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The development of life course epidemiology

L'émergence de l'épidémiologie biographique

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Abstract

The present paper reviews the development of life course epidemiology since its origins during the 1990s from biological programming, birth cohort research and the study of health inequalities. Methods of studying the life course are examined, including birth cohort studies, linked register datasets and epidemiological archaeology. Three models of life course epidemiology are described: critical periods, accumulation, and pathways. Their conceptual and empirical differentiation can be difficult, but it is argued that accumulation is the underlying social process driving life course trajectories, while the critical period and pathway models are distinguished by their concern with specific types of aetiological process. Among the advantages of the accumulation model are predictive power, aetiological insights, contributions to health inequality debates and social policy implications. It is emphasised that the life course approach is not opposed to, or an alternative to, a concern with cross-sectional and current effects; major social disruption can have a large and immediate impact on health. Other limitations of the life course approach include a spectrum of impact (life course effects can be strong in relation to physiology, but often are weaker in relation to behaviour and psychological reactions to everyday life) and, more speculatively, the possibility that life course effects are diluted in the older age groups where morbidity and mortality are highest. Three issues for the future of life course epidemiology are identified. Many life course data are collected retrospectively. We need to know which items of information are recalled with what degree of accuracy over how many decades; and what methods of collecting these retrospective data maximise accuracy and duration. Second, the two partners in life course research need to take more seriously each other's disciplines. Social scientists need to be more critical of such measures as self-assessed health, which lacks an aetiology and hence biological plausibility. Natural scientists need to be more critical of such concepts as socio-economic status, which lacks social plausibility because it fails to distinguish between social location and social prestige. Finally, European comparative studies can play an important part in the future development of life course epidemiology if they build on the emerging infrastructure of European comparative research.
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Résumé

L'article présente l'émergence de l'épidémiologie biographique dans les années 1990 à partir de trois domaines de recherche : la « programmation biologique », le suivi de cohortes de naissance et les inégalités de santé. Après avoir passé en revue les méthodes utilisées dans l'étude des parcours de vie : le suivi de cohortes, la mise en relation de données appariées à partir de différents registres et « l'archéologie épidémiologique », les auteurs décrivent trois modèles de l'épidémiologie biographique : les périodes critiques, l'accumulation, les itinéraires. S'il peut être difficile de différencier ces trois modèles d'un point de vue conceptuel et empirique, les auteurs défendent l'idée selon laquelle le principe d'accumulation est le processus social fondamental qui oriente le cours des trajectoires alors que les deux autres modèles concernent des mécanismes étiologiques spécifiques. Le modèle d'accumulation présente, entre autres avantages, d'avoir un bon pouvoir prédictif, d'éclairer les mécanismes étiologiques, de contribuer aux débats sur les inégalités sociales et d'orienter les politiques. Toutefois, l'approche biographique ne prétend se poser en alternative aux approches transversales centrées sur les effets à court terme, certaines ruptures sociales pouvant avoir un impact important et immédiat sur la santé. Elle présente en outre certaines limites ; si elle parvient à mettre en évidence des effets robustes du parcours de vie sur certains indicateurs biologiques, les liens avec les comportements et les réactions psychologiques quotidiennes sont plus ténus. On peut également penser que les effets du parcours de vie se diluent aux âges élevés, là où la morbidité et la mortalité sont les plus fortes. Les auteurs pointent trois orientations pour la recherche à venir. Tout d'abord, la plupart des données biographiques étant recueillies

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rétrospectivement, il est nécessaire de connaître le degré de précision qui peut être atteint dans la mémorisation d'informations portant sur plusieurs décennies et d'identifier les méthodes qui optimisent cette précision. En second lieu, les différentes disciplines impliquées dans l'approche biographique doivent travailler en concertation plus étroite. Notamment, les chercheurs en sciences sociales doivent faire preuve de circonspection lorsqu'ils manipulent des données déclaratives de santé qui n'ont pas de validité biologique ; de même, les biologistes doivent être plus rigoureux lorsqu'ils recourent à une notion telle que le statut social qui confond position sociale et prestige. Enfin, le développement de l'épidémiologie biographique doit s'appuyer sur l'infrastructure émergente des recherches comparatives européennes.

