GLOSSARY

Life course epidemiology

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The aim of this glossary is to encourage a dialogue that will advance the life course perspective.

A life course approach offers an interdisciplinary framework for guiding research on health, human development and aging. Psychologists,[1] sociologists,[2 3] demographers,[4] anthropologists,[5] and biologists[6] have actively promoted such an approach for many years. The interdisciplinary research area of developmental science,[7 8] also brings together psychological, cognitive, and biological research on developmental processes from conception to death. Epidemiologists have been more recent converts to a life course approach.[9–15]

We have previously defined life course epidemiology as the study of long term effects on later health or disease risk of physical or social exposures during gestation, childhood, adolescence, young adulthood and later adult life.[9 15 16] The aim is to elucidate biological, behavioural, and psychosocial processes that operate across an individual's life course, or across generations, to influence the development of disease risk. The catalyst for a life course approach in epidemiology stemmed from the revival of interest in the role of early life factors in cardiovascular and other chronic diseases,[17] in particular the ecological and historical cohort studies used to explore the fetal origins hypothesis.[18] According to this hypothesis, environmental exposures such as undernutrition during critical periods of growth and development in utero may have long term effects on adult chronic disease risk by ''programming'' the structure or function of organs, tissues, or body systems. This idea of ''biological programming'' was presented as an alternative paradigm to the adult lifestyle model of adult chronic disease that focuses on how adult behaviours (notably smoking, diet, exercise and alcohol consumption) affect the onset and progression of diseases in adulthood.[19 20]

To counteract the increasing polarisation of biological programming in utero and adult lifestyle approaches to chronic disease aetiology, life course epidemiology was built on the premise that various biological and social factors throughout life independently, cumulatively and interactively influence health and disease in adult life.[9] A life course approach does not deny the importance of conventional risk factors, such as smoking and hypertension, which were so successfully identified by the early post-war adult cohort studies. Rather its purpose is to study the contribution of early life factors jointly with these later life factors to identify risk and protective processes across the life course. So far, life course epidemiology has paid particular attention to the long term effects of childhood and adolescent risk factors on later disease. This is partly a response to the emphasis on adult factors in most post-war aetiological models of chronic disease. This is in contrast with the focus of life span developmental psychology on adult human development to counter the dominance of child centred developmental psychology.[1]

Life course epidemiology attempts to integrate biological and social risk processes rather than draw false dichotomies between them. The interests of life course epidemiology overlap with social epidemiology, that branch of epidemiology that studies the role of social factors in the production of health and disease in populations.[21] Life course epidemiology studies how socially patterned exposures during childhood, adolescence, and early adult life influence adult disease risk and socioeconomic position, and hence may account for social inequalities in adult health and mortality. Socioeconomic factors at different life stages may operate either via *social chains of risk* or by influencing exposures to causal factors at earlier life stages that form part of long term *biological or psychological chains of risk*. Differential health effects according to the timing or duration of exposure to socioeconomic circumstances may provide important clues to aetiology.[22–24] Life course epidemiology also provides a fresh perspective on explanations for secular disease trends[10] and for gender,[25] ethnic,[26] and geographical[12] inequalities in health.

Life course epidemiology implies more than a longitudinal study. The first is a theoretical model whereas the second is a study design. The purpose of life course epidemiology is to build and test theoretical models that postulate pathways linking exposures across the life course to later life health outcomes.[16] These models explicitly require the temporal ordering of exposures and their inter-relationships. The development of these models in life course epidemiology provides a persuasive rationale for time related study designs. It has led to a growing interest in existing and new longitudinal studies that capture certain time windows or other potentially significant features of the life course.[27 28] They include new birth cohorts[29 30] and the revitalisation of old historical cohorts.[18 28 31] Some guidance on the practicalities of conducting life course studies exists[32–34] and more is planned.[35]

The implementation of life course models present many methodological challenges beyond study design[16 24 36] that are also being addressed. They include the analytical problems associated with modelling repeat observations, hierarchical data, latent exposures, or multiple interactive or

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small effects. These problems are common to epidemiology and other related disciplines, but are particularly relevant for testing life course models. Multilevel models,[37] latent growth models,[38] Markov and graphical chain models[39] are among the techniques being used by epidemiologists for this purpose. The problems of missing data, omitted exposures, and measurement error may be particularly salient in longitudinal studies where observations have taken place over a prolonged time period.

