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Contemporary Epidemiology: A Review of Critical Discussions Within the Discipline and A Call for Further Dialogue with Social Theory

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Abstract

The discipline of epidemiology, which holds major influence on public health policy as well as on clinical medical practice, has in recent decades to a large extent been concerned with the identification of factors and markers of risk for disease. Much health information and intervention is thus informed by a wealth of studies on a variety of risk factors, of which the individual is encouraged to keep informed and to be responsible about. Meanwhile, risk factor epidemiology has been subject to intense debate, both within and outside the discipline. The following review offers an overview of critical intradisciplinary debates. It then opens discussion on three partially overlapping areas where social theory has been called upon to contribute to epidemiological inquiry, namely analysis of macro-social determinants of health and disease, of categories of human difference and of embodiment. The review ends with, and is motivated by, a plea for further integration of and dialogue between epidemiology and social theory.

Introduction

The discipline of epidemiology, which holds major influence on public health policy as well as on clinical medical practice, has in recent decades to a large extent been concerned with the identification of factors and markers of risk for disease. Much health information and intervention is thus informed by a wealth of studies on a variety of risk factors, of which the individual is encouraged to keep informed and to be responsible about. Meanwhile, risk factor epidemiology has been subject to intense debate, both within and outside the discipline. The following review offers an overview of critical intradisciplinary debates. It then opens discussion on three partially overlapping areas where social theory has been called upon to contribute to epidemiological inquiry. These are analysis of macro-social determinants of health and disease, of categories of human difference and of embodiment (i.e. how social structures ‘get under the skin’ and thus affect patterns of health – e.g. Ferraro and Shippee 2009). The review ends with, and is motivated by, a plea for further integration of and dialogue between epidemiology and social theory.

The subject matter of the review is extensive, and the article lays no claims on being in any way exhaustive. It is intended as an introductory overview of debates relevant to social theorists interested in epidemiological knowledge production, as well as to epidemiologists drawn to social theory and/or self-reflexive inquiry.

Epidemiological knowledge in question

Epidemiology, as defined by the WHO (2015), is ‘the study of the distribution and determinants of health-related states or events (including disease), and the application of this study to

the control of diseases and other health problems'. In Moon's words, epidemiologists strive to 'describe, explain, predict and control' disease (Moon et al. 2000, 5). As major causes of mortality in western countries shifted from infectious to chronic illnesses around the mid 20th century, modern epidemiology developed in conjunction with a multifactorial model of disease aetiology. Specific, often bacterial- or virus-oriented, models adhering to infectious diseases were thus supplanted with the assumption that a range of influences or risk factors, metaphorically envisioned as a web of causation, contributes to disease development. Early investigations like the Framingham and Seven Countries studies, largely focused on cardiovascular disease, laid the groundwork, and the discipline's continued trajectory has been supported by synchronic growth of information technology, rapidly growing amounts of data and increasingly sophisticated statistical methods. Today, clinical guidelines informed by epidemiological study, and abundant media reports on what risks should be avoided or considered in the interest of health, have become familiar parts of many contemporary settings.

Within social theory, researchers have framed risk factor epidemiology in various ways. Many have pointed to the central role of epidemiological knowledge in expansion of medical interest, reaching beyond disease to also include risk-of or risk-as disease. This expansion has been related to processes of medicalisation (e.g. Aronowitz 2009; Conrad 2007; Greene 2007; Kawachi and Conrad 1996; Klawiter 2002; Skolbekken 2008), pharmaceuticalisation (e.g. Abraham 2010; Dumit 2012; Pollock and Jones 2015; Williams et al. 2008) and biomedicalisation (e.g. Clarke et al. 2003, 2010). Others have used the Foucaultian concept of biopower (e.g. Dean 1999; Gottweis 2005; Hacking 1991; Helén 2004; Jackson 2003; Lemke 2011; Rose 2007; Shim 2014; Wheatley 2006) and other strands of social theory on risk (e.g. Beck 1992; Douglas 1984; Douglas and Wildavsky 1982; Giddens 1991; Lupton 1999; Lyng 1990) to grapple with epidemiological notions of risk. What is not always forefronted in such theorisation, nor for that matter in mainstream epidemiological literature, is the wealth of divergent views, often incorporating calls for social theory, which can be found within the discipline itself.

