examine processes that generate health inequalities for the individual. New methods for undertaking such studies show that it is not always necessary to use long-term prospective methods.

Biologically it seems arguable that in some important respects early life health and development delineate the parameters of health possibilities for adulthood. In that case the adverse social factors that affect infant health and development would be important originating aspects of social differences in health and health potential during adulthood. Social factors experienced in childhood encompass also many important aspects of the potential for later educational attainment, and for the socio-economic circumstances of adult life. These factors make it possible for children born in poor socio-economic circumstances to experience upward social mobility, and they are associated also with improved chances in health. So although biological programming may have a deterministic effect on the range of adult health parameters, the social and family circumstances of childhood are the beginnings of pathways which will be protective to health or increase vulnerability to ill health. The social factors that affect both biological programming and the social and educational pathways from childhood to adult socio-economic circumstances, vary with historical time, and so the extent of health inequality in actuality and in potential in a cohort of a given age is likely to be different from that of a cohort of another age.

So far, life history contributions to the study of inequalities in health show that health is a lifelong development for the individual. The implication of these findings is that chances of reduction of inequalities for any given generation will be greater, the earlier that attempts at reduction are begun. It is unlikely that health inequalities can be easily or rapidly reduced, increasingly so as the individual ages, since individuals carry an accumulation of health potential which is hard to change.

REFERENCES