Life course epidemiology borrows many of its concepts and ideas from other scientific disciplines. Some of these ideas are defined in this glossary for use by epidemiologists. As life course epidemiology is a dynamic new field, terms are often used loosely or inconsistently. Our intention is not to impose definitions but rather to encourage a dialogue among epidemiologists that will advance the life course perspective. Clarification of terms will aid the development of life course theoretical models and their operationalisation in terms of testable hypotheses, analytical strategies, and the use of appropriate statistical techniques.

There is a structure in the glossary that is hidden by the alphabetical ordering of the concepts. Most concepts fall into one of three categories. Firstly, there are the concepts referring to the causal pathway in relation to time (see accumulation, chain of risk, trajectory). Secondly, there are concepts about the timing of causal actions (see birth cohorts, critical and sensitive periods, induction and latency periods). Thirdly, there are concepts referring to different types of mechanisms (see embodiment, mediating and modifying factors, resilience, susceptibility and vulnerability).

ACCUMULATION OF RISK

One of the earliest descriptions of *accumulation of risk* was Riley's concept of insult accumulation,[40] the notion that life course exposures or insults gradually accumulate through episodes of illness and injury, adverse environmental conditions, and health damaging behaviours. One of the purposes of life course epidemiology is to test the extent of cumulative damage to biological systems as the number, duration or severity of exposures increase, and as body systems age and become less able to repair damage. The accumulation of different types of exposures (such as environmental, socioeconomic, and behavioural) may cause long term damage[41] with exposure risk being either independent (fig 1, model (a)) or clustered (fig 1, model (b)). The latter is known as an *accumulation model with risk clustering*.[16] Life course epidemiology shares with social epidemiology a particular interest in exposures that cluster because they are often related to an individual's or family's socioeconomic position in society.

BIRTH COHORT EFFECTS

Birth cohort refers to the location of an individual in historical *time* as indexed by their year of birth. An environmental change (such as an improvement or deterioration of living standards) that affects the health of children may show up several decades later as birth cohort differences in adult mortality.[42] Cohort differences in adult disease can also occur if there are cohort variations in risk factors such as childbearing characteristics or the adoption of habits such as smoking[43] that have long term effects on health. Birth cohorts may also be differentially affected by rapid or extensive social change.[44] The impact of such change, creating possibilities for *turning points* on life *trajectories* with short and long term effects on health, often varies by age.[2] The changing size of birth cohorts (such as the post-war "baby boom" generation) is itself one of the forces for social change. Age-cohort models identify possible cohort effects. More complex age-period-cohort models can be fitted to help distinguish these effects but require assumptions in order to estimate the parameters of the model because of the linear dependency among the three factors.[45 46] Cohort effects are easiest to distinguish when disease trends have accelerated, decelerated, or changed direction. Where they are steady and linear they cannot be reliably distinguished from period effects.[43]

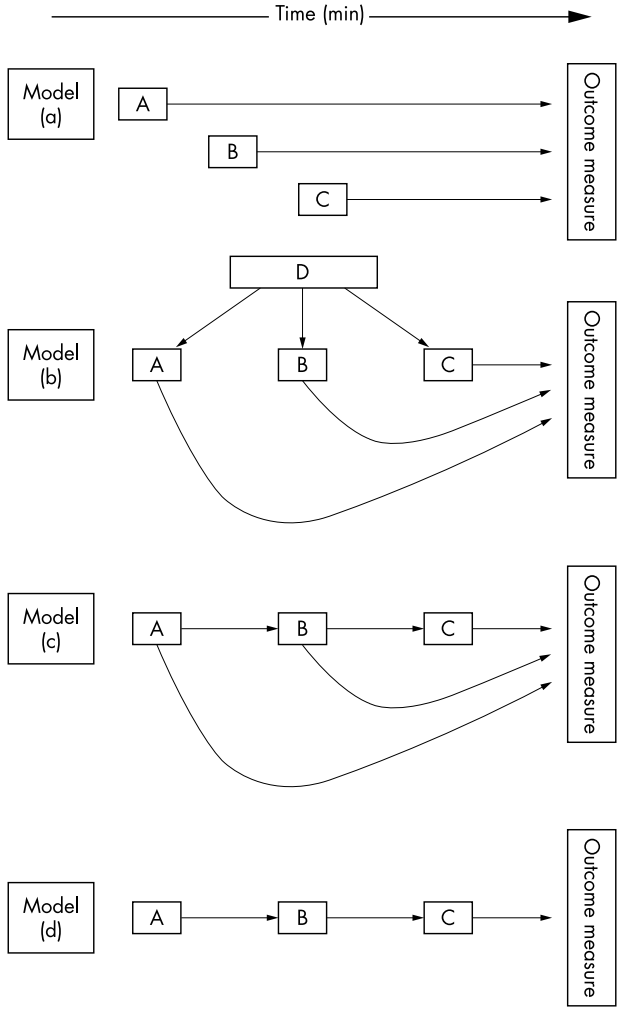

Figure 1 Life course causal models.

