Commentary: Society, biology and the logic of social epidemiology

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Perhaps one useful response to the thesis advanced by Zielhuis and Kiemeney, summed up in the article’s title, ‘Social epidemiology? No way’.¹ can be provided by four evidence-based linked logical arguments.²⁻¹⁷ These are:

Argument #1
Thesis 1: People are social beings who live in socially-constituted societies.
Thesis 2: People are biological organisms, Homo sapiens.
Deduction: People live in the world simultaneously as social and biological beings.

Argument #2
Thesis 1: Expression of biological traits depends on the conditions under which biological organisms live, including their interactions with other organisms.
Thesis 2: Disease, disability, death, and health are states of being involved expression of biological traits.
Deduction: Disease, disability, death, and health are embodied expressions of conditions under which organisms live.

Argument #3
Thesis 1: One component of explaining a phenomenon is HOW it occurs.
Thesis 2: One component of explaining a phenomenon is WHY it occurs.
Deduction: Explanations of phenomena that address HOW and WHY they occur are more complete than explanations addressing only HOW they occur.

Argument #4
Thesis 1: Epidemiology is the study of population distributions of disease, disability, death, and health and their determinants and deterrents, across time and space.

Taken together, these arguments imply that epidemiologically adequate explanations of current and changing distributions of population health entail simultaneous social and biological explanations.¹⁸⁻¹⁹ Thus, training in—and application of—social and biological theories and reasoning are important for epidemiologists, as is development of an historical perspective on changing epidemiological profiles across diverse societies.²⁻¹⁷ Also relevant is training in concepts and methods of quantitative population sciences, including but not limited to biostatistics.²⁻¹⁷ Training in or comprehension of solely biomedical sciences is insufficient. The study of biological phenomena is broader than ‘biomedicine’: other non-medical aspects of biological sciences are highly relevant, including constructs and content of evolutionary biology and ecology.⁵⁻⁷

Consider, for example, the challenge of explaining excess hypertension among African Americans.²⁴⁻²⁵ Despite advances in population genetics, which emphasize the non-biological and social basis of ‘racial’ categories as well as the tremendous ‘mixing’ of African, European, and American Indian lineages (by both rape and consensual unions),²⁶⁻²⁷ biomedical literature remains rife with studies attempting to discern whether ‘race’—as an alleged biological trait—explains US black/white disparities in hypertension (not to mention other health outcomes).²⁵⁻²⁷⁻²⁹ Hidden from view are other relevant and promising hypotheses, especially regarding the role of racial discrimination in the aetiology of hypertension and other conditions related to chronic exposure to threats to mental and somatic well-being.³⁰⁻³⁵ As the small but burgeoning literature on social inequalities in health

reveals, ignoring social determinants of social disparities in health precludes adequate explanations for actual and changing population burdens of disease and death, thereby hampering efforts for prevention.\textsuperscript{13,36,37}

Consider, too, the phenomenon of parity in relation to risk of cancer. The focus of epidemiological research informed solely by biomedical assumptions is on links between parity and risk of cancer via pathways related to pregnancy-induced changes in diverse hormone levels.\textsuperscript{38,39} Add, however, the question of whether parity reflects social factors that are also determinants of risk of cancer, investigate links between parity and risk of cancer among men, and new knowledge emerges.\textsuperscript{38} Notably, parity is equally associated, among women and men, with risk of oral and pharyngeal cancer and malignant melanoma—for which the parity/risk association had been previously interpreted in other terms. One implication is not to presume parity exerts effects solely by pregnancy-related biological processes; the other is to consider the social meaning of parity even when the biology of pregnancy is relevant. Simplistic divisions of the social and biological will not suffice. Study adrenal glands only among cadavers of the poor, long since hypertrophied due to excess excretion of cortisol, and—as occurred in the early 20th century—the wealthy will be diagnosed with adrenal deficiency disorders.\textsuperscript{40}

In summary, if Zielhuis and Kiemenej choose to conduct epidemiological work premised solely upon ‘biomedical theory’, and to castigate ‘social epidemiology’ for producing ‘trivial statements, useless for society’, that is their prerogative. It would, however, be interesting to know what texts Zielhuis and Kiemenej would recommend to aspiring epidemiologists for the study of ‘biomedical theory’. As any systematic search of library databases will show, none exist, at least by this name, even as medical textbooks abound. Instead, tenets of ‘biomedicine’ typically have been named and analysed by its critics who, while generally appreciating the knowledge gained by study of biological phenomena, nevertheless highlight the ignorance produced and knowledge forsaken by restricting inquiry to questions generated by individualistic frameworks which deliberately ignore social determinants of health, disease, and well-being.\textsuperscript{41–44} If logic and an open mind are prized features of scientific inquiry, the illogic and narrowness of Zielhuis and Kiemenej’s argument are surely counterproductive to the further development of the enlightening and useful epidemiological research that we all desire.

References

Certainly let us share Zielhuis and Kiemeney’s exasperation 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. (These boundaries fluctuate by language, as Mielck and Bloomfield 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 routes to intervention.

However, that task surely entails some hesitations over Zielhuis and Kiemeney’s substantive belief: ‘that shopping in 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 fetus 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 fetal hormone system, that transmission is prevented by the placental enzyme 11β-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 fetal ‘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 mechanism at this level is not a prior condition of avoiding nonsense and writing sense on 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 expect thorough medical knowledge as well is implausible and unnecessary. Of course there are apparent counterexamples. A classic tale is Stott’s study in 1958 showing that, in the terminology of Krieger N, Sidney, S. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. Am J Public Health 1996;86:1570–78.


