

Social epidemiology? No way

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Although research into social and behavioural determinants of health and illness was conducted throughout the 20th century, it has become more common since the 1980s. Because of this, the term 'social epidemiology' is increasingly encountered in the biomedical literature.

Recent commentaries by Kaufman and Cooper^{1,2} and Muntaner³ on causal explanations in social epidemiology, although of interest to the research field, take the term 'social epidemiology' for granted. With reference to Syme⁴ and Susser *et al.*,⁵ the authors claim that social epidemiology is 'a distinctly recognised specialisation within epidemiology', and has been for some 30 to 40 years.

This field of research has been defined as the science which studies the link between social environment and the development and distribution of disease in populations,^{1,6,7} a definition which emphasizes a hybrid area of interest between sociology and epidemiology. Apparently, the term social epidemiology is so established that it is even used to describe the frequency of behaviour, without any reference to disease.^{8,9}

Epidemiology has evolved into many different fields of application, leading to specialities such as occupational epidemiology, cancer epidemiology, and pharmaco-epidemiology. Can 'social epidemiology' be regarded as just another branch? No, we believe the term is a misnomer.

Epidemiology is part of medical science and rests on a human-biological (scientific) background. Biomedical theory about, e.g. carcinogenesis, atherosclerosis, and teratogenesis is the input for scientific discussion and empirical studies in epidemiology. All epidemiological hypotheses are, or at least should be, derived from such theories by deductive reasoning. Similarly, results of epidemiological studies are used to adapt these biomedical theories by inductive reasoning. Therefore, every epidemiologist should have a basic training in biomedicine, comparable to the training that medical practitioners need as a basis for medical practice.

In broader terms: epidemiology, like every other discipline derived from medicine, rests on a human-biological (scientific) background.

Frequency research (the most succinct characterization of epidemiology) is also an important approach in other scientific fields, including psychology and sociology. Unlike epidemiology however, empirical studies to assess the (determinants of) frequency of psychological problems rely predominantly on socio-psychological theories. Should we call this 'psycho-epidemiology' or 'social epidemiology'? An argument in favour of using the term epidemiology outside the field of biomedicine is to stress

the similarities in methodology: the statistical approach. But epidemiology is not just defined by its statistical outlook, nor is sociology or psychology. Epidemiology deals with medical knowledge itself. Similarly, sociology and psychology deal primarily with the subject-matter of behaviour (including behaviour related to health and disease) of societies and individuals.

An epidemiologist confronted with a research problem from the field of sociology or psychology is merely a technician who knows how to handle statistics with only a layman's ideas on the topics that are statistically described. Similarly, a sociologist has his common sense only to rely on when confronted with a medical problem. Unless scientists obtain a thorough training in both biomedicine and social sciences, they cannot contribute to scientific development in both areas. In fact, those who use the term 'social epidemiology' acknowledge the failure of this 'field' to identify underlying mechanisms that account for the relation between social environment and health outcomes.^{3,7}

We therefore believe that epidemiologists, sociologists and psychologists should stick to their field of scientific inquiry. This does not imply that epidemiologists cannot use social or psychological determinants (income, stress) or outcomes (e.g. quality of life) in their studies. But even then, biomedical theory should link these items to the core parameters of a study (e.g. income as a determinant for nutritional status; stress as a determinant for hormonal imbalance; quality of life as a sequel of particular diseases). Similarly, sociologists and psychologists may enter medically defined variables (e.g. mortality, infertility) into their studies.

This is not just a statement of purity. We firmly believe that shopping in neighbouring scientific fields, without thorough subject-matter knowledge, will lead to statistical results without relevant meaning. As a consequence, research is reduced to statistically correct procedures without proper inference. Stretching borders between epidemiology as a biomedical discipline and sociology only leads to trivial statements, useless for society.

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Commentary: Society, biology and the logic of social epidemiology

Nancy Krieger

Perhaps one useful response to the thesis advanced by Zielhuis and Kiemeny, 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 involving 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.

Thesis 2: Population patterns of disease, disability, and death reflect population distributions of exposure, susceptibility, and resistance to conditions comprising sufficient causes for (or deterrents to) the specified outcomes and occurring during the aetiologically relevant time interval.

