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Social epidemiology for the 21st century

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ABSTRACT

Social epidemiology, as defined by the textbook of the same name (Berkman et al., 2014) is "that branch of epidemiology concerned with the way that social structures, institutions, and relationships influence health" (p. 2). As our Special Issue commemorates the 50th anniversary of Social Science & Medicine, it is worth noting that the Social Epidemiology office within the journal has existed only for a fraction of that time (fifteen years). So what has been learned in the fifteen years since the establishment of the new office? In this commentary, we spotlight some of the achievements, substantive topics, and future trends in the research papers that we have featured in our Section of the journal.

1. The contours of social epidemiology

Social epidemiology, as defined by the textbook of the same name (Berkman et al., 2014) is "that branch of epidemiology concerned with the way that social structures, institutions, and relationships influence health" (p. 2). As our Special Issue commemorates the 50th anniversary of *Social Science & Medicine*, it is worth noting that the Social Epidemiology office within the journal has existed only for a fraction of that time (fifteen years). As we look back, what have been the distinctive contributions of social epidemiology, especially as reflected by the papers published in our section? First of all, the discipline has been positioned from the outset at the confluence of public health with other currents of social sciences such as economics, geography, psychology, and sociology. More than any other branch of epidemiology, social epidemiologists have engaged with other social scientists – for example, entering critical debates with economists on the role of income inequality in influencing population health (Clarkwest, 2008; Zimmerman, 2008; Glymour, 2008). This level of critical engagement also spurred the early adoption of analytical techniques imported from other fields, such as the use of econometric techniques to strengthen causal inference. As we noted in our inaugural 2002 editorial:

> "In contrast to other specialties within epidemiology that are defined by health outcomes (e.g., cancer epidemiology), we are a field defined by our concern for describing and intervening on social conditions that either promote or harm health. In this cross-disciplinary enterprise, sometimes we have borrowed and applied *theories* from other fields, e.g., theories of social capital, imported from sociology and political science. Sometimes we have borrowed *measurement tools*, e.g., measures of income distribution from welfare economics, or measures of control from social psychology. Sometimes, we have borrowed *analytical techniques*, e.g., multi-level analysis from medical geography and educational statistics." (Kawachi, 2002, p. 1739).

That description of the field still resonates today; our editorial office (if not all of *SSM*) remains committed to inter-disciplinary "arbitrage", i.e. leveraging theories, measurements and analytical tools from other social sciences to shed light on questions relevant to population health. Indeed, in doing so, perspectives shaped by health inequalities also changed the nature of research conducted by social scientists. Studying health related issues is no longer just for mavericks in fields such as sociology or economics where "theoretical" research is often privileged over "applied" research. The emergence – and importantly, broader recognition and prominence – of social epidemiology as a legitimate field of inquiry in itself may have played some role in mainstreaming health in social sciences.

So what has been learned in the fifteen years since the establishment of the new office? First of all we have witnessed a tremendous growth of empirical studies. The rise in research output is reflected in the trends in submissions to *Social Science & Medicine* (not to mention trends in our reviewers' workload). In its first year, the Social Epidemiology office received 50 manuscript submissions; last year we handled almost 1000. In parallel, the first edition of the textbook *Social Epidemiology* (Berkman and Kawachi, 2000) weighed in at 391 pages. Fourteen years later, the 2nd edition came in at 615 pages (Berkman et al., 2014). Not only is the volume of submissions rising steadily, but there has been a parallel increase in the quality of evidence. Studies are moving from description (e.g. descriptive studies of health inequalities) toward explanation (studies that interrogate the causal association between social

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determinants and health outcomes). Study designs have also moved from cross-sectional and ecological studies toward longitudinal – and increasingly – quasi-experimental designs.