In many settings, epidemiological calculations still tend to have an air of impeccably standardised and neutral quantification. Still, Latour's (1987) description of the double-sided Janus face of science, where one side displays neat, hard facts and the other reveals the messiness of controversies and contingencies, is very apt here. Critical discussions abound, as in any living field of study, and waves of debate have at times run notably high. During the 1990s, Poole and Rothman referred to the 'epidemiology wars' (Poole and Rothman 1998), while McKinlay stated that 'business as usual simply cannot continue' (McKinlay 1993, 109), and Susser argued that epidemiologists 'need either to adopt a new paradigm or face a sort of eclipse' (Susser 1998, 609). As major threads of such debates can be traced into present times, our review begins there.

1990s: on subject matter, methodology and notions of paradigmatic change

Epidemiological writings from the 1990s can, as Camargo et al. (2013) observe, be divided into two major groups. The first offers an abundance of calculations on various risk factors for disease, while the second voices criticisms towards the former, in debates construed by Shy (1997) as a legal battle between a mainstream defendant and a critical prosecutor. As the topic of the present review is critical debate, focus here lies on arguments of the latter.

A reiterated critique concerned mainstream epidemiology's focus on the individual, as its unit of observation, analysis and intervention. Numerous authors observed that although epidemiology according to basic definition was to be regarded as population medicine, it still tended to focus on the body, lifestyle, behaviour, sex/gender, race/ethnicity and perhaps the personality, emotional state or socioeconomic status of the single person (e.g. Anonymous 1994; Dean and

Hunter 1996; McKinlay 1993; Poland et al. 1998; Susser 1989; Syme 1996). Consequently, Susser observed, epidemiology had 'little regard for the social structures and social dynamics that encompass [individuals]' (Susser 1998, 609). While a number of epidemiologists did pursue analyses of social determinants of disease (e.g. Kaplan 1999; Krieger et al. 1993; Lynch et al. 1997; Marmot and Wilkinson 1999; Marmot et al. 1991; Townsend and Davidson 1982), authors like Syme (1996) and Krieger (1992) noted that categories of socioeconomic status and/or class had generally been omitted or de-prioritised in epidemiological research, particularly in the United States. Meanwhile, only a minority of studies considered health effects of phenomena like racism, sexism or environmental destruction (e.g. Dean and Hunter 1996; Krieger and Sidney 1996; Krieger et al. 1998; McMichael 1995). As an example of how larger contextual phenomena were displaced from study, Wing (1994) brought up smoking, the establishment of which as a major risk factor was typically considered one of epidemiology's great successes. Wing observed that in epidemiological study, as in related prevention strategies, smoking was typically construed as a habit of individual consumers. Other parts of the scenario, like those involving tobacco industry, agribusiness or social circumstances conducive to smoking, did not provoke as much interest. As a result, smoking did indeed decrease in the western world, especially among the more privileged, while in poorer countries it increased. Despite the alleged success of epidemiology, then, many people still became ill due to smoking, while health disparities were exacerbated. Shy's (1997) prosecutor wrote, accordingly:

It is one thing to identify the risk factors for lung cancer in individuals and another to understand what changes occurred in society to result in an epidemic of lung cancer in the 20th century [...] Academic epidemiology failed to study the underlying societal factors that are causes of disturbances in health at the population level (Shy 1997, 480f).

Such underlying questions should be addressed, many argued, through investigation of macro-level, socioeconomic and/or power-related societal parameters (e.g. McKinlay and Marceau 2000; McMichael 1995; Muntaner 1999; Pearce 1996; Poland et al. 1998).

For Shy's defendant, i.e. mainstream epidemiology, such phenomena were not considered part of epidemiological subject matter. Critics argued, however, that they should be (Shy 1997; Syme 1996), some by drawing attention to earlier periods in the history of the discipline, notably 19th century Europe where epidemiologists like Chadwick, Farr, Engels and Virchow agreed that issues of population disease were societal in nature (Krieger 1992; Wing 1994). While political motivations of both theorisations and prevention efforts may have differed, focus was directed towards the societal level. Over the 19th century this orientation was progressively overshadowed by germ theory, until by the mid-20th century mortality due to chronic illness rose to prominence together with the framework of multicausality. This model re-opened the case, at least in principle, for stronger inclusion of social parameters in analyses of disease causation. Such work was indeed done, not least within social epidemiology (i.e. the evolving sub-discipline concerned with social determinants of disease), but as argued by Krieger (1994), attention still tended to be directed towards the factors or strands of the web of causation deemed 'closest' to the disease outcome. These were typically biological agents or lifestyle factors relating to bodies and behaviours of individuals. McKinlay (1993) commented that epidemiology thereby supported tendencies towards blaming the victim of disease.