1. Origins

During the 1990s three strands of social and medical research came together in life course epidemiology. Biological programming, which holds that organ development in utero and during early infancy determines the maximum functional capacity that an individual can attain; and that thereafter, for the remainder of the life course, the individual functions only within the limits set during this unique developmental phase. Lung development in utero and during infancy, for example, influences the likelihood of developing chronic obstructive pulmonary disease in later life; kidney development during foetal and infant life influences the likelihood of later high blood pressure; pancreatic development influences later diabetes; and so on [1,2]. Various markers of foetal growth and development were investigated, but birth weight was used most widely.

The second strand came from the British birth cohort studies, particularly the oldest of these, the National Survey of Health and Development, born in 1946 [3]. By the 1990s the members of this cohort had reached the stage of the life course when adult chronic diseases start to become clinically manifest. The idea of accumulation was used first to describe the risk of lower respiratory disease and reduced lung function at age 36 years; risk which accumulated with chest disease and a poor and crowded home during early childhood, living during later childhood in an area of high atmospheric pollution from coal burning sources and cigarette smoking during early adulthood [4].

The third strand came from health inequalities research, where it had long been known that social class differences in the prevalence of behavioural and other classic risk factors accounted for only around one-third of the class difference in mortality risk [5]; and, more recently, that mortality risk was graded *fine grain* against socio-economic circumstances [6]. The social structure was suggested as the mechanism that could produce this fine-grained distribution of mortality, by structuring exposure to a range of non-behavioural hazards, which are clustered cross-sectionally and accumulated longitudinally via advantage or disadvantage in the various spheres of life [7].

These three strands—biological programming, accumulation, health inequalities—combined with epidemiology's traditional concern with cause-specific disease pathology [8,9] to ensure that the life course approach became by the late 1990s an established theme in social epidemiology [10–12].

2. Methods of studying the life course

Birth cohort studies are the best method of studying the life course. Data on pregnancy, birth and neonatal development are enhanced prospectively throughout life; at short intervals during childhood and less frequently later, timed to coincide with key biological and social events. The study design combines the security of prospective data with certainty about temporal sequencing; and, when sequential birth cohorts are used together, the ability to separate age, period and cohort effects. Good fortune and the scientific vision of a few people, like John Bynner, mean that Britain has four birth cohorts (1946, 1958, 1970, 2000), so it is not surprising that British social scientists and, later, medical scientists have made a substantial contribution to life course research. Data from the 1946 cohort at ages 36 and 43 years, for example, demonstrated the importance of childhood illness to adult health; parental social class previously had been shown to influence the incidence of serious illness during childhood, particularly during the first 5 years of life [13]. Illness and disability during childhood, together with parental and adult social class, were found to influence health at age 36 years. Disadvantaged parental social class and low educational qualifications predicted poor diet [14] and obesity [15] at age 36 years, while those with the best health were characterised by both advantaged parental class and high educational qualifications [16]. The same factors, parental social class, adult social class and illness and disability during childhood, independently influenced the chance of physical disability and handicap at age 43 years; with the socio-economic consequences of disability being more severe for manual workers [17]. In a further example from the 1946 cohort, early life factors, including breastfeeding during infancy and physical growth and cumulative socio-economic disadvantage during childhood, influenced the timing of the menopause more strongly than adult factors [18,19].