CHAINS OF RISK MODEL

A *chain of risk model* refers to a sequence of linked exposures that raise disease risk because one bad experience or exposure tends to lead to another and then another. Different types of chains can confer increased or decreased risk. Our definition of chains of risk (or protective chains) is based on the idea of "chain reactions" put forward by Rutter[47] to explain continuities between early experiences and adult psychosocial function. It also resembles what has been described as a "pathways model".[13 48] Social, biological, and psychological chains of risk are possible and involve "*mediating factors*" and often "*modifying factors*". The sequential links are probabilistic rather than deterministic. It is possible to conceive of two different types of chains of risk. One model is that each exposure not only increases the risk of the subsequent exposure but also has an independent effect on disease risk irrespective of the later exposure (fig 1, model c). When each adverse experience increases the risk of disease in a cumulative fashion this is called an "additive effect" and may be a special case of an accumulation model.

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Alternatively, earlier exposures have no effect on disease risk without the final link in the chain that precipitates disease onset (fig 1, model (d)). Such a ''trigger effect'' describes the situation when it is only the final link in the chain that has a marked effect on disease risk.

CONTEXT

Context refers to the location of an individual by *time* and place. Place refers to both geographical location and to group membership in terms of family, friends or age, and on the basis of class, ethnicity, residence, and gender that arise out of the social and economic structure of society. Context may affect exposure to risk and the individual's response strategies. Application of a life course perspective to contextual as well as individual effects on health implies understanding the effects of changing contexts over time on the life course of individuals.

CRITICAL PERIOD

In the natural sciences a *critical period* of development refers to a time window when intrinsic changes in the organisation of living systems or sub-systems towards increasing complexity, greater adaptivity and more efficient functioning occurs rapidly and may be most easily modified in a favourable or unfavourable direction.[49] In life course epidemiology the relevance of changes during a critical period is in respect of their long term effects on disease risk many years later. Thus, we define a critical period as a limited time window in which an exposure can have adverse or protective effects on development and subsequent disease outcome. Outside this developmental window there is no excess disease risk associated with exposure.

CRITICAL PERIOD MODEL

A *critical period model* of disease causation is when an exposure acting during a critical period of development has effects on the structure or function of organs, tissues or body systems that are not modified in any dramatic way by later experience, and that precipitate disease later in life. Also known as ''biological programming'' or as a ''latency model'',[13] it underlies the fetal origins of adult disease hypothesis. An expanded version of the basic critical period model includes the possibility that the effect of an exposure during a critical period of development on later disease risk may be dramatically changed by later physiological or psychological stressors. The *critical period with later effect modifiers* implies investigating plausible interactive effects between early and later life risk factors.

EMBODIMENT

Embodiment is a term common to both social epidemiology[21] and life course epidemiology. It describes how extrinsic factors experienced at different life stages are inscribed into an individual's body functions or structures. This may be through developmental processes associated with critical periods, habituation, learning, damage, and repair. The term ''biological embedding''[48 50] has a similar meaning although it tends to be applied to neurobiological or psychobiological mediators of the early social environment on child development and life course health.

INDUCTION AND LATENCY PERIODS

While different definitions of these terms appear in the epidemiological literature, we favour those used by Rothman.[51] *Induction period* is defined as the time between exposure and initiation of the disease process, hence it is a characteristic of a relation between a specific exposure and a specific disease. Life course epidemiologists have a particular interest in causal relations with long induction periods. *Latency period* refers to the period between disease initiation and detection, and is a characteristic of the disease (onset of symptoms) or the healthcare system (diagnosing the disease). If an exposure has a long induction and/or latent period, its public health importance may vary according to when in the life course one is exposed. Variations in the time between *induction* and *latency* periods make it difficult to detect the true causal agents, and the strength of association between an exposure and disease can be diluted or missed if the wrong time frame is measured. Often it is difficult to observe the time of disease initiation and this prevents researchers from distinguishing empirically between the induction and latent periods. Latency period should not be confused with latency model, which is an alternative term for a critical period model.