These discussions took place on several levels, involving questions of epistemology, ontology, knowledge application and the issue of whether knowledge could or should be neutral or value-free (e.g. Camargo et al. 2013). Regarding the point of neutrality, some explicitly argued for adherence to such ideals (Savitz et al. 1999; Stolley 1985). Others drew attention to the contingency of all knowledge (Brown 1993; Krieger 1994, 1999; Moon et al. 2000; Wing 1994).

While arguing against what they perceived as a general absence of social theory in epidemiology, authors like Krieger (1994), Pearce (1996) and McKinlay (1998) asserted that this absence did not equate to non-existence of underlying assumption or values but merely meant that these were not made visible, or conscious. Along similar lines, some pointed to limitations and/or values inherent in epidemiological research methodologies themselves. According to Schwartz and Carpenter (1999), these often tended towards 'providing the right answer for the wrong question', for example, by studying causation of homelessness through looking for traits differentiating homeless people from non-homeless ones, leaving other questions unasked and thus unanswered. To McKinlay's mind,

[l]ike good servants, these epidemiological approaches and methods are always available, do whatever is asked of them, but seldom question the underlying reasons (McKinlay 1998, 370).

While McKinlay (1993) noted that the randomised controlled clinical trial, the 'gold standard' among epidemiological methods, did not lend itself to study of macro-level forces, Wing (1994) and McMichael (1995) observed that within the framework of mainstream epidemiology, efforts to investigate social context almost inevitably became fraught with issues of confounding, i.e. of ambivalent or mixed influences requiring disentanglement for the reliable measurement of individual factors:

it is only by excluding the context and focusing on particular factors considered independently of historical conditions that science can produce objective knowledge (Wing 1994, 82).

Rather than actually achieving objectivity, however, Wing argued that mainstream epidemiology made a political commitment to the status quo by excluding socioeconomic, political and cultural issues from consideration (also Brown 1993; Krieger 1994).

Alongside these debates, a different set of discussions pointed to other problems or uncertainties involved in the production and interpretation of epidemiological knowledge. In an article based on interviews with leading epidemiologists, Taubes (1995) noted that these seemingly converged in the view that regardless of how carefully or with which mathematical techniques studies were done, epidemiological study almost invariably struggled with error and bias, largely because the vast majority of risk factors accounted for such subtle influences that their significance was difficult to measure. 'We're pushing the edge of what can be done with epidemiology', Rothman stated (quoted in Taubes 1995, 167). Meanwhile, debates surrounding the scientific underpinnings of more or less established risk factors, notably for cardiovascular disease, continued. The controversy surrounding cholesterol was a case in point (e.g. Thompson 2009). On a more general note, some epidemiologists argued that established risk factors could not adequately explain cardiovascular disease trends within and between populations (Kannel and Thom 1984; Nieto 1999; Syme 1996). It is 'somewhat striking', Nieto stated with reference to cardiovascular epidemiology, 'how little new substantive knowledge has been gained in this area in the last four decades' (Nieto 1999, 293). There had been no lack of potential candidates, he added, as already in 1981 Hopkins and Williams (1981) listed 250 possible cardiovascular risk factors, and the number continued to rise. In McKinlay and Marceau's words, then,

Established epidemiology is analogous to a maze (in this case, a maze of risk factors) with no opening or exit in sight (McKinlay and Marceau 2000, 26).

As part and parcel of such debates, several epidemiologists argued for radical renewal of the discipline, and to many an increased inclusion of social parameters was an overarching goal.

Multilevel analyses encompassing collective as well as individual levels were recommended and developed (Diez-Roux 1988, 2000; Susser 1989, 1998), while some argued for incorporation of ethnographic methods and other forms of qualitative study (Anonymous 1994; Dean and Hunter 1996; McKinlay 1993). A few evoked notions of paradigmatic change. While discerning co-existing paradigms within epidemiology and public health, McKinlay (1998; McKinlay and Marceau 2000) argued for increased attention towards collective or macro-social dimensions of disease causation, within a framework primarily concerned with health rather than disease. Susser (1998; Susser and Susser 1996) proposed a new Eco-Epidemiology, aiming to reach beyond the present focus on risk factors towards inclusion of societal as well as individual and molecular levels, all conceptualised as nested within each other in mutual and interactive interrelationship. Going further, while calling for reengagement between epidemiology and social sciences in the 21st century, Krieger (2000, 2001) proposed an ecosocial framework. Within it, study should tend to multiple levels of organisation, be concerned with social as well as biological determinants of disease, investigate processes of embodiment and include social theory.

2000 and onwards: the debates go on

The discipline of epidemiology continues to develop and has, again tending towards polarisation, produced two major lines of study. One is the molecular, fuelled by investigation of genes, genomes and biomarkers, while the other is the social, continuing to grow out of the work of the 1990s (e.g. Galea and Link 2013).