Deduction: Epidemiological explanations of current and changing patterns of disease, disability, and death must be compatible with temporally relevant and changing distributions of determinants of and deterrents to the specified outcomes.

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.^{2,18–22} Also relevant is training in concepts and methods of quantitative population sciences, including but not limited to biostatistics.^{2,20,22–23} 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).^{25,28,29} 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.^{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.^{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.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.⁴⁰

In summary, if Zielhuis and Kiemeny 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 Kiemeny 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.^{41–44} If logic and an open mind are prized features of scientific inquiry, the illogic and narrowness of Zielhuis and Kiemeny's argument are surely counterproductive to the further development of the enlightening and useful epidemiological research that we all desire.

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Commentary: Social epidemiology. A way?

KI Macdonald

Certainly let us share Zielhuis and Kiemeny'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. (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 routes to intervention.

However, that task surely entails some hesitations over Zielhuis and Kiemeny'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 'thrifty' 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 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 fetal 'memory'. Zielhuis and Kiemeny 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 ballpark. *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 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 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 socioeconomic factors generating such shocks were important. Some (now elementary) knowledge of chromosomal abnormalities readily shows Stott to be mistaken. 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 realized 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 canonized in the secondary literature as truths. Brown and Harris,⁴ 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 (Table 1) 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'. 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 ($P = 0.17$) by a log-linear model with no three-way interaction terms (using a first-letter shorthand, the model is: [ED][VD]

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^a (collapsed from Brown and Harris⁴ Ch. 11; Table 3)

Vulnerability	Depression		N
	Yes	No	
Low			
Event	10	90	88
No event	1	99	193
Mid			
Event	21	79	62
No event	4	96	53
High			
Event	79	21	14
No event	0	100	9

^a 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').

[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 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 compatible.⁵ 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 women's esteem 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.

We should support Zielhuis and Kiemeny'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 *et al.*⁶). 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.

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Commentary: Epidemiology? Keep it broad and deep

Klim McPherson

Zielhuis and Lambertus¹ are asking us to draw a boundary around epidemiology that includes only so called 'biological' determinants of disease. They invoke no clear justification or claim any advantage for so doing. Needless to say their argument seems to assume that biology is scientific and that the social sciences like sociology and psychology are not, and they also implicitly connect biology with medicine. This is a normal part of a sloppy argument. Public health in the UK is currently likewise professionally protected for doctors, and hence it has come to be synonymous with public health medicine. Then the illegitimate protection is self-justified and the dominant protectors of the boundaries (the BMA in this case) no longer need to justify them, except when challenged.

I believe we must resist (or simply ignore) this derivative proposal for similar reasons. Epidemiology is understanding the systematic determination of ill health by time, place and person. Firstly, it is the study of causes and determinants of disease and as such it must allow specialization within its boundaries to study, with enthusiasm, any plausible set of determinants. Secondly, most determinants interact in complicated ways, so even without specializing in social causes every epidemiologist must surely understand the rudiments of their potential effect. To exclude these experts by assigning them necessarily to another enterprise is simply stupid. To describe an epidemiologist

who confronts questions from sociology as 'merely a technician' describes precisely why.

What, moreover, is the point of attempting to exclude parts of the complex epidemiological landscape? I believe this argument is grounded in an elitist agenda (just like UK public health medicine currently is). All such elitist agendas must have, at the very least, a clear net benefit for the purpose of epidemiology, and in this case no such benefit can be possible. The single argument proposed in favour is that 'shopping in neighbouring scientific fields' invites inexpert interpretation. Well not necessarily! The solution is not to ban such progress but to require appropriate knowledge to enable people to make progress. After all the 'social' component of human immunodeficiency virus, of coronary heart disease and of most cancers is palpable and germane, but complex. The psychological component is less obvious possibly because we have all kept it on the periphery of our concerns. We should instead be encouraging rigorous and systematic study from knowledgeable specialists to advance this understanding too. To tell the best psychologists that they have no place in the epidemiological enterprise can only be justified by evidence, and these authors have none.