Birth cohort studies are not without their disadvantages; sample attrition and bias tend to accumulate over time, information that becomes important only subsequently is not collected at the relevant time, repeatability may lock the study into not-best measures, and so forth. More important than these for social epidemiology, birth cohort studies take many decades to reach the high morbidity–high mortality age groups. As a

result, other study designs have become popular, including historical studies extended by follow-up data, longitudinal studies enhanced by retrospective information, and data-linkage using information from population censuses, civil registers and social surveys. Scandinavian countries are particularly blessed in this respect, because information relevant to medical research from a variety of sources can be linked via each citizen's unique identification number. One study, for example, linked retrospectively the blood pressure of some 1300 Swedish men aged 50 years with their archived birth weights. The relationship between birth weight and blood pressure at age 50 years was found to be linear and inverse, as predicted by biological programming, but rather weak and statistically significant only for systolic pressure [20]. A second study used Swedish record linkage to track through life some 14,600 people born during 1915–1929, including all deaths up to 1995. Birth weight was found to be related inversely to later cardiovascular disease mortality among men but not, at conventional levels of statistical significance, among women. Ischaemic heart disease mortality in later life was one-third lower in those from the top quarter of the distribution of birth weight-for-gestational age than in those from the bottom quarter of its distribution [21]. While these Swedish studies tested the biological programming hypothesis, a Norwegian study examined accumulation. National census data from 1960, 1970, and 1980 were related to the approximately 180,000 deaths up to 1985. Mortality risk was examined in relation to lifetime socio-economic circumstances, expressed as both the type of circumstances and their temporal sequencing. Mortality risk among men, for example, was found to be highest for life courses which combine: an education that ended at primary level; employment in manual occupations followed by early retirement from work; and housing conditions which were poor in earlier life and often remain poor through to later life [22,23].

Greater ingenuity is required outside Scandinavia. One approach has been to reactive studies conducted several decades previously, either by finding which study participants have been registered as dead, and at what ages, or by tracing study participants to their present-day locations and re-interviewing them. The West of Scotland Collaborative Study, for example, screened some 5500 male employees in the early 1970s, with 21 years of follow-up mortality data by the late 1990s. Analysis of these mortality data showed that most of the prevalent causes of death were related independently to both childhood and adult social circumstances [24]. The Boyd Orr study provides a second example. Originally some 5000 British children were examined during the late 1930s for a survey of child health and nutrition. The majority were re-contacted in the mid-1990s, now mostly aged 65–75 years; with a small sub-sample interviewed during 1998. Blood pressure, lung function, height and weight were recorded at the 1998 interview. Analysis of these physiological measures showed that they relate to the life course in different ways [25]. Childhood circumstances were found to have an independent effect on blood pressure, but not lung function, in early old age. Childhood height, as measured in 1938, was related inversely in the same individuals to blood pressure in

1998. This relationship survived adjustment for a range of potential confounders, including the inclusion of both child and adult height in the same model, so providing an estimate of child physical growth. Child growth in this dataset predicted blood pressure 60 years later [26].

Such epidemiological archaeology produces datasets that lack information from the period between the original survey and later follow-up. The deficit can be filled with data collected retrospectively, preferably with the well-recognised biases inherent in retrospective data minimised by the use of a life-grid. The life-grid technique uses a framework of securely remembered dates (external and personal events such as wars and own marriage) to increase the accuracy of recall of the dates of changes within the areas of interest, such as residences and occupations [27]; and enhances recall of conditions in these, to continue the example, residences and occupations by focusing on the humdrum, background details of life [28]. Life-grid data on the Boyd Orr sub-sample, for example, included information on the characteristics of adult employment, with the evidence suggesting child growth interacts with adult working conditions, such that raised blood pressure in early old age is most likely when slow growth in childhood is followed by lack of job autonomy in adulthood [25].

3. Models of the life course

Life course epidemiological processes increasingly are discussed in terms of three models: critical periods, accumulation and pathways [29]. The critical period model extends the original idea of biological programming to include infant and child development, so that it describes, for example, both intra-uterine and child growth. The notion has been extended also to social development, in the form of key social transitions [30,31]. The accumulation model builds on the previously noted tendency of the social structure to cluster advantages or disadvantages cross-sectionally and to accumulate them longitudinally. Cross-sectionally, a person whose working environment is free of hazards is likely to reside in good quality housing, to live in an area with little air pollution and to have an income which permits a varied diet. In contrast, someone who is exposed to physico-chemical and psychosocial hazards during work is at greater risk of occupying damp and inadequately heated accommodation, of being exposed to industrial and road traffic exhaust atmospheric pollution in their area of residence and of earning an income which restricts dietary choice. Longitudinally, a child raised in an affluent home is likely to succeed educationally, which will favour entry to the more privileged sectors of the labour market, where an occupational pension scheme will provide financial security in old age. At the other extreme, a child from a disadvantaged home is likely to achieve few educational qualifications and, leaving school at the minimum age, to enter the unskilled labour market where low pay and hazardous work combine with no occupational pension, which ensures reliance on welfare payments in old age [32]. The pathway model shares similarities with the accumulation model, but differs on the timing of aetiological exposure, with early advantage or disadvantage setting a person on