LIFE CYCLE/LIFE SPAN/LIFE COURSE

The concept of a *life cycle* has generally been used in other scientific disciplines to describe a series of distinct, bounded life stages which are socially or biologically determined.[52] In contrast the concept of the *life span* used in psychology assumes that development and aging form a continuous process from birth to death. The distinction between life span and life course, the term used more commonly in sociology, is mainly a matter of scientific history.

MEDIATING FACTOR

A risk or protective factor *mediates* the association between exposure and disease when it chronologically follows the exposure and is conceptualised as lying, at least partly, on the causal pathway. Life course epidemiology requires an explicit temporal theoretical model that distinguishes between mediating factors (post-exposure of interest) and confounding factors (conceptualised as prior to and/or tangential to understanding the effects of the exposure of interest).

MODIFYING FACTOR

A risk or protective factor *modifies* the association between an exposure and disease when the causal effect of the exposure of interest differs across levels of the modifying factor. Investigation of modifying factors provides information about the nature of the causal process. Effect modification is known as interaction—''synergism'' if the modifying variable enhances the effect of the explanatory variable or ''antagonism'' if it diminishes it. Interactions are thought to be common features of life course processes and should be investigated where plausible biological, behavioural or social hypotheses exist. Well defined theoretical life course models are needed to interpret the biological or social significance of observed interactions. Empirical assessment of interaction is the source of much debate in epidemiology and needs to be carefully considered in terms of additive or multiplicative risk models.[51 53]

PLASTICITY

Plasticity is the potential for change in intrinsic characteristics in response to environmental stimuli. It is measured by within person variability. Like life course perspectives in other disciplines,[54] the search for the range of plasticity and age associated changes and constraints is a fundamental quest of life course epidemiology.

RESILIENCE

Resilience is a dynamic process of positive adaptation in the face of adversity.[55] The focus of research has been on the intrinsic and extrinsic factors associated with educational, emotional and behavioural resilience of children.[55 56] There has been less focus on health outcomes and on long term outcomes in general.[57] A question that has hardly been

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addressed is whether resilience in development, and its associated factors, have long term health benefits or costs. For example, the fetal origins hypothesis assumes that adaptations for survival made by the fetus in response to adversity (maternal undernutrition) raise the risk of chronic disease risk in later life.[18] Life course epidemiology has contributions to make to an integrated science of human adaptation and development[56] that also includes an aging perspective. Both share a focus on lifelong biological, psychological, and social processes and their interrelationships.

SENSITIVE PERIOD

Like critical periods, *sensitive periods* are also times of rapid individual change but there is more scope to modify or even reverse those changes outside the time window. Thus a sensitive period in life course epidemiology is a time period when an exposure has a stronger effect on development and subsequent disease risk than it would at other times. Outside the time period any excess risk will be weaker.

SUSCEPTIBILITY

Within epidemiology *susceptibility* has been defined in terms of Rothman's ''causal pie model'',[51] which describes susceptibility as ''a term that refers to the condition of having one of two interacting causes already and therefore being susceptible to the effect of the other.'' (page 172). It is usually used where host factors increase the likelihood that another exposure will produce disease. Host factors refer to both intrinsic factors as well as prior exposures that have become embodied over time. Thus susceptibility is a process occurring over time that may eventually lead to disease via the completion of the last piece of the causal pie. This idea has been given greater salience with the emergence of genetic epidemiology and the growing attention to gene-environment interactions.[58] In the wider scientific literature susceptibility is often used synonomously with *vulnerability* and conveys a broadly similar process.

TIME

Time is a fundamental concept in life course epidemiology, both in terms of life time (as indexed by the chronological age of individuals) and historical time at the population level (as indexed by membership of a *birth cohort*). Changing individuals must be studied in a changing environment (see *context*). The effect of an exposure on a health outcome may be dependent on the duration or timing of exposure. *Critical* and *sensitive periods* are so defined because the age of the individual at the time of exposure is assumed to affect the long term outcome. In other words, they represent qualitatively different exposure-time interactions. For critical periods there is no excess risk associated with exposure outside this time window whereas for sensitive periods it is merely weaker. This is similar to Elder's ''life stage principle''.[2] Similarly, social roles and life events have different social meanings and possibly differential health effects depending on their timing and whether they are typical for a person's age, gender, or culture.