As for the molecular, the Human Genome Project was introduced in Europe as an endeavour towards 'Predictive Medicine' (Rose 2000). Massive investments alongside powerfully communicated narratives of pending ability to predict all sorts of diseases (e.g. Collins and McKusick 2001; Zwart 2009) nurtured expectations of exciting new realms of risk factors. Some time down the track, however, a limited amount of epidemiologically useful information had so far been yielded (e.g. *The Lancet* Editorial 2010; Hayden 2010). In the case of cardiovascular disease, for example, despite the identification of relevant markers, the 'effect sizes per risk allele have been modest' (Lieb and Vasani 2013, 1134), only explaining an 'extremely small increase in risk' (Cappola and Margulies 2011, 90). On a methodological note, Lara-Pezzi et al. remark,

analysis of common multifactorial diseases such as CVD [cardiovascular disease] is hindered by the interdependence of genetic and environmental factors and the difficulties that are inherent in separating the influence of individual factors (Lara-Pezzi et al. 2012, 434).

While recent efforts have included attempts towards development of new cholesterol-lowering drugs (PCSK- and CETP-inhibitors; Dorey 2015; Durrington 2012) and future breakthroughs are possible, to date, then, complexities encountered in fields of genetics, genomics and epigenetics have mitigated against the anticipated 'revolution' (Collins 2010) in (predictive) medicine. So far, molecular epidemiology has not made the study of social parameters redundant, in other words. On the contrary, Meloni (2014) speaks of a social turn in the life sciences, pointing to (epi)genetics as one of the areas of natural science research through which it has become increasingly difficult to separate the biological from the social or cultural. It is debatable, however, how traceable the implications of such a social turn are in epidemiology and related health sciences (e.g. Krieger 2011; Lock 2013).

Meanwhile, the sub-discipline of social epidemiology has grown. A wealth of studies focusing on health inequalities and/or including social factors in explanatory models of health and disease has evolved (e.g. Berkman and Kawachi 2000; Commission on Social Determinants

of Health 2008; Cwikel 2006; Navarro 2007; O'Campo and Dunn 2012; Oakes and Kaufman 2006). According to Galea and Link (2013), it would nowadays be hard to find an epidemiologist claiming that social factors are not relevant to disease causation. Still, scholars argue that current (social) epidemiology has severe limitations, in terms of its faltering ability to provide adequate tools for addressing existing health disparities.

This failure to contribute to alleviation of health inequalities should be met, Harper and Strumpf (2012) argue, by increased focus on what they call answerable questions. They hereby refer to narrowly specified research questions that can be addressed by means of experimental or quasi-experimental methods, preferably randomised controlled trials, enabling epidemiological study to make itself useful to policy-making in concrete and direct ways. Meanwhile, other authors argue against such a narrowing of horizons. Scott-Samuel and Smith (2015) assert, for example, that the dominant 'policy paradigm' that, within the framework of neo-liberalism, prioritises economic growth as an overarching goal, renders it virtually impossible for policy-makers to effectively reduce health inequalities (see also Schrecker and Bamba 2015). Along similar lines, O'Campo and Dunn (2012) observe that the identification and description of health inequalities and their links with various risk factors, which has been a major concern of (social) epidemiology, provides insufficient knowledge on which to base effective solutions (also Muntaner 2013; O'Campo 2003). What is missing, they argue, is adequate inclusion of macro-level societal forces and structures in epidemiological inquiry. Echoing discussions from previous decades, O'Campo and Dunn (2012), like Bauer (2014), Ng and Muntaner (2014), Muntaner (2013), Galea and Link (2013) and Krieger (2011), note that macro-level structures are still largely absent from study, as is social theory. Perhaps confirming the latter, a citation study of the influential *American Journal of Epidemiology* found that the proportion of references to social science journals remained, during the 22-year study period, around 0.2 percent (Oakes 2005).

Commenting on the persisting 'dominance of implicit, rather than explicit, use of epidemiologic theory to inform epidemiologic research', Krieger (2011, 4) observes that this implicit theory typically continues to rest on the ontologies and epistemologies of biomedical and lifestyle approaches. A central feature of both, Krieger notes, is individualism, as the primary causes of disease both on individual and population levels are taken to be biophysical agents, genes and 'risk factors' to which exposure is largely determined by individual characteristics and behaviours. A second feature is reductionism, as explanatory models centred on molecular processes and other mechanisms occurring within biological organisms are typically assumed to sufficiently explicate disease occurrence and distribution at the population level. Accordingly, a web of causation for myocardial infarction printed in a 2004 epidemiology textbook attends to

[i]ndividual-level risk factors identified by biomedical and lifestyle hypotheses and does so with scant attention to the larger societal and ecologic context in which these exposures are produced and distributed, let alone whether the depicted factors are sufficient to explain extant and changing population distribution of disease (Krieger 2011, 154).