These proposals are therefore just silly! Worse, they invite further disparagement and scientific neglect of important areas. For what scientific purpose I simply cannot discern from their article.

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Commentary: Social epidemiology? Way!

Jay S Kaufman

The central assertion contained in the essay by Zielhuis and Kiemeny is that epidemiology is a 'part of medical sciences and rests on human-biological (scientific) background'.¹ On the basis of this broad contention, the authors attempt to draw two corollaries. The first is that training 'comparable to the training

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that medical practitioners need as a basis for medical practice' is necessary for an individual to practice epidemiology. The second is that epidemiologists must eschew study of social factors because their narrow clinical training provides them with insufficient understanding of these quantities.

Inarguably, 'basic training in biomedicine' is potentially useful for anyone engaged in the scientific study of health and disease. Nonetheless, people achieve intellectual development through varied means, of which formal progress through a degree programme in order to obtain a credential is but one. Ultimately, we do not judge scientific knowledge on the basis of the authors' credentials, but rather on the objective merit of the work.

For example, we do not reject the biostatistical techniques developed by Jerry Cornfield and James Robins, simply because these individuals never obtained doctoral degrees in statistics. Similarly, Gertrude Elion was awarded the Nobel Prize in Medicine in 1988, although she possessed no degree in medicine, nor in fact, any doctoral degree at all. While not seeking to denigrate formal training programmes, I merely note that these are a means to an end; namely, knowledge and understanding. We must never become so obsessed with our little merit badges (MD, PhD, etc.) that we mistake these for the actual professional competence they are meant to portend.

Moreover, if the authors believe that epidemiology 'rests on ... scientific background', then would it not be more advantageous for aspiring epidemiologists to obtain training in the conduct of scientific research, rather than the clinical instruction that prepares practitioners of the healing arts? Clinical training is appropriately geared to provide the skills necessary for the noble pursuits of diagnosis and healing. Even if we focus narrowly on 'medical knowledge', would the task of untangling disease aetiology not be well served by a doctorate in, say, physiology or biochemistry? At very least, formal training in an academic discipline provides some introduction to scientific conduct, research design, and statistical analysis—topics that justifiably might never surface in the training of an expert clinician.

These remarks are not intended to detract in any way from the many profound contributions made to our field by clinically trained epidemiologists. Rather, the accomplishments of such individuals demonstrate clearly a phenomenon that Drs Zielhuis and Kiemeny apparently fail to appreciate, which is the capacity of individuals to broaden themselves beyond their formal credentials. Though some of our finest methodologists, including Olli Miettinen, Kenneth Rothman and James Robins, obtained formal credentials as clinicians, they subsequently availed themselves of additional training or self-study in the quantitative dimension, without which they could not have distinguished themselves so remarkably as epidemiological pioneers and leaders. Likewise, many distinguished epidemiologists began professional life with a foundation in clinical training, and broadened themselves by pursuing greater understanding and expertise in the sociological dimension, including luminaries such as Mervyn Susser, John Cassel and Michael Marmot. Is it so difficult to imagine, therefore, that one might fruitfully approach epidemiology by obtaining doctoral training in a social or quantitative science, and subsequently, by one means or another, master elements of pathophysiology and anatomy?

The authors' central polemic, however, concerns the question of whether social factors constitute a legitimate province of epidemiological inquiry. Petulant and derisive remarks such as 'Epidemiology deals with medical knowledge itself' are intended to locate the causes of human disease strictly within the confines of the visceral body, as though this collection of organs and tissues is a lone satellite that floats haplessly through empty space. The greatest achievements of our field, however, belie such simplistic and parochial perspective. What would we know of the distribution of malaria without entomology and ecology? What would we know of the spread of HIV/AIDS without a detailed ethnology of human sexuality? Consider, for example, the West African Diaspora: several geographically

isolated populations, each composed of individuals with the same basic human physiology and the same distribution of basic genetic potential, but in radically different environments, ranging from subsistence agriculture in Nigeria to post-industrial late-Capitalism in Chicago, USA.² The variation between these groups in diseases such as hypertension and diabetes is striking, and the determining role of social institutions (e.g. diet, political economy) is readily apparent.