a pathway to a later exposure that is the aetiologically important event. Women who are successful in higher education, for example, tend to delay their first pregnancy until older ages, which increases their risk of developing breast cancer; in this example, success in higher education sets them on a pathway towards the later, aetiologically important event [33].

The present paper considers the accumulation model to be the most fundamental of these three models, because it describes the underlying social processes that drive the life course's impact on health. In the Boyd Orr life-grid sub-sample, for example, lifetime social disadvantage was measured as years of exposure to a range of occupational and residential hazards to health [34]. Socio-economic disadvantage during childhood forward-predicted the amount of disadvantage experienced throughout life, with the most disadvantaged one-sixth in childhood experiencing subsequently, across their lifetime, around four times as much exposure to health hazards as the most advantaged one-sixth [35]. Similarly, social disadvantage in early old age back-predicted the amount of disadvantage experienced throughout life, with the more disadvantaged half of people in early old age having experienced around twice as much exposure to disadvantage, across their lifetime, as the more advantaged half [36]. In other words, as implied by the accumulation model, the social structure ensures that childhood disadvantage tends to be followed by health-relevant disadvantage across adulthood, which tends to be followed by social disadvantage in early old age. In one sense, the critical period and pathway models add little to this process of accumulation. Birth weight is graded by parental social class [37]; and birth weight, in turn, predicts the level of social deprivation experienced during childhood [38]. As a result, it is legitimate to regard the critical period model as a mechanism by which socio-economic disadvantage accumulates across the generations. If the critical period model is a sub-set of accumulation, the pathway model is virtually identical to accumulation. In its early formulation "… *the pathways model focuses on the cumulative effect of life events along developmental trajectories, and it thereby implicates conditions of life throughout the lifecycle in adult disease causation*" [33]. To argue that the critical period and pathway models are less fundamental than accumulation is not the same thing as dismissing them. The life course approach demands both biological and social plausibility; and the great strength of the critical period and pathway models is their attention to the aetiological part of the life course approach. There is good evidence that sub-optimal foetal and infant growth does have an independent, if modest, effect on later adult health (see, for example, the previously mentioned Swedish record linkage studies). Similarly, the pathways model is correct to distinguish between social disadvantages that are and are not relevant aetiologically to the health outcome of interest. Life course epidemiology is a collaboration between the social and natural sciences. The natural science process of aetiology supplies the disease outcomes, while the social science process of accumulation ensures the social patterning, by social class and suchlike, of these diseases.

Empirically also there are good reasons for treating accumulation as the fundamental process in life course epidemiology. The most serious attempt to date to distinguish empirically between the three models, using data from the Stockholm heart epidemiology programme, found that the theoretical differences between the models, in terms of the timing of the hazardous exposures, were not matched by differences in mortality risk [39]. As well as the empirical difficulty of distinguishing critical period and pathway models from accumulation, there also may be little point in trying, because empirically all three are intermeshed. As mentioned previously, in the Boyd Orr lifegrid sub-sample slow physical growth during childhood was related to raise systolic blood pressure in early old age. Childhood height also forward predicted among women to the number of years during adulthood spent in occupations with low job control [40]. Slow child growth interacted statistically with low adult job control; those in both the shortest childhood height group and the longest adult low job control group had an adjusted relative increase of 35.2 mmHg (95%CI 6.0, 64.4; $p = 0.02$) and 25.8 mmHg (95%CI 3.5, 48.2; $p = 0.02$) in systolic blood pressure and pulse pressure, respectively [26]. These results could be interpreted as an interaction between a critical period effect (psychosocial stress influencing both prepubertal growth and formation of the mechanisms involved in control of blood pressure in later life) and accumulation (material and psychosocial conditions that produce slow child growth are part of a disadvantaged life trajectory that includes, among other things, lengthy exposure to low job control). An alternative, perhaps more useful, interpretation would point to the pattern of life course accumulation of biological and social disadvantage that is specific to blood pressure at older ages.