A pertinent example of what we mean is illustrated in the area of neighborhood contextual influences on health. Early studies sought to demonstrate the presence of meaningful variation in health behaviors and health outcomes between areas that could not be explained by the compositional characteristics of residents (Subramanian et al., 2001). Today it would be difficult for an author to get past desk rejection by submitting (yet) another cross-sectional study replicating the correlation between (say) neighborhood food environment and dietary practices. Michael Oakes threw down a formidable challenge to neighborhood researchers in 2004 when he pointed out the "impossibility of identifying useful independent neighborhood effect parameters, as currently conceptualized with observational data" (Oakes, 2004). In his article (which is now assigned in many social epidemiology classes, and has garnered 332 citations at the time of this writing), Oakes argued that research on neighborhood effects frequently violated the assumption of "positivity", i.e. the requirement for sufficient people to be present within each stratum of confounders when contrasting health outcomes between different neighborhoods. To illustrate, when attempting to identify the causal impact of living in a poor neighborhood on infant mortality, it is difficult (if not impossible) to "control" for compositional confounding by individual socioeconomic status, because very few affluent people live in poor neighborhoods due to the phenomenon of economic segregation. To the extent that rich people can be found living in poor neighborhoods, it raises the suspicion that they are different from other rich people in subtly unobserved ways.

Notwithstanding the broader range of inquiry that can be meaningfully pursued via the development of advanced analytical techniques, the impact of Oakes' critique of structural confounding can be seen in the way researchers have been forced to engage more deeply with causal inference, such as taking advantage of natural experiments to test long-held hypotheses. Occasionally, doing so has produced conclusions that public health advocates had not hoped for. For example, when Sturm & Hattori evaluated the "Los Angeles Fast-Food Ban" – a zoning moratorium that restricted the opening/remodeling of standalone fast-food restaurants in South Los Angeles – they could find no evidence for a reduction in either fast-food consumption or overweight/obesity rates among residents. Presumably the residents went elsewhere to get their fast food (Sturm and Hattori, 2015). At other times, the results of natural experiments have been more encouraging for the neighborhoods research enterprise. When Giles-Corti et al. (2013) evaluated the longitudinal impact of an urban planning natural experiment in metropolitan Perth, Western Australia, they found that both transport and recreational walking behaviors responded positively to changes in the availability and diversity of local transport- and recreational destinations. The jury is still out on neighborhood contexts contribute to health behaviors such as physical activity and diet. There is more to learn from natural experiments like these.

As social epidemiologists have been engaging more with casual inference and counterfactual reasoning, we are also beginning to see a robust (and much needed) debate on how much we can learn from going down this path. For example, Sharon Schwartz has questioned whether the framing of all causal questions in social epidemiology in terms of a well-defined intervention could be too restrictive and even politically conservative (Schwartz et al., 2016).

2. Engagement with biology

The establishment of the Social Epidemiology office at SSM coincided with the explosion in genomic research (as well as other kinds of omics). The decoding of the human genome (announced in February 2001) was swiftly followed by a major challenge to a long-held belief in the social sciences, viz. the meaning and interpretation of the variable "race". Prior to 2001, there was widespread consensus among the social sciences (and even population biologists) that "race" had no inherent biological meaning, based on work by scholars such as Richard Lewontin suggesting that the vast majority of human genetic variation was observed *within* so-called racial groups, and not between them. That changed with the publication of the November 2004 issue of *Nature Genetics* ("*Genetics for the Human Race*", volume 36, No 11s), which confirmed the existence of major genetic clusters based on people's continental origin, using multi-allelic microsatellite loci and single nucleotide polymorphisms (SNPs). These clusters were found to (roughly) correspond to the major "racial" groups reported on the U.S. Census – i.e. African-Americans, white Americans, Asian-Americans. With that discovery, genetic researchers could once again reclaim the use of "race" as a meaningful biologic variable in their analyses. On the other hand, what relevance our ancestors' continental origin has for explaining racial *disparities* in health remains highly contested. For example, in reviewing the sixty-eight genome-wide association studies (GWAS) of cardiovascular disease published between 2007 and 2013, Kaufman et al. (2015) came to the sobering conclusion: "Despite the rapid increase in the number of genomic studies over the past decade … the accumulated evidence for a genetic contribution to cardiovascular disease disparities in blacks versus whites has been essentially nil." In the meantime, there is plenty of evidence of the unequal and unfair treatment of racial minorities that could give rise to health disparities – and keep social epidemiologists busy for the foreseeable future.

