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Psychosocial epidemiology (that is, pertaining to the influence of social factors on a person’s behaviour, and to the interrelation of behavioural and social factors) is a controversial field within epidemiology. Here, we restrict our critique to epidemiological studies of psychosocial constructs and we acknowledge the relevance of neuroscience and neuroendocrinology to understand the proximal pathways by means of which psychosocial exposures affect the health of organisms. Our goal is not to attack the work that has been done on psychosocial factors, but argue for the need to further integrate psychosocial factors with social structure. Important research has also been conducted on the psychosocial pathways that might mediate the effect of social structure on individual physical and mental health. This research includes studies of job control (despite negative findings in the Framingham study), effort reward imbalance, social isolation/social support, and early psychosocial exposures. In addition, the Whitehall study has already provided evidence suggesting that control explains an important part of the health gradient among workers using both self reports and independent assessments. These studies lay the ground for future research that might integrate social structure, psychosocial exposures, and health.

However, some psychosocial constructs seem to gain and lose popularity without a strong justification for their fortunes. If this suspicion were correct, lack of strong cumulative progress might be attributable to methodological roadblocks such as the large number of omitted variables, the large number of associations that can be usually uncovered with a large enough sample, the occasional over-reliance on self reports, conflating independent and dependent variables (for example, social capital, violence, drug use), and the use of competing hypotheses that are as weak as those being tested.

Limitations also originate from psychosocial theory. Psychosocial constructs are expected to provide generalised risk factor associations across time and place; ignoring the determining social structure. Even when constructs are conceived under historical and social constraints, researchers seem to transform them into a-historical, psychological attributes. For example, the “type A” coronary prone behaviour pattern originated in clinical findings of the work related behaviour of mostly white men middle managers in post-war USA bureaucratic corporations. The construct thus referred to a specific period, cohort, age, class, sex, race, and organisation. Soon enough, health and social psychologists, epidemiologists, and public health researchers transformed this construct into the search for a “type A” personality or trait of presumed universality devoid of relations to its social context. The result was the narrowing of the concept to some forms of hostility (a psychological construct). Type A is not the only psychosocial construct that has been stripped of its link to historically specific social structures: “stressful life events” are apparently randomly distributed in the population; social cohesion and social support are implicitly defined in terms of US middle class social psychology, and “job stress” is presented as independent from the labour process, employment contracts, social class, or class exploitation. The absence of social structure is even more glaring with psychological constructs such as self esteem or sense of coherence. But some putative sociopsychological risk factors might be too dependent on a changing social structure to attain great generality across time and place. Without understanding the link between social psychology and social structure (for example, the “type A” behaviour might have been determined by post-second world war upward mobility, bureaucratic corporations with many middle managers, job stability, the Protestant work ethic, and meritocratic work values), psychosocial constructs are reduced to their common psychological denominator (for example, hostility) in a quest for replications with populations from different nations, large study samples, long follow up periods, and application of new statistical techniques. Although such a strategy, typical of risk factor epidemiology, can lead to high quality productive research programmes (for example, job demands and lack of autonomy as a risk factor for high blood pressure) it fails to deliver in terms of mechanisms and explanations in social epidemiology.

While there are virtually hundreds of measures of psychosocial variables (for example, think of “stress” scales), introducing measures of property relations and control over the labour process, the legal and economic foundation of our society, seems “off limits”. Instead researchers are expected to use “SES” rankings of education, income, or occupation with the functionalist assumptions that predominate in mainstream epidemiology. Unfortunately too many EU epidemiologists are willing to accept this state of affairs, presumably to harmonise with their US colleagues (for example, as in a recent sociological analysis of the Spanish civil war 1936–1939 in terms of lack of social capital). There are exceptions however, such as Richard Wilkinson and his collaborators who dared to challenge the “SES” orthodoxy by generating interest in income inequality. Not surprisingly, these researches have encountered a great deal of opposition, as in a recent publication by the American Enterprise Institute, that otherwise extols the virtues of research on job control. It is noteworthy to highlight that individual differences psychobiology and functionalist accounts of social inequalities in health feed on the reluctance to draw explicit social mechanisms (for example, exploitation) without moving beyond “SES” orderings (that is, as in references to mysterious gradients or fundamental causes; see Gottfredson). We understand ideology as a system of factual statements and value judgments that inspires social, including public health, policies. Given such definition, psychosocial epidemiology becomes mostly ideological when the policies it inspires lack scientific justification (for example, “subjective stress is the major social determinant of cardiovascular mortality, therefore we should focus on changing people’s perceptions of their social and work environment.”) Thus, to provide more accurate and useful accounts of how society affects health, a-historical and structure-less psychosocial constructs (for example, “social capital”, “sense of coherence”, “hostility”, “life events”, “job stress”, “social support”, “self esteem”) could be replaced with less ideological, historically specific (for example, age-cohort-period) models in which social structure and psychosocial exposures are integrated into mechanisms that influence population patterns of mortality and morbidity.
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