TRAJECTORY, TRANSITION AND TURNING POINTS (ADAPTED FROM ELDER[2])

A *trajectory* provides a long term view of one dimension of an individual's life over time. These may be social states (such as work, marriage, socioeconomic position), psychological states (such as depression) or physiological states (such as lung function). Implicit is the idea of a normative trajectory around which individuals deviate. *Transitions* are short-term and embedded in trajectories, marking a change in social, psychological, or physiological states. A marked change of direction is referred to as a *turning point*.

VULNERABILITY

Vulnerability is the opposite of resilience and refers to a dynamic process of negative adaptation in the face of adversity. The negative physiological or behavioural response is shaped by prior *embodiment* of extrinsic factors as well as intrinsic characteristics.

CONCLUSION

Life course epidemiology has blossomed in the past five years as a natural response to the limitations of previous aetiological models of chronic disease. We have welcomed the opportunity here and elsewhere[16] to clarify its purpose and guiding principles as misunderstandings have inevitably arisen. A life course perspective in epidemiology has been erroneously limited to notions of risk accumulation,[59 60] or associated only with psychosocial theories of social epidemiology.[21] Rather we would argue that it represents an attempted synthesis of previous models of disease causation (such as adult lifestyle, biological programming, and social causation) that is scientifically interesting, conceptually and methodologically challenging, inherently interdisciplinary and policy relevant. A life course approach has been adopted in all but name by researchers whose focus was initially restricted to fetal and infant life but who have now extended their frame of reference to include postnatal growth and development.[59 61] Childhood indicators of adult disease risk include childhood stature, particularly leg length,[62] relative weight,[63] energy intake and IQ.[64 64a] There is also more willingness among those who focus on adult risk factors to recognise the importance of early life factors.[65] In epidemiology there is a growing appreciation of the need for a temporal perspective for conventional so called ''adult'' risk factors.[66–68] Age at onset, duration and changes in levels of exposure to conventional risk factors may alter their effects on adult disease risk and impact on long term disease trends.[69]

What are the future challenges for life course epidemiology? One challenge is the continued development of testable theoretical models that elucidate the risk and protective factors at each life stage, and the underlying biological, psychological, and social pathways that link these together across one or more generations. The life course models defined in this glossary are not mutually exclusive and may operate simultaneously. It may not be easy to distinguish these models empirically[24] nor to develop standardised and acceptable methods of combining cumulative exposures.[36] These are just two of a number of complex methodological problems facing life course epidemiology. The second challenge is the ability of life course epidemiology to help explain temporal, geographical, and social patterns of disease distribution. Thirdly, changing individuals need to be studied in a changing world and we would like to see a life course approach fully integrated into the study of broadly defined contextual health effects. Fourthly, the growing genetic knowledge and development of cheap and reliable methods to analyse DNA on large population samples, has opened up possibilities for studying the interactive effects of genetic and environmental factors across the life course. Lastly, and most important of all, there is the need to translate our new knowledge into interventions and policy recommendations designed to improve the long term health of individuals, social groups, and societies.[14 70–72]

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Speaker's corner

Understanding the functional components of public health surveillance

Developing a public health surveillance system is similar to developing a housing estate. Besides the actual building process (setting up infrastructure for data collection, analysis, interpretation and dissemination) there are many other requirements to be identified in planning—and care must be taken to ensure products will stand up to the test of time.

The first step is to identify ''wants'' of homebuyers (recognise and integrate all the ''needs'' of various stakeholders in a manner that will lead to quality, efficient outcomes). Consultation with government agencies, town planners, and homeowners (relevant authorities and community groups) will ensure planning processes are appropriate to the ''authorising environment''.

It is then necessary to enlist architects (epidemiologists and public health professionals) who will conceptualise the project and describe scenarios in the form of drawings and models (show the relevance to information needs and applications to public health policy). Architects will also scope engineering, structural and costing issues and other aspects of the design that will be attractive to the client.

The stakeholder can now make serious decisions about implementation—choice of builder (data collection contractors), preferred building specifications (data collection methodology), and submission of a planning permit (data quality control plan).

A visit to an accountant and banker is now important to ensure the project is economically viable (budget plan).