A different approach to engaging with advances in biological science has been characterized by the use of novel biomarkers to understand how socioeconomic disadvantage "gets under the skin" to produce the widespread health adversities, collectively referred to as the "SES gradient" in health. In the last 20 years, researchers have tackled this question by studying the nexus between SES and HPA axis activation ("allostatic load"), epigenetic changes (DNA methylation), genome-wide gene expression, and other stress-related mechanisms (Kubzansky et al., 2014). Our journal (both the Social Epidemiology and the Health Psychology sections) also has published studies that focused on leukocyte telomere length (LTL) as a marker of cellular aging, based on the theory that the chronic stress associated with socioeconomic disadvantage induces a form of "accelerated aging". As our former Editor-in-Chief, Sally Macintyre once remarked (personal communication), socioeconomic disadvantage is correlated with a "speeding up" of critical life events. Disadvantaged babies are more likely to be born premature, disadvantaged school-children are more likely to drop out of school earlier, disadvantaged working adults are more likely to retire early due to disability, and all of these cumulative disadvantages tend to speed people's lives toward a premature death. Many of these phenomena have sociological explanations, but there has also been growing interest in identifying the biological signature of cumulative chronic stress that could help to explain the broad range of adverse health outcomes associated with socioeconomic disadvantage. Leukocyte telomere length (LTL) shortening has been proposed as one such marker of chronic stress and accelerated physiologic aging.

Needham et al. (2012) demonstrated in a sample of US black and white children (aged 7–13) that parental education was positively associated with child LTL, net of controls for sex, age, race/ethnicity, and family income. Compared to children with at least one college-educated parent, children whose parents never attended college had telomeres shorter by 1,178 base pairs, which is roughly equivalent to 6 years of additional aging (Needham et al., 2012). In a separate study based on adults in the National Health and Nutrition Examination Survey, 1999–2002, the same team of authors found that individuals who completed less than a high school education had significantly shorter telomeres than those who graduated from college (Needham et al., 2013).

3. Health inequalities – old debates and new directions

Understanding the origins and consequences of health inequalities –

or the health consequences of social stratification – continues to be the bread & butter of social epidemiology, and indeed, of most other sections of the journal. Indeed Social Science & Medicine was identified as the top most cited source of articles on health inequalities during the past half century (1966–2014), according to a bibliometric analysis conducted by Bouchard et al. (2015).

In parallel with the trends we described earlier, submissions on the topic of health inequalities have moved beyond description toward a more causal and mechanistic understanding of why socioeconomic status is such a robust predictor of health outcomes. Social epidemiologists have also fully embraced the bidirectional associations between health disparities and social/political processes. For instance, the excess mortality of marginalized populations is often theorized to be a *consequence* of unequal political representation (e.g. the systematic disenfranchisement of colored and low income voters in the United States via voter suppression laws and electoral purging). Rodriguez et al. (2015) turned that correlation on its head, to ask the question: What impact do racial inequalities in longevity have on electoral participation? The authors estimated the impact of mortality differentials between blacks and whites from 1970 to 2004 on the racial composition of the electorate in the US general election of 2004. The authors estimated that approximately 1 million black votes were "missing" in 2004 due to excess mortality, and that of these, 900,000 votes were lost by the defeated Democratic presidential nominee.

Another major theme to emerge in social stratification research in the last two decades focuses on the population health effects of income inequality. Income inequality in many parts of the world have risen to levels not seen since the Gilded Age (Piketty, 2014), and our Social Epidemiology section has frequently showcased the research drawing the connection between income distribution and population health, including state-of-the-art reviews by two of the pioneering researchers in the field, Richard Wilkinson and Kate Pickett (2006, 2015). The correlation between income distribution and health has been repeatedly observed. Earlier debates in the field dismissed this correlation as a "statistical artifact" arising from the concave shape of the relationship between income and health (Gravelle, 1999). The argument proceeded as follows: Given the stronger association between income and health among poor people compared to rich people, it must follow that a society with more poor people will end up with both greater income inequality as well as worse average health achievement. Ergo, the problem is not income inequality, but poverty. What these critics failed to notice, however, is that the same argument applies to the potential for income *redistribution* to raise the level of health in society. Some researchers have now attempted to quantify the implied gains in population health as a result of income redistribution (Blakely and Wilson, 2006; Brodish and Hakes, 2016). For example, based on a simulation using data from the National Longitudinal Mortality Study, Brodish & Hakes (2016) estimate that each 10% reduction in income inequality, as assessed by the Gini coefficient at the state?? level is consistent with a 5% reduction in all-cause mortality. While this may seem like a modest population health gain, it is in fact equivalent to eliminating all unintentional injury deaths in the United States.