Traditional academic disciplines, like nation states, have borders that are constantly in flux because they have no objective justification. Lines are needed on the world map for governmental convenience, just as academics need disciplinary identities for purposes of training, providing credentials and administration of our professional societies and university departments. Nevertheless, nature resists such intellectual Balkanization, and great scientists frequently find themselves at the cusp of traditional fields. Was Linus Pauling, for example, a chemist or a physicist? Or to pick an example closer to home, is Roy Anderson an epidemiologist? A zoologist? A mathematician? Perhaps all of the above?³

In the attempt to make intellectual progress as epidemiologists, most of us find that we must also strive to be 'all of the above', simply because the facts of human epidemogenesis do not lie squarely within a single traditional discipline. Social interactions and structures, in particular, govern our diets, environmental exposures, physical activity, sexual contacts, and so forth. No modern epidemiologist would deny the centrality of such factors in determining disease incidence, so how could we justify *not* studying them? Zielhuis and Kiemeny offer several weak and disjointed arguments, advocating that we leave these quantities to others who better understand them (e.g. sociologists and psychologists). By this logic, however, we might also be forced to relinquish use of statistics to 'real' statisticians.

The task of integrating sociological and psychological expertise into our field is aided by the very fact that Zielhuis and Kiemeny decry: the prominent contributions made by numerous epidemiologists with formal training in these subjects, including Sherman James, George Kaplan, Stan Kasl, Carles Muntaner and Len Syme to name but a few. The social context in which organisms live is part of the 'human-biological (scientific) background' that Zielhuis and Kiemeny locate as the foundation of epidemiology, and is therefore an essential ingredient in the development and elaboration of the sorts of modern epidemiological theories to which they refer. In light of this central position of social relations in the processes by which human diseases wax and wane in populations, our task is clearly to develop a meaningful and fruitful social epidemiology, not to abandon study of these important questions to other disciplines.

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Commentary: Social epidemiology—a promising field

J Siegrist

The history of science tells us that the rise and fall of scientific disciplines depends on the successes and failures of respective research programmes rather than on the claims expressed and pressures exerted by individual members or groups. In this process, disciplines may even overcome their original paradigm by developing new cross-fertilizations, as is the case, for instance, in molecular biology. At a less prominent level, social epidemiology has evolved as one such cross-fertilization where theoretical and methodological knowledge and expertise derived from social and behavioural sciences (in particular sociology and psychology) have been introduced into epidemiology and public health research on determinants of human health and disease. In its short history social (or psychosocial) epidemiology has witnessed remarkable scientific progress. To mention just a few examples, a social gradient has been identified for a broad range of highly prevalent chronic diseases, and explanations of this gradient have been successfully advanced using models of health-related behaviour and of psychobiological stress research.^{1–3} Measures of innovative sociological and psychological concepts were introduced into prospective epidemiological investigations, and their direct and indirect effects on disease incidence were estimated.

Examples of newly discovered protective or risky psychosocial conditions for chronic diseases include social support,⁴ social network,⁵ high demand and low control at work,⁶ effort-reward imbalance,⁷ hostility,⁸ self-efficacy⁹ and optimism,¹⁰ among others. Above and beyond this prospectively established evidence some of these concepts have been associated with markers of psychobiological or pathophysiological mechanisms linking social environment and cognitive/affective processes in individuals with their physical responses.^{11–13}

Instead of evaluating this body of knowledge Zielhuis and Kiemeny¹⁴ put their energy into identifying a few authors who have used the term 'social epidemiology' in a broad way that does not reflect mainstream science. So what?

It is probably time to approach the problem of causality in biomedical epidemiology in a more critical way than is the case in the Zielhuis and Kiemeny article. Clearly, the causality criteria established by Hill¹⁵ in 1965 remain an important achievement, but we can no longer disregard epistemological advances taking place in contemporary biology and in a growing number

of biomedical fields where modern systems theory calls into question simple notions of causality.¹⁶ It may well be that biopsychosocial research on health and illness is better equipped to face these challenges than traditional biomedical epidemiology.

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