Letters to the Editor

Childhood leukaemia and socioeconomic status
From MARILYN J BORUGIAN,1,2* JOHN J SPINELLI1,2 GABOR MEZEI,3 RUSSELL WILKINS4 and MARY MCBRIDE1,2

In a recent issue of the journal, we were pleased to see continued interest by Smith and colleagues1 in the relationship between socioeconomic status (SES) and the development of childhood leukaemia. We agree with the authors that the completeness and representativeness of their data as well as the ability to measure deprivation in several ways are strengths of their study.

We would like to point out to the authors that although on the surface the methodology of the UK investigation was different from our recent registry-based Canadian study,2 in fact, the studies were similar. The major difference was that our study did not include enhanced surveillance to identify the childhood leukaemia cases. Due to the centralized cancer registration and treatment system in Canada, however, it is likely that our registries do not suffer from the underreporting problems which seem to have been present in the UK cancer registration.3,4 Therefore, it is likely that both studies captured most, if not all, childhood leukaemia cases. The other differences were minor. We used the entire population as our comparison group rather than selecting a subset of the population as a control group, and we included a longer time period (1985–2001 for most provinces) in the analysis. Finally, our study was unable to look at SES at different time points.

In fact, the results of two studies gave very similar results for risk associated with SES at diagnosis/reference date. For example, based on 4024 cases of acute lymphoblastic leukaemia, comparing the poorest of five categories with the richest, we found a rate ratio (RR) of 0.86 [95% confidence interval (CI) 0.78–0.95] whereas based on 1578 cases the UK study found an odds ratio (OR) of 0.90 (95% CI 0.75–1.07). Due to the larger sample size of our study, we concluded that SES was significantly associated with the risk of childhood leukaemia. Had the UK study examined cases over a longer time period, it is quite possible that they would have also found a significant association. It is clear that continued discussion of this relationship is worthwhile, since SES may very well be an important factor in childhood leukaemia risk.

References
doi:10.1093/ije/dym061
Advance Access publication 31 July 2007

One mechanism underlying contrasting health-economy findings
From I A JACOBS,1,* M T PODOBNY2,3 and D BILUSICH4

Tapia Granados1 recently presented health-economy findings, which in the short-term, identify the decline in mortality to slow or even temporary reverse during economic upturns. These counterintuitive ‘ecological’ results initiated a lively debate in the International Journal of Epidemiology2–6 as historically epidemiological results have shown adverse health outcomes among the unemployed.7,8 In response to Tapia Granados’s1 short-term pro-cyclical health-economy oscillations, authors have suggested that such results represent: an ecological fallacy;2 may possibly represent reality;5 reveal
nothing new—as in the long-term, mortality fluctuates in a counter-cyclical health-economy fashion. A minority of commentators have also suggested that such macro-level findings need not necessarily be inconsistent with individual level research identifying worse health among the unemployed.

We contribute to this health-economic literature by highlighting that the concept of allostatic load may be one biological mechanism which gives rise to adverse health outcomes in both the unemployed and employed. (An anonymous referee suggested work by Sterling and Eyer on employment and stress to contribute to this concept. However, if one is concerned about making medical discoveries ‘first’, our research identifies Jethro[Exodus 18: 13–23] as the earliest pioneer to identify overwork as detrimental to health.)

We thereby extend upon the notion that the theories associated with pro-cyclical (short-term) and counter-cyclical (often long-term) health-economy findings need not necessarily be inconsistent with each other.

More than two decades ago, it was noticed that biological changes occur among the unemployed which resemble allostatic load development. Olafsson and Svensson summarize a number of these study results:

in various follow-up studies on an individual level the loss of job or the mere prospect of becoming jobless, have been found to cause elevated blood pressure, serum cholesterol and uric acid, elevation of blood concentration of catecholamins and... increased stress, psychosomatic disease and increased elimination of noradrenaline has been found to persist up to 2 years after the loss of a job (p. 1107).

While such physiological changes may occur among the unemployed, recent findings also identify allostatic load to be associated with those in employment. In a prospective study (1973–2000) on Finish industrial workers, Kivimaki and colleagues found those employees whom seldom recovered from work—after controlling for age, sex and 16 risk factors—had an elevated risk of cardiovascular disease and increased elimination of noradrenaline.

As socioeconomic status (SES) is a prominent health issue in all developed countries, allostatic load development has also importantly been associated with SES in a graded fashion which may begin in childhood. These, other such findings and environmental conditions (such as air pollution) associated with SES point to a single underlying mechanism operating for the employed and unemployed.

The LiVicordia studies and similar future research may further advance our knowledge of how environmental factors, particularly employment and unemployment conditions, impact underlying biological processes that in turn contribute to adverse health outcomes for communities and individuals.

In this regard, the contrasting (pro-cyclical and counter-cyclical) health-economy findings need not only be associated with each other, but may also result due to one underlying mechanism.

Conflict of interest: None declared.

References


doi:10.1093/ije/dym123
Advance Access publication 12 June 2007

Metaphorical measurements and theories
From GIO BATTA GORI

A recent letter of mine asked what consideration should be given to individual recalls of lifetime exposures and experiences: a question that is central to the relevance of most modern epidemiology.1 In their reply, Drs. Fox, Lash and Greenland mistook the question and addressed the different issue of exposure misclassification and other binary biases.2 Rather, the raised problem focuses on the quantitative measurement of the primary variables tested, and on the resulting continuous data such as: how much was each individual exposed over a lifetime to specific dietary items, to occupational and environmental toxicants, to medicines, to second-hand tobacco smoke, and the like. The bulk of these data is obtained from personal recalls of individual study participants or from recall proxies from next of kin of deceased subjects.

Epidemiologists traditionally interpret such recalls as ‘measurements’. Yet, by any factual standards—scientific or otherwise—this qualification is not sustainable, because the sine qua non of statistical elaborations is that discrete characteristics of individuals must have been physically measured using the same meter, that the measurement error is known from prior testable experience, or that it has been determined in the study at hand by multiple measurements of each characteristic, on a sufficient number of subjects.

The interpretation of recalls as ‘measurements’ is grounded on the illusory assumption that individual recalls represent the