A more contentious line of research has sought to isolate the "contextual effect" of income inequality on individual health. This theory – originally proposed by Richard Wilkinson – posits that everyone in society (or almost everybody except the very rich) will end up paying the price of inequality, not just the poor (as the "concavity hypothesis" would suggest). The proposed mechanisms for this effect include the erosion of social cohesion accompanied by systematic under-investment in social safety nets (because the "haves" no longer care about the welfare of the "have nots"), as well as chronic stress induced by feelings of insecurity, frustration, and shame among those left behind. One of the biggest challenges to this line of theory is that when it has been tested with econometric techniques – specifically fixed effects regression – most studies have failed to find an association between income inequality and individual health. That is, when researchers have attempted to correlate *changes* in income distribution to *changes* in health status, the conclusions have been null. In 2008, our section spotlighted this debate, with an article by Andrew Clarkwest, accompanied by commentaries by Fred Zimmerman and Maria Glymour. Clarkwest confirmed that when state-level changes in life expectancy from 1970 to 2000 are regressed on changes in Gini, there is no significant association between income inequality and longevity. However, when he included *baseline* Gini as a right-hand variable in his fixed effects regression, the coefficient for the *change* in Gini became statistically significant, indicating an association between rising Gini (i.e. rising inequality) and slower improvements in life expectancy. It was the latter "fix" which vexed our commentator Fred Zimmerman. He argued that introducing baseline Gini as an adjustment variable into fixed effects regression likely *introduced* bias, and ended up sacrificing the advantage of fitting a fixed effects model in the first place. The commentary by Maria Glymour (2008) sides with Zimmerman's critique, and her careful argument is, itself, a terrific tutorial on fixed effects analysis that ought to be assigned to every classroom in social epidemiology.

More importantly, Glymour's commentary also pointed out a critical assumption in fixed effects analysis, which is that they typically focus on contemporaneous changes in income inequality and health over relatively short periods of time (typically 3–5 years), whereas growing evidence points to much longer lag periods between exposure and health effects and/or potential "sensitive" periods of exposure. If the relevant etiologic period is longer than the time period specified in fixed effects models, the answer ("no effect") could be wrong. For example, research suggests that exposure to income inequality during infancy & childhood (Lillard et al., 2015; Siddiqi et al., 2016; Elgar et al., 2017) may be most relevant to health outcomes. Hui Zheng (2012) identified the lag period to be up to a decade. This may be the reason why fixed effects analyses have failed to confirm the association between income distribution and health. In some countries, such as China (Bakkeli, 2016), sufficient time may not have elapsed between exposure to inequality and population health effects. In the United States, after more than three decades of sharply rising income inequality, we are beginning to witness the reversal of life expectancy among white Americans (Case and Deaton, 2015). White Americans are now dying at alarming rates from premature causes of mortality –notably drug overdose, suicide, and alcoholic liver diseases – that collectively point to despair and disillusionment.

In short, there is still much to debate about the population health impacts of income inequality. During the decade since Zimmerman (2008) called for expanding the scope of inquiry to embrace the role of economic institutions and other contexts in which social policies operate, others have echoed his suggestion. For example, David Coburn (2015) has argued for more focus on the causes and not just the consequences of income inequalities, e.g. by examining the rise of neoliberalism and its impacts on population health. Richard Eckersley (2015) argues for a greater emphasis on the role of culture. Clearly, expanding our focus to embrace more complexity is going to pose an even greater methodological challenge. In the words of Glymour, "the grass is not necessarily greener" on these new pastures.[1] All of this leads us to predict that the big themes of income inequality and social stratification will continue to engage our section for the foreseeable future.