Processes such as these are all necessary to ensure surveillance systems are sustainable and that core data elements remain stable over time. As buildings must be resistant to harsh environmental conditions, surveillance systems must also be resistant to changes in the political or bureaucratic environment.

Bringing in builders and technical teams is now easy—but architectural supervision will be required throughout. Use will be made of fast turnaround, automated building machinery (software) and pre-fabrication where appropriate to ensure efficient construction and fit-out of buildings (analysis and reporting).

Following a successful building project, a sales office is needed to attract homebuyers. Again, this process will be made easier if early consultation and planning has been thorough. Surveillance systems (like buildings) also require marketing. Information must continually be communicated to stakeholders in the most appropriate way.

The key message here is that architects (epidemiologists) must engage with all stakeholders to ensure their needs are met (product relevance and application) and that the quality of life of the new residents (public health outcomes) will be improved through construction of the new housing estate (health surveillance).

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LETTERS

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Social epidemiology, intra-neighbourhood correlation, and generalised estimating equations

The recent editorial by Merlo[1] offers an interesting critique of the generalised estimating equations (GEE) analysis of a paper published in the same issue of the journal. In the editorial, the author notes that the paper's GEE analysis treats ''the intra-neighbourhood correlation as a 'nuisance' that needs to be adjusted in the analysis but not explicity investigated'' (page 550).

The editorial then becomes a call for an alternative, more innovative approach in social epidemiology: ''Estimation of the extent to which individuals within a given neighbourhood are correlated with one another in relation to health (that is, the concept of intra-neighbourhood correlation) has value in the context of ideas about the efficacy of focusing intervention on places instead of people'' (page 551). The finale is a logical conclusion that studies of intra-neighbourhood correlation may ''...present themselves as a new epidemiological approach that may prove very useful in social epidemiology'' (page 551).

The author is appartently speaking of first order GEEs but the *JECH* readership may not appreciate that second order GEEs (GEE2) treat the intra-neighbourhood and inter-neighbourhood correlations into deliberate objects of study and estimation.[2] Although we ourselves deserve absolutely no credit for biostatistical innovations, the ''alternating logistic regressions'' (ALR) approach[3] we use in our forthcoming article in this journal[4] is a computationally efficient alternative to GEE2 in the case of a binary outcome. As such, it estimates the pairwise odds ratio, which quantifies the degree to which health conditions, behaviours, or perceptions might cluster within neighbourhoods (or other nested structures of community life) to a degree other than one might expect if these health conditions, behaviours, or perceptions were distributed at random across neighbourhoods.

Because we believe our work is responsive to the author's call for a new approach in social epidemiology that measures intra-neighbourhood correlation, we would welcome an editorial comment on the potential value (and possible shortcomings) of the GEE/ALR approach we used in our forthcoming article in *JECH* on clustering of cocaine incidence in the United States. We hope you will concur that our application of the ALR approach is a step in the right direction for research on contextual influences and health.

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Author's reply

I have read with great interest the comments made by Petronis and Anthony on my editorial.[1] I have also read their forthcoming article,[2] and I believe they apply an analytical approach that seems to be, in my opinion, a step in the right direction for research on contextual influences and health that focus on investigation of clustering. I will be very pleased to write a more extensive comment and send it for consideration and possible publication in the journal.

Measuring clustering with the aim of obtaining substantive scientific information is so far an uncommon approach in social epidemiology and most multilevel analyses have in fact been plain ''contextual analysis''[3] focused on measures of association. This seems to be true not only for studies using GGE techniques but also for studies using multilevel hierarchical regression. However, the original standpoint of multilevel hierarchical regression analyses is the investigation of complex patterns of variation[4] rather than dealing with residual correlation.

Regarding the use of ''GEE2'' and measurement of clustering, the comment of Petronis and Anthony is certainly right.

From an epidemiological point of view the most interesting question is the conceptual rather than the mathematical approach used. I agree with Petronis and Anthony in their conceptual approach and I believe that they put context back in epidemiology using ''GEE2'' techniques.[5] The pairwise odds ratio and other techniques for measuring neighbourhood heterogeneity and clustering like the median odds ratio and the interval odds ratio[6 7] deserve more development and spreading.

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CORRECTION

An editorial error occurred in this article by Kuh and colleagues (2003;**57**:778–83). In figure 1 the top of the figure should read Time [not Time (min)].