Sticky webs, hungry spiders, buzzing flies, and fractal metaphors: on the misleading juxtaposition of “risk factor” versus “social” epidemiology

A fly, buzzing about in a sunny meadow one afternoon, veers off towards some trees and suddenly becomes ensnared in a spider’s sticky web. The spider bites, paralysis, and then eats the fly. What caused the fly’s demise? Its change of course? The sticky web? The venom? The spider?

Meanwhile, another fly heads towards the same tree, yet avoids the web and buzzes on to another field, even as a third fly briefly stuck in a similar web frees itself—followed by another fly who dies in that same spider’s web. In fact, of the 100 flies in the meadow that afternoon, 20 die trapped and eaten in spiders’ webs, while 80 live on for another day. What caused one fifth of the flies to die as a spider’s meal? The sum of factors leading to each individual fly’s demise? The number and ratio of spiders to flies that afternoon? A decline in predators of spiders? A good growth of adjacent trees hospitable for spiders weaving webs? And why, in another glade, were flies more likely to perish, the ratio of trapped to free flies 1:2, not 1:4?

At first glance, these are not the kinds of questions that epidemiologists usually consider. And yet the conceptual issues they raise go to the heart of a growing debate—as to perish, the ratio of trapped to free flies 1:2, not 1:4? 

At one level, the contours of the current epidemiological controversy are not new, and raise issues thoughtful people have debated for centuries. At an abstract level, these contested topics include: whether “wholes” can be “reduced to” or “explained” by their “parts”, whether statistical “objects” analysed in empirical research are “real” or “constructed”, whether scientists study “causes” or “correlations”, whether science is “objective” or “value laden”—and even whether posing such “either/or” propositions is useful or misleading. In the case of epidemiology, these debates translate into concrete concerns over how we comprehend and interpret social and biological phenomena as determinants of population health. A related issue involves what public health recommendations, if any, we offer—to whom—based on results of our studies, and if we have any responsibility to conduct research on social inequalities in health. These are all critical issues worthy of consideration, especially at a time of stunning advances in molecular biology and equally stunning increases in global economic inequalities and insecurities, both within and between countries.

To further this discussion, I question a curious and increasingly common juxtaposition: that of “risk factor” versus “social epidemiology”. Framing these two types of epidemiology as mutually exclusive misleadingly implies: (1) that epidemiological studies of social determinants of disease, disability and death do not analyse “risk factors”; and (2) that “risk factors” are atheoretical constructs. Rather, the point is that all epidemiological studies include data on variables hypothesised to be component causes of the specified outcomes, thereby raising critical questions as to which variables are included, which are excluded, and why.

Firstly, “social epidemiological” research examines myriad specific factors—appropriately termed “risk factors”—in causal pathways to disease occurrence; selected examples from an enormous array include lead, organochlorines, occupational hazards, high fat diets, hormone levels, receptor levels, molecular biomarkers, and so on. Concomitantly, however, such research investigates how population distributions of these factors is shaped by social processes within and across social groups over time (within the lifecourse of individuals and also across generations) and whether such factors explain the current and changing societal burdens of disease.

Beyond, this social epidemiological research expands the purview of “factors” considered determinants of health by analysing how social phenomena—such as racial discrimination, violence, income distribution, capital flight, residential segregation, welfare policies, or violation of human rights, along with people’s individual and collective responses to these phenomena—affect our biology and our health, both directly and through mediated pathways. In all cases, for each type of “factor” considered, both social and biological plausibility matter, whether in terms of relevant aetiological period or actual impact of exposure on population health.

Secondly, “risk factors” do not simply “show up” in our epidemiological data sets, devoid of theoretical considerations. Instead, we choose to obtain and analyse data on specified “risk factors” because of how we think about determinants or confounders of the exposure-susceptibility-outcome relations we are studying. The problem then becomes one of understanding the framework(s) guiding selection of variables, and also, as importantly, omission of unmeasured factors relevant to disease occurrence and its societal distribution. To use a familiar example, denoting “lack of condom use” as a “risk factor” for HIV infection among women, absent data on gender and economic inequality as determinants of men’s condom use, suggests reliance on
individualistic approaches to understanding and investigating disease aetiology; by contrast, studying “lack of condom use” by men as a risk factor in the pathway from violence and lack of economic resources to exposure to HIV provides insight into why poor women are at excess risk of HIV infection.20 22 23

Or, alternatively, consider research on health consequences of cortisol mediated stress responses. Conceptualising cortisol induced levels of corticotropic releasing hormone (CRH) as a “risk factor” for preterm delivery and seeking to reduce risk by administering CRH inhibitors,24 absence consideration of social inequalities in occurrence of preterm delivery (for example, higher risk in the US among black compared with white women and poor compared with affluent women44 46), signals an individualistic, non-contextualised approach to explaining and changing distribution of adverse health outcomes. Conversely, investigating CRH levels in relation to experiences of racial discrimination and economic stress and the social distribution of preterm delivery potentially can lead to alternative explanations and interventions that focus attention on risky environments for fetuses and, as importantly, the overall well being of women and infants.44 46 48 At issue, then, are not “risk factors” themselves, but rather what factors we study, how, and in what context.

