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Invited Commentary

Invited Commentary: What Social Epidemiology Brings to the Table—Reconciling Social Epidemiology and Causal Inference

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In response to the Galea and Hernán article, "Win-Win: Reconciling Social Epidemiology and Causal Inference" (*Am J Epidemiol.* 2020;189(3):167–170), we offer a definition of social epidemiology. We then argue that methodological challenges most salient to social epidemiology have not been adequately addressed in quantitative causal inference, that identifying causes is a worthy scientific goal, and that quantitative causal inference can learn from social epidemiology's methodological innovations. Finally, we make 3 recommendations for quantitative causal inference.

causal inference; quantitative; social epidemiology; triangulation

Galea and Hernán describe their commentary (1) as "a guide for social epidemiologists." Although we agree with the authors' conclusion that "questions of social epidemiology may be particularly fertile ground for causal thinking," we disagree that the onus of introspection should be on social epidemiology's shoulders. Quantitative causal inference can learn much from social epidemiologists, many of whom have been conducting ground-breaking research that has advanced causal inference and population health, even when that research occurred outside of quantitative causal inference.

DEFINITION OF SOCIAL EPIDEMIOLOGY: A RESPONSE

The authors characterize social epidemiology as being "concerned with the health effects of forces that are 'above the skin'" (1). We prefer Honjo's definition: social epidemiology is "concerned specifically with the health effects of social institutions, structures, relationships, and dynamics over time" (2). Unlike Hernán and Galea's definition, Honjo's captures the dynamic nature of social organization and allows for the fact that social organization can be embodied under the skin. Social epidemiologist Nancy Krieger's ecosocial theory has challenged epidemiology to investigate how the social and material world becomes literally embodied in our biology. Furthermore, Krieger and others have illuminated that we cannot fully understand our biology without incorporating the historical, contemporary, and intergenerational social dynamics that shaped all our bodies' developments (3, 4).

MISCONCEPTION 1: A RESPONSE

Galea and Hernán argue that individual-level medical treatments, behaviors, and biomarkers "are arguably in the same relative position along [their experimental manipulation] spectrum as income, residential segregation, and race" (1). Having stated this position, they conclude, "This sheds the notion that social factors are in some way distinct and stand apart from other epidemiologic exposures." We emphatically disagree.

Methodological advances in quantitative causal inference have been driven by the research agendas of its prominent advocates. Because many of these leaders conducted research on individual-level, biomedically focused questions in pharmacoepidemiology, human immunodeficiency virus, and occupational health, quantitative causal inference has advanced more quickly around these issues, producing accessible tools and applied examples. In contrast, complex processes of particular interest to social epidemiology have received less thoughtful attention. Social epidemiology's questions often involve data structures that are necessarily multilevel (versus individual-level), effects that are interdependent (versus conceptualized as acting in isolation), variables existing within feedback loops (because human agency leads people to respond to interventions), and effects

that are dependent on context within the lifecourse, across generations, and in time and space. We find that applications of quantitative causal inference (e.g., directed acyclic graphs, individual attribute-focused potential outcomes) are not yet well developed for many of the research challenges most salient in social epidemiology.

MISCONCEPTION 2: A RESPONSE

Misconception 2 ("The goal of causal inference is to identify 'causes'" (1)) led us to read the excellent 2016 commentary (5) in which Hernán clarifies that quantitative causal inference "can be used to define causal effects, but it cannot generally be used to identify causes." In social epidemiology, our goal often is to provide evidence to establish or rule out causation. In fact, some of our most important questions have centered on establishing or ruling out causes. Is income inequality good or bad for population health (6)? Are black-white racial disparities in cancer subtypes due to genetic destiny, or are they historically contingent (7)? Does residential segregation cause higher hypertension among black Americans (8)? Did the racialization of Arab Americans after September 11, 2001, worsen their birth outcomes (9, 10)?

Establishing causation matters scientifically (and practically) because the direction a research field takes depends on basic beliefs about what factors are causal. In social epidemiology, as in other fields like environmental health, climate change, and vaccination safety, there are prominent voices that deny scientific evidence that validates asking certain questions—in our case, questions about how social processes and access to resources affect health. Given the politicized nature of many research questions in social epidemiology, investigating whether social factors, indeed, affect health is an important part of the work in social epidemiology. Remaining agnostic about causes or shifting the questions toward more proximal processes tends to constrain our work to the terrain of the skeptical denialists.

MISCONCEPTION 3: RESPONSE

In "Misconception 3," the authors reassure social epidemiologists that "experimental manipulation is not a prerequisite for meaningful causal inference" (1). However, later, Galea and Hernán rank social factors on a spectrum based on specificity of hypothetical experiments, placing experimental manipulation on the top of the hierarchy for causal inference. The authors state, "If our goal is to improve health, ... we need to move to the right on the aforementioned spectrum," which usually translates to moving further "downstream" on the web of causation without talking about the spider(s) that created the web in the first place (1, 4).

The author of the article cited in this section of the Galea and Hernán paper argues that conceptualizing research questions as experiments improves adherence to the consistency assumption, which requires framing treatments precisely (5). The author concedes, "It is impossible to provide an absolutely precise definition of a version of treatment" (5). He concludes, however, "Declaring a version of treatment sufficiently well-defined is a matter of agreement among experts based on the available substantive knowledge."