4. Advantages of the accumulation model

The accumulation life course model has several advantages, of which the first is predictive power. The afore-mentioned 1946 birth cohort study [4] was the first to demonstrate accumulation in terms of aetiologically plausible hazard exposures. The West of Scotland Collaborative study was the first to demonstrate accumulation in terms of socioeconomic disadvantage. Each male study subject was assigned to three social class positions: social class during childhood, based on father's occupation; social class at labour market entry, based on own first occupation; and social class during adulthood, based on own occupation at the time of screening. The number of times, between zero and three, that subjects were assigned to manual as opposed to non-manual social classes was related to many aspects of health at screening during adult working life, with the best health being found among those who had been in non-manual social classes at all three stages of life. Systolic blood pressure, diastolic blood pressure, serum cholesterol concentration, height, body mass index, forced expiratory volume in one second (FEV_1), and the symptoms of angina and chronic bronchitis were all related in a graded, stepwise fashion to this measure of cumulative lifetime social class. Each move away from thrice non-manual produced worsening health. All-cause

mortality during the 21 years of follow-up showed the same relationship with lifetime cumulative class, being more than 50% higher in the group assigned to the manual social classes on all three occasions than in those assigned thrice to non-manual classes [41]. These findings are consistent with those from the 1958 birth cohort study at age 33 years, where social class differences in a range of self-reported health measures were related to childhood material and psychosocial circumstances and rate of physical growth; educational qualifications and health behaviours, such as tobacco smoking, in adolescence; and early adulthood material and psychosocial circumstances, obesity risk, job security and exposure to psychosocial job strain [42]. In a third example, the health effects of accumulating labour market disadvantage, operationalised as being unemployed or working in unskilled or semi-skilled occupations, were shown in the Office for National Statistics Longitudinal Study. Men aged 15–40 years in 1971 were scored 0–5 according to whether they had reported working in semi/unskilled occupations at the 1971, 1981 and 1991 censuses and reported unemployment at the 1971 and 1981 censuses. The chances of reporting a limiting long-term illness at the 1991 census were graded stepwise by this labour market disadvantage score, with the most disadvantaged group having some four times the risk of a limiting long-term illness as the most advantaged group [43]. Similar results have been reported from the USA [44] and Scandinavia [45,46].

The second advantage of the accumulation life course model is the aetiological insights it can provide. The West of Scotland Collaborative Study, for example, examined cause-specific mortality in relation to both childhood social class and adult social class; and found that diseases relate to the life course in different ways. Most cancers and accidents and violence were related to adult, but not to childhood social class. Coronary heart disease, stroke, lung cancer, stomach cancer and respiratory disease were related independently to both childhood and adult social class. When these relationships were adjusted for adult social class and adult risk factors, the association between lung cancer and childhood class was eliminated, the relationship of childhood class with coronary heart disease and respiratory disease was attenuated, but the relationship between childhood class and both stroke and stomach cancer was not altered. In other words, lung cancer may derive overwhelmingly from adult circumstances, coronary heart disease and respiratory disease from both childhood and adult circumstances, while stroke and stomach cancer appear to be associated to an unusual extent, and independently of any continuity of social disadvantage throughout life, with adverse social circumstances during childhood [24]. In a further example, using retrospective data collected by life-grid, doubt was cast on the hypothesis that women's higher prevalence of osteoarthritis of the knee joint could be due to prolonged wearing of high heeled shoes [47]. Other life course analyses have investigated cause-specific mortality [48], diabetes [49,50], metabolic syndrome [51], obesity [52,53], cognitive function [54,55] and atherosclerosis [56–58].

