

Social epidemiology. A way?

(reply to *Social epidemiology? No way*)

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Certainly let us share Zielhuis and Kiemeney's exasperation¹ at the proliferation of disciplinary branches. There is no merit in devising particular insular subdisciplines; the world out there after all is what we are trying to explain, and the world comes with no guarantee that it respects (or even notices) these boundaries. (And these boundaries fluctuate by language, as Mielck and Bloomfield² report.) The task is to understand the genesis and maintenance of illness and disease, using whatever tools best work, with some hope (and perhaps indeed constraint) that such understanding maps to routes to intervention.

But that task surely entails some hesitations over Zielhuis and Kiemeney's substantive belief: 'that shopping in the neighbouring scientific fields, without *thorough* subject-matter knowledge, will lead to statistical results without relevant meaning.' Of course if I wish to assess the impact of early social factors upon subsequent health (an important social policy issue) I need to know that it is medically plausible that the development of the human foetus may be affected by maternal nutrition, medically implausible that astrological influences are active. It may even help me to be aware that there is some evidence suggesting that babies born small through deprivation in the womb may become 'biologically thrifty' and if environmental circumstances improve, and there is more food about, biologically thrifty individuals may be more prone than others to the adverse consequences of obesity. These assumptions structure the variables and models considered, and (as in the 'thrift' case) may lead to evaluation of interactions which 'common sense' might have ignored. But this is very far from *thorough* subject matter knowledge. Allow me now the supposition that maternal glucocorticoids can reprogram the foetal hormone system, that transmission is prevented by the placental enzyme *11 β hydroxysteroid dehydrogenase* (11b-HSD), add to this the observation that disadvantaged rats (small and suffering from high blood pressure) can be shown to have sluggish 11b-HSD activity ... so suggesting a possible theory for foetal 'memory'. Zielhuis and Kiemeney would presumably have it that: 'All epidemiological hypotheses are ... derived from such theories by deductive reasoning.' But surely this is nonsense. Of course resolution of the mechanism is important, and could

have real consequences – knowing it we might, for example, be able more cheaply to intervene in the consequential than in the generative mechanisms of malnutrition (and this might have implications for developing, as well as developed, economies). Admittedly, not having ‘thorough subject matter knowledge’ I have no idea whether the 11b-HSD hypothesis is even in the right ball-park. *But* – and this is what matters for the present dispute – resolution of the mechanism at this level is *not* a prior condition of avoiding nonsense and writing sense in the social policy question with which I started.

It is very easy to write nonsense about social processes (glance at the collection that passes for ‘sociology’ in any university bookshop). It is sadly – still – disconcertingly easy to generate nonsenses through the uncritical application of now well-documented multivariate procedures. To educate out of these nonsenses is sufficient skill for an academic lifetime. To expect thorough medical knowledge as well is implausible and unnecessary.

Of course there are apparent counterexamples. A classic tale is Stott’s study³ in 1958 showing that, in the terminology of the time, mothers of mongol children reported that they had more shocks during pregnancy than had mothers with normal children. He concluded that socio-economic factors generating such shocks were important. Some (now elementary) knowledge of chromosomal abnormalities readily shows Stott to be mistaken. But Stott is to be faulted not because he did not have the requisite (though then limited) medical knowledge, but because his social science was bad social science,. He should have realised that mothers confronted by a ‘bad’ outcome would have constructed retrospective narratives to explain to themselves the event.

So we need good social research. And we need cautious data analysis. And these are not simply other routes to laboratory medical knowledge. Indeed at times the apparent state of knowledge can mislead. Take a study of depression, whose findings are by now canonised in the secondary literature as truths. Brown and Harris⁴, having carefully ascertained that severe events were associated with depression, went on to explore the possibility of an intervening variable which acted between events and depression, to protect some women. For their ‘vulnerability’ variable they present apparently persuasive evidence (see below) of its effect: we are shown that of those low on vulnerability who experience a severe event 10% became depressed; for the medium group 21% became depressed; the highly vulnerable attain a striking 79% figure for the onset of depression following a severe event. The authors (who do indeed have *thorough*

subject-matter knowledge) treat this as support for a well established cognitive theory of depression, and tie ‘vulnerability’, as an intervening variable, to the absence of ‘self esteem’. But here a ready match to what was presumed known led to over interpretation of the findings. Inspection of the panels of the table shows that the odds-ratios underlying these disparate percentages are very similar. Indeed the full table (of Events by Depression by Vulnerability by Joblessness) can, be fitted ($p=.17$) by a loglinear model with no three-way interaction terms (using a first-letter shorthand, the model is: [ED][VD][VE][JD]). The fitted values from this two-way model are very close to the figures Brown and Harris read as evidence for interaction. So the data are fully compatible with a much simpler model of cumulative additive effects, with no interaction, and *a fortiori* no vulnerability tied to self-esteem, despite the study’s having entered the secondary literature as demonstrating the salience of self-esteem.

The point is that it is difficult to be alert and sceptical when analysing data. Expectations can lead researchers too readily to accept – and present as ineluctable – a particular model when the data fit their presuppositions, without enquiring whether alternate narratives are equally compatible⁵. It is also difficult to intelligently analyse social behaviour and deploy appropriate concepts (indeed – though this is a tale for another day – one could query the labelling of the observed construct as ‘vulnerability’, and also wonder why Brown and Harris moved from using the measurable term ‘self confidence’ – the womens’ own vocabulary – to the unmeasurable ‘self esteem’). Perhaps because of the relative development of the two subtending disciplines, it is easier to talk nonsense about ‘class’ than about ‘nutrition’. Sociological concepts are not, at present, very well defined; to avoid nonsense about ‘class’ requires considerable sociological knowledge. Which is why I see room for well trained, intelligent, quantitative sociologists to contribute to epidemiological discussion. Some medical literacy would be an advantage, but the case for requiring ‘a thorough training in ... biomedicine’ is unproven.

So we should support Zielhuis and Kiemeneney’s campaign to get rid of the term ‘social epidemiology’, but from a precisely contrary motive. Let us encourage each other to go ‘shopping in ... neighbouring scientific fields’. That is, after all, how many advances in science were made. To say that is not to say that all that passes for social epidemiology makes sense (on this see Lynch, Due, Muntaner and Davey Smith⁶). Nor that it can answer all problems (and our perception of those which it can profitably address will fluctuate as our knowledge expands – as we know which of our posited mechanisms, social or

medical, are favoured by the world-out-there). But to say 'No way' is to pretend to more ignorance than we collectively possess.

Table

Vulnerability: Low	Depression		N
	Yes	No	
Event	10	90	88
No Event	1	99	193

Vulnerability: Mid	Depression		N
	Yes	No	
Event	21	79	62
No Event	4	96	53

Vulnerability: High	Depression		N
	Yes	No	
Event	79	21	14
No Event	0	100	9

Table 1: Percentage of women in Camberwell who suffered onset of depression in the year by whether they had a severe event and by vulnerability (collapsed from Brown and Harris⁴ Ch 11; Table 3). 'Vulnerability' defined: 'low' = 'having close, intimate and confiding relationship with husband or boyfriend'; 'high' = (not 'low') and (('early loss of mother') or ('3+ children under 14 at home')); 'mid' = (not 'high') and (not 'low').

References

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