The subject matter knowledge that social epidemiologists have about context-specific social dynamics is key here. Galea and Hernán imply that social epidemiologists resist framing all our research questions as randomized controlled experiments because we do not appreciate that slightly different versions of treatment can have very different health effects. In fact, the opposite is true. Take Galea and Hernán's approving example of income as a social variable "closest to the 'perfect consensus' extreme of" the causal spectrum (1). They propose an experiment they believe is sufficiently precise: "supplementing salaries with a 30% bonus during the study period" (1). To a social epidemiologist, much "meaningful vagueness" remains in this hypothetical experiment: What about retired adults on Medicare or stay-at-home parents? What are median incomes like? A 30% bump means different things to a millionaire than to someone making minimum wage. To whom exactly is the money distributed: only wage earners, all members, a female head of household? What is public sentiment about these payments: are they stigmatized in any way? Each of these specifications could alter the intervention's effects, especially in the long term (as well as exacerbating or ameliorating geographic, gender, racial, and wealth disparities).

The subject matter knowledge of social epidemiology teaches us that context matters. In fact, we believe context matters more for the study of social processes on health than it does for the biological processes that Galea and Hernán argue are exactly the same as social processes. Because we are so interested in the ways in which contexts and social positioning shape processes and causal effects, we resist reducing away the context that would complicate a "sufficiently well-defined" intervention. As Hernán noted in 2016, there is a trade-off between absolute precision and meaningfulness (5). Our subject matter expertise leads us to believe pursuing the most specific versions of social exposures would sometimes back us into research so nongeneralizable (e.g., $n = 1$) that it is no longer meaningful. Fortunately, for the field of social epidemiology, scholars have grappled with these issues and devised ways to generate causal inference without stripping away context, as we discuss in the final section.

A NOTE ON CONSEQUENTIALIST EPIDEMIOLOGY

One note before we conclude: One of the most surprising elements of the commentary is the argument that adopting quantitative causal inference, particularly moving right on the experimental spectrum, is the most effective strategy for epidemiology to influence policy and lead to actionable interventions to improve population health and reduce racism. Colleagues who work at the intersection of public health and policy do not support this view (11). In democratic nations, systemic change tends to be driven not by precise quantitative estimates but by narratives about causation, cultural values, and attribution of moral responsibility (12). Moreover, we fear that focusing heavily on variables on the right end of the experimental spectrum reinforces a propensity in the United States and other parts of the

world to attribute causes and responsibility to individuals versus systems and structures. We fear that reproduction of an individual-focused "social" epidemiology will steer the field toward interventions that rely on stigma toward individuals versus the possibility of structural change or collective action (13).

A WAY FORWARD FOR QUANTITATIVE CAUSAL INFERENCE

Misconceptions about social epidemiology's openness to causal inference have hampered the progress of quantitative causal inference. Here, we propose a way forward that illustrates how the accumulated expertise of social epidemiology can inform the development of better and more accessible tools for applying quantitative causal inference to a broader range of research questions.

First, quantitative causal inference should learn from social epidemiology how to better conceptualize social variables. For example, the authors operationalized variables like income, segregation, and race by imagining simplified and decontextualized experiments in which the unit of analysis would be an individual or household. However, in social epidemiology, subject matter experts have distinguished among operationalizations of interdependent concepts like "race," "racism," and "racialization" at varying levels of potential intervention, from the individual to the society, and nested within moderating contexts (14). Incorporating richer operationalizations of treatments will only strengthen quantitative causal inference.

Second, quantitative causal inference should innovate to address methodological questions of particular interest in social epidemiology. For example, quantitative causal inference should develop more accessible tools for integrating multilevel contexts, life course and intergenerational processes, interference between levels of causation, and heterogeneity across contexts into causal models. In addition, tools and frameworks incorporating historical trajectories, interdependent social dynamics, and the embodiment of both would find a welcome audience among social epidemiologists and others in public health and biomedical fields.

Third, we believe quantitative causal inference has much to learn about study design from the elegant work conducted by our social epidemiology "causal ancestors" (ha!). In particular, we suggest that methodologists in quantitative causal inference analyze study designs used in social epidemiology. Encoding these study designs into the visual language of directed acyclic graphs and structural language of quantitative causal inference would advance epidemiology as a field. Lawlor et al. (15) have demonstrated this approach well. In "Triangulation in aetiological epidemiology," she and her colleagues model an approach by which quantitative causal inference could be used to integrate and evaluate findings from fields outside the quantitative causal inference tradition (15). They present diverse study designs and encode them in the language of quantitative causal inference, without dismissing designs that do not conform to quantitative causal inference's cultural norms.

As an example of how quantitative causal inference could learn from social epidemiology, Galea and Hernán argue in their commentary that it is difficult to identify and measure confounders for a vague exposure like "race." We don't disagree: race is a social construction hinged upon processes of racialization and racism. Furthermore, even with perfect conceptualization and state-of-the-art measurement, it is difficult to imagine capturing variables on every confounding pathway involved with questions about racial inequalities in health. To counter this challenge, social epidemiologists have used study design to "block" confounding pathways. From studies of breast cancer rates among Japanese-Americans immigrants and their descendants (16) to studies of infant mortality among people from the African diaspora to (17) to ecological studies of geographic differences in changes in breast cancer rates (18), a common bond is researchers who carefully constructed studies in which confounding pathways were plausibly blocked even when those paths were populated by unmeasured variables.

One of us took a similar approach when investigating racial differences in the sex gap in obesity prevalence among US adults (19). We leveraged the study of LaVeist et al., Exploring Health Disparities in Integrated Communities (20). In that study, the researchers addressed confounding among race, socioeconomic status, and segregation by focusing on health disparities within racially integrated communities where socioeconomic status did not vary by race (20). One of the most satisfying aspects of our study was constructing a directed acyclic graph that revealed the causal structure underlying the intuition of our study design (see Figure 2 in (19)).

CONCLUSION

We agree that questions of social epidemiology are fertile ground for causal thinking. We hope this dialog spurs quantitative causal inference to integrate concepts from, address methodological challenges in, and learn from existing methods used in social epidemiology.

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