A third advantage of the accumulation life course model is its contribution to two debates within the study of social inequalities in health. It had long been considered possible that the broadly similar social class differences in mortality risk from a wide range of different causes of death could be due to a general susceptibility to premature death [59]. The life course accumulation model suggests an alternative explanation of this phenomenon; namely, that a person's position in the social structure determines their mortality risk, via the balance of advantage and disadvantage inherent in their social location, while the specifics of that person's life trajectory, in terms of the types of disadvantage it clusters and accumulates, determines their specific cause of death [32]. The second debate within health inequalities research concerned the contribution of social mobility to social class differences in health; with many researchers arguing that health inequalities are at least partly due to the upward social mobility of healthy people and the downward mobility of sick people [60,61]. In contrast, the life course accumulation model implies that a person's level of health tends to be a function of the proportion of their life course spent exposed to disadvantage. As a result, the upwardly mobile may be healthier than those they leave behind in their class of origin, but they tend to be less healthy than those they join in their class of destination; and vice versa for the downwardly mobile. The net effect of such health-related social mobility is to constrain, rather than amplify or create, social inequalities in health [62,63].

The final advantage of the accumulation life course model is its social policy implications. Welfare states traditionally are designed to provide a safety net, which will sustain those exposed to misfortune while they regain their earlier more advantaged circumstances; for example, unemployment benefit for those made redundant. The accumulation life course model, in contrast, draws attention to the strong probability that any one episode of misfortune, say redundancy, will have been preceded by other misfortunes; and that what is required is less a safety net and more a springboard, to both sustain presently and repair the effects of previous damage [30]. The *Foyer* scheme for young unemployed men is an example of the springboard approach. In addition to welfare payments for subsistence, the *Foyer* scheme offered remedial education in numeracy and literacy (because the young unemployed often had failed formal education), accommodation (because the young unemployed often came from disturbed and disrupted families) and access to a counsellor or adviser (because the young unemployed sometimes lacked a stable relationship with a mature adult). The accumulative nature of this process means that such springboard interventions are always worthwhile, whatever the person's age [42].

5. Limits of the life course approach

The West of Scotland Collaborative Study found that adult cardiovascular disease risk factors related in different ways to the life course. The behavioural risk factors (tobacco smoking, recreational physical exercise) were associated primarily with adult socioeconomic circumstances, while the physiological risk factors (serum cholesterol, blood pressure, body mass index, lung function) were associated to varying extents with

socio-economic circumstances in both childhood and adulthood; BMI was associated particularly with childhood circumstances [64]. These findings were supported broadly by analyses of Whitehall II study data [65], leading to the generalisation that behaviour is influenced primarily by current circumstances, while physiology is influenced by the whole life course. Subsequent work has elaborated this generalisation. In the full Boyd Orr cohort the consumption of a healthy diet in early old age was influenced primarily by current circumstances, although weak influences from earlier in life could be identified both qualitatively [66] and quantitatively, with for example high vegetable consumption during childhood predicting a healthy diet some 50 years later [67]. The relationship to the life course appears to be attenuated further in the case of quality of life. Although some influences from adulthood were identified in the Boyd Orr lifegrid sub-sample [68], quality of life in early old age was influenced primarily by circumstances current in early old age [69]. Similarly, in the same dataset, resilience in early old age, defined as flourishing despite adversity, was not related to life course factors [70]. Plausibly, life course influences are strongest where they can be objectified physiologically, in the form of health and disease; of intermediate influence where they shape preferences and taste; and of smallest direct influence on psychological reactions to everyday life.

Just as there are many aspects of existence where the life course approach has little relevance, so there are areas of health where its explanatory power is not required. An interest in the life course approach to health does not imply belief that the short-term and cross-sectional are unimportant. Nothing has demonstrated more dramatically the importance of the latter than the acute shortening of life expectancy that followed the end of the Union of Soviet Socialist Republics in 1989 [71,72]. The occurrence of this massive, acute change in population health is indisputable, although its causes remain unclear. What is certain is that a life course perspective is not required to explain it, although life course factors may help explain why the health impact of this social disruption was greater on some individuals than on others; why single men were most affected, for example, and the role of alcohol consumption [73–75].