Thus, to the extent that distinctions are useful, rather than juxtapose “risk factor” to “social” epidemiology, the contrasting approaches would perhaps be more fruitfully termed “individualistic” versus “social” epidemiology, or “isolated” versus “contextualised” investigation of causal components. Even so, such distinctions potentially may adversely compartmentalise epidemiological research and thinking into “micro”, “meso”, and “macro” levels.1 49 Suppose, for example, that epidemiological research finds that:

1. increased placental CRH levels are associated with higher risk of premature delivery, independent of other known risk factors, and

2. levels of placental CRH are higher (a) among poor compared with more affluent pregnant women (taking into account their economic history, spanning from mother’s childhood through their own adulthood), and (b) among economically equivalent pregnant black women who report more versus less exposure to interpersonal racial discrimination.

If so, then it is not as if “CRH levels” independently exist at the “micro” level, with “poverty” or “racial discrimination” consigned to the “macro” level and “stress response” to the “meso” level. Rather, in this example, placental CRH levels are the embodied biological expression of class, race, and gender relations,1 4 17 18 19 with such expression conceivably reflecting the hypothesised evolutionary role of CRH in pacing embryonic development in relation to environmental stress.41

The importance of epidemiologists recognising embodied expressions of social experiences is highlighted by Sapolsky’s accounts of researchers overlooking how size of both the adrenal and thymus gland are affected by chronic poverty and stress,42 thereby creating what Brunner has termed a “biology of inequality”.43 Thus, in the early 1900s, physicians determined “normal” size of adrenal glands based on cadavers of the poor, the group most likely to have a necropsy; “on the infrequent occasion when the body of someone with a higher income was examined, it was noted that the adrenal glands seemed oddly undersized”—a condition termed “idiopathic adrenal atrophy”. As Sapolsky notes, “This ‘disease’ flourished in the early 20th century; then physicians caught on, and the disorder was transformed overnight into an embarrassing footnote”.43 Misconceptions of “normal” thymus size had more severe public health consequences: unnecessary irradiation of infants with allegedly “large” thymus glands (a condition termed “status thymicolympathicus”) to prevent sudden infant death, leading to iatrogenic thyroid cancer.44 The source of this error was a comparison of thymus gland size among infants who died suddenly versus of chronic illnesses, the latter a more common cause of death among the poor. Such illness—along with other aspects of poverty—would have heightened cortisol excretion, thereby suppressing immune function and shrinking the thymus; thus, what was deemed a “large” thymus among infants who died of non-chronic illness was in fact a thymus gland of “healthy” size. We cannot adequately study people or assess variation in traits unless we specify social context.

Ultimately, our challenge as epidemiologists—no matter what level our research—is for us all to grapple conceptually with the simultaneously of “macro” to “micro” levels45 and their embodied expression in current and changing population distributions of disease, disability, and death. This challenge by definition requires uniting social, biological, and statistical reasoning. It is the desire to advance this conceptual integration that motivates calls for development of such frameworks as ecocultural theory19 and eco-epidemiology24 and related “ecologic . . . dynamic, interactive, life-course models of disease risk acquisition”.25 And it is why, when I raised questions about the dominant individualistic and biomedical epidemiological metaphor of a “web of causation”—sans spider—that I offered, as an alternative ecocultural image, a fractal metaphor asking us to consider the inherently conjunct expression of biological and social factors at every level, from molecular biology of our cells to population rates of disease.19 This image of social and biological engagement, repeatedly expressed at each and every scale, differs from models with distinct levels “interacting”. And it requires us to frame, in context, our particular investigation and choice of specified risk factors, whether we study relations among webs, spiders, flies, and forests, or people in the world that we inhabit and transform. By engaging this challenge, we will more effectively address fundamental empirical, theoretical, methodological, and practical tasks of our field, so that we may better describe, explain, and predict trends in—and generate knowledge useful for improving—societal patterns of health, disease, and well being.

NANCY KRIEGER

Department of Health and Social Behavior, Harvard School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA

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