4. Social capital and population health

A second major theme to emerge in the social epidemiology section

[1] Somewhat ironically, the "big picture" theorists who have critiqued income inequality research on methodological grounds often advocate replacing income distribution with their favored variables – e.g. welfare regimes, democratic institutions, or consumer culture – for which there is less evidence, at least in the sense of being subjected to a comparable degree of methodological scrutiny that income inequality studies have received.

in the last 15 years is the investigation of social capital as a determinant of population health. Over time, two distinct streams have developed in the conceptualization and measurement of social capital: the social cohesion approach and the social network approach (Moore and Kawachi, 2017). Regardless, the common definition that researchers from both streams seem to agree upon is that social capital consists of the resources that individuals and groups can access through social connections. The cohesion approach tends to emphasize resources such as group solidarity, maintenance of norms, and the ability of the group to engage in collective action for mutual benefit. These are usually assessed through survey questions that inquire about trust in other people, perceptions of belonging, as well as actual behaviors such as civic or social participation. By contrast, network approaches tend to rely on formal social network analysis methods to measure social resources and networks. In both approaches, social capital is seen as an ecological-level property (e.g., interpersonal, organizational, neighborhood and societal) with individual-level health consequences (Moore and Kawachi, 2017).

In the social epidemiology section, we have featured empirical studies of social capital rooted in both the cohesion approach and the network approach. Our section has also spotlighted the ongoing debate about whether the two approaches are, in fact, drawing on the same underlying construct, i.e. resources derived from social connections. For example, Crapiano and Fitterer, 2014 contend that trust measures are conceptually distinct from social capital, in that they are imperfect proxies for personal social networks. Our personal view is that this privileges the network approach to social capital, i.e. it excludes anything from the definition of the concept that is not a direct measure of "personal social networks", whereas it seems to us that the stock of trust within a network fits squarely within the definition of a "resource embedded in social relations". Trust lubricates the exchange of favors (reciprocity) between members of a group, and facilitates collective action for mutual benefit. Perhaps a way forward is for future investigators to isolate the distinct components of social capital and report them separately – e.g. "network-based social support", "social participation and informal socializing", "social cohesion", and so on.

A productive direction in social capital research has sought to extend the concept to different social contexts. Following Robert Putnam's (2000) influential lead, the initial wave of studies applying the concept to population health focused on national level or (U.S.) state-level indicators of social cohesion (e.g., Subramanian et al., 2001). The next wave of studies drilled down into neighborhoods, following the example set by groundbreaking studies such as the Project on Human Development in Chicago Neighborhoods (Lochner et al., 2003). Since then, studies have begun to examine social capital in diverse (and progressively more micro-level) settings such as the workplace (Oksanen et al., 2008; Tsuboya et al., 2016), and schools (Takakura, 2011). [2]

In parallel with the trend toward studying the effects of social capital in smaller social contexts, more studies are beginning to grapple with the question of how to intervene in communities to build social capital. Unlike the case of income inequality where it would take a regime change to conduct an experiment on redistribution, in the field of social capital, researchers are beginning to directly manipulate social capital in an attempt to improve health outcomes, and are also implementing rigorous methods to handle measurement issues. Ichida et al. (2013) reported an intervention by one municipality in Japan to boost social capital (trust and social participation) by opening a dozen community centers – called "*salons*" in Japanese – designed to encourage older community residents to congregate and socialize. As the intervention was not randomized, the participation of residents in salon activities was likely to be confounded by baseline differences in health status, sociability, etc. Hence the researchers adopted an instrumental variable estimation strategy, utilizing the inverse of the distance between each resident's dwelling and the nearest salon as the instrument. The results of the IV analysis suggested that participation in the newly-opened community salons was associated with both a significant improvement in trust of neighbors over time, as well as the participants' self-rated health.