A third possible limitation at present is speculative. Data from Norwegian linked registers followed some 19,000 people who were aged around 70 years in 1990 back in time to 1960, when they were aged around 40 years; and forward in time to all deaths among these individuals during 1990–1998. Most of the variation in death risk between the more advantaged and the more disadvantaged social groups was explained by the person's social position in 1990, with little evidence of an important cumulative effect across 1960–1980 [76]. Similar evidence, in terms of physiological and self-reported clinical status at ages 65–75 years, comes from the Boyd Orr lifegrid sub-sample where a life course cumulative effect is found for some, but not all, of the measured dimensions of health [77]. Also, early life socio-economic circumstances were unrelated to blood pressure [78] and obesity [79] among Spaniards aged 60 years and older. Speculatively, it is possible that life course effects become less important at older ages, where rates of mortality and morbidity are highest (if confirmed, reconciling this with accumulation will be an interesting challenge).

6. Future issues

It seems to me that there are two main issues for the future. First is the need to move the focus of life course epidemiology to the high mortality, high morbidity age groups. One possibility is to wait until the birth cohort studies mature into these ages; or to make do with the limited data available from linked register datasets. An alternative, and I think increasingly attractive option, is to resuscitate long forgotten studies, trace the study subjects to their present locations and interview them to collect, among other things, retrospective life course information. Central to this strategy is replacing the old blanket suspicion of retrospective data with research into the questions: Which items of information are recalled with greatest accuracy over what period of time? And which methods of retrospective data collection maximise accuracy and duration of recall? A few studies have started to investigate these issues [28,80,81], but much more work is required.

Second, the two partners in social epidemiology (epidemiology and social science) need to take more seriously the intellectual traditions of their partners. Social scientists need to pay more attention to epidemiology; and epidemiologists need to pay more attention to social science. Many social scientists, for example, run life course analyses with self-assessed health as the outcome variable. Given that nobody knows the proportions in which mental and physical health influence self-assessed health, nor whether the measure responds primarily to past experience, present circumstances or future expectations, it is not surprising that these life course analyses sometimes lack biological plausibility (What is the aetiology of self-assessed health?). Of equal importance, medical epidemiologists run analyses that ignore social science. Consider three examples:

- ignoring the complexity of the social structure, by confusingly using the same term *socio-economic status* to refer to three separate dimensions of social position, namely: social class or employment relations; the material conditions of life; social status or prestige;
- failing to recognise that historical social change may involve the transition from one type of socio-economic system to another; for example, failing to recognise that urbanisation and the disappearance of the peasant layer of small farmers cannot be incorporated easily into life course analyses of the health status of older people in the recently urbanised societies of Europe;
- lagging behind advanced social statistical methods which address such problems as the temporal sequencing of events, missing data in longitudinal studies and multilevel, geographically clustered data.

Finally, and I believe crucially for life course epidemiology, is international collaboration and comparison. In this respect,

Europe is taking important steps forward. Among the most important are the Rotterdam studies, led by Anton Kunst and Johan Mackenbach, of social inequalities in health in different European countries; the Study of Health, Ageing and Retirement in Europe (SHARE), led by Axel Börsch-Supan from Mannheim and Johannes Siegrist from Duesseldorf; the European Social Survey led by Roger Jowell from London; and the comparisons of GAZEL and Whitehall II led by Marcel Goldberg and Archana Sing-Manoux from Paris. The spirit of such initiatives needs to be taken into the field of life course epidemiology; in this respect, Marjo-Riitta Jarvelin and Michael Wadsworth from London (and Oulu) are pioneers. These comparative studies lead inevitably to questions about which differences between the countries of Europe are most important for the topic under investigation. Here Gosta Esping-Andersen [82,83] is a great pioneer.

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