Community-based interventions that leverage social capital to promote health are also emerging in low- and middle-income (LMIC) countries. Pronyk et al. (2008) conducted an intervention in rural South Africa that combined group-based microfinance with participatory gender and HIV training in an attempt to catalyze changes in solidarity, reciprocity and social group membership as a means to reduce women's vulnerability to intimate partner violence and HIV. After two years of conducting a cluster-randomized trial in eight villages, both cognitive and structural dimensions of social capital were improved in the intervention villages, demonstrating that social capital can be exogenously "built". In western Kenya, Bisung & Elliott (2014) theorized that village-level social capital is a major facilitator of collective action for community-based watershed management and sanitation. Public water supply and sanitation are examples of classical collective action problems – i.e. both are public goods with external benefits, eliciting the dilemma of cooperation (i.e. should an individual cooperate with others at some personal cost – or sit back and enjoy the "free" fruits of other people's labor?). As Bisung et al. (2014) argue, barriers to the provision of these public goods are not solely rooted in financial scarcity; they also depend on community stocks of trust, norms of reciprocity, and collective efficacy.

Perhaps the most compelling application of social capital in public health to date is in the area of disaster resilience, e.g. after the 2007 earthquake in Pisco, Peru (Flores et al., 2014), the 2008 floods in Morpeth, England (Wind and Komproe, 2012), the 2010 Deepwater Horizon oil spill (Rung et al., 2017), or the 2011 Great Eastern Japan Earthquake and Tsunami (Aldrich and Sawada, 2015). Disasters are becoming both more frequent and more damaging in recent decades as a result of both man made climate change and the increasing settlement of populations in disaster-prone areas. As Aldrich argues, recovery in the wake of disaster is not simply a matter of getting "stuff" (water, medical supplies) to stricken areas; the local stock of social capital also matters. Strong social bonds in the community serves as a form of "informal insurance" through which residents assist each other during the recovery process. Communities with social capital also have more "voice" to lobby for federal assistance to affected areas (Aldrich, 2012). [3]

5. Conclusion

As we commemorate the 50th anniversary milestone of the journal, it is worth reflecting on the fact that the Social Epidemiology editorial office is an *arriviste* among the other established offices of the journal. Our office was established in 2001, and by doing so, Social Science & Medicine was the first major journal to publicly recognize the existence of a field called "social epidemiology". The timing was fortuitous because the field had struggled for recognition in the preceding decade. In the inaugural editorial to mark the establishment of our office, Kawachi offered the following sentimental anecdote:

> "Ten years ago, I advised one of my doctoral students at the Harvard School of Public Health to declare "social epidemiology" as one of her fields of concentration on her prospectus. Within 24 hours, the Chair of the school-wide Committee on Admissions and Degrees (who was an epidemiologist) shot back a curt note, demanding to

[2] It is interesting to reflect that the research on social capital "started big" and subsequently moved in the direction of drilling down deeper into ever smaller social contexts. This shift seems to parallel the attempts to reconcile the two strands of research in social capital, viz. the "cohesion approach" and the "network approach". In particular, the methods to study social networks are more tractably deployed in smaller setting such as workplace and schools.

[3] Disasters can also act as catalysts for increased community solidarity.

know what was “social epidemiology”, and whether such a thing existed.” (Kawachi, 2002, p. 1739)

The timing of the establishment of the new office at SSM was also fortuitous because it happened just a year after the publication of the first textbook in the field, aptly titled “Social Epidemiology” (Berkman and Kawachi, 2000). To our younger colleagues working today, that might not seem like a big deal. After all, plenty of researchers (some of them highly eminent) had already been practicing social epidemiology for their entire careers. They simply hadn't chosen to brand themselves in that way. Nevertheless, for an early career epidemiologist working in the 1990s, it was important that the legitimacy of one's chosen field should be recognized by the broader community. It was not so long ago that eminent epidemiologists seriously questioned whether problems such as poverty should be even considered a legitimate subject of inquiry by epidemiologists (Rothman et al., 1998). For researchers early on in their career aiming for promotion and progression, it mattered that they could give a name to the discipline they had chosen to work in, and to have a venue to publish their work.

We are unable to do justice to our vibrant field in the space of a brief overview. We did not have the chance to spotlight other promising emerging themes in our section – such as stigma, intersectionality, the growing evidence on the social determinants of health in LMIC settings. When Galea and Link (2013) published their article, “Six paths for the future of social epidemiology” (alas in the *American Journal of Epidemiology*, not SSM), the accompanying commentary asked a pointed rhetorical question: “Isn't all epidemiology social?” (Kawachi, 2013). In the short space of 15 years, the field could be said to have “arrived” at last.

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