Biomedical and lifestyle orientations thrive, Krieger (2011) concludes, in 21st century science. Meanwhile, efforts continue to be made towards further integration of social parameters and theories into epidemiology, for example, through development of Krieger's ecosocial theory (e.g. Buffardi et al. 2008; Krieger 2012; Leslie and Lentle 2006; Yamada and Palmer 2007), Latin American Social Medicine (e.g. de Almeida-Filho 2000; Granda 2008; Krieger 2011; Muntaner 2013; Tajer 2003), theorisation of psychosocial determinants of health and disease (e.g. Marmot 2004; McEwen 2008; Wilkinson and Pickett 2009) and complex systems frameworks (e.g. Diez-Roux 2007; Jayasinghe 2011).

Another line of debate, reminiscent of previously mentioned discussions on epidemiological method (Taubes 1995), concerns conflicts arising from translation of aggregate-level risk, as in probabilistic concepts based on measurements in populations (average causal effects), into risk estimates regarding individuals (individual causal effects). This may perhaps not appear as a point of real controversy, as basic, introductory books on epidemiology emphasise that such translations are indeed problematic (Gerstman 2003; Webb and Bain 2011). As elaborated in seminal work by Rose (1992), who was inspired by Durkheim, it is well known that population averages may obscure considerable heterogeneities of responses between individuals and groups. Imposition of average values on the individual, termed the ‘tyranny of the means’ by Tabery (2011), has long been criticised (Bernard 1957; Hogben and Sim 1953), not least by epidemiologists promoting ‘n-of-1’ design (i.e. studies made on single persons; Guyatt et al. 1986) and/or personalised medicine (Lillie et al. 2011). Similar critiques have been raised in political (Downs and Roche 1979) and biological sciences (Gould 1996a, 1996b; Kaplan and Gronfeldt Winther 2013). Nonetheless, average-level risk continues to be applied to the individual level in a wealth of ongoing clinical and public health practices. Some epidemiologists therefore argue that consideration of individual heterogeneity has not been sufficiently addressed or integrated. Merlo et al. (2013a, 2013b, 2014), Smith and Egger (1998), Pepe et al. (2004) and Levine (2001) emphasise that as tools for distinguishing between individuals who will become ill or not, and thus who should or should not be (pharmaceutically) treated, population-average risk factors are often quite blunt. Most risk factors (social as well as biological) have, in other words, low discriminatory accuracy (Merlo 2014). In the case of cardiovascular disease, Levine (2005) refers to studies where risk factors were found to be almost as prevalent among those non-diseased as among those diseased, while Merlo et al. (2013a, 2013b) stress that neither established risk factors like hypertension nor newly found biomarkers do much to improve cardiovascular disease prediction as compared with estimates based merely on age and sex. Merlo (2014) adds that measurements often used to quantify the burden of cardiovascular disease (population attributable fractions, e.g. Yusuf et al. 2004; Björck et al. 2009) and thereby motivate public health interventions tend to be misleading as they may exaggerate the relevance of risk factors by not considering people who are exposed to the factor but do not develop disease. The adequacy of many risk factors, as bases of prevention strategies and explanations of disease trajectories, is thereby called into serious question. Epidemiologists pursuing this line of argument have handled the question in different ways. Levine (2007) takes a step away from quantitative risk calculation, while Smith’s (2011) solution to the ‘Gloomy Prospect’ of epidemiology is an acknowledgement that probabilistic, or even stochastic, models of prediction are all that remain. Merlo et al. (2003, 2004, 2005, 2009, 2012, 2014) assert, rather, that epidemiology should continue to develop beyond consideration of population-average risk, through multilevel analyses of individual heterogeneity, taking discriminatory accuracy into central account. This is important not least, Merlo argues (also Mulinari et al. 2015a, 2015b), as population averages may stigmatise certain groups by making them appear more homogenous, and different to others, than they are.

These discussions relate, finally, to coexistence of probabilistic and mechanistic forms of knowledge. In epidemiology, mechanistic approaches basically seek to establish causal hypotheses by explaining ‘how something works’ (Broadbent 2011, 49). While this stance relates comfortably, in principle, to biomedical visions of specific aetiology (Mulinari 2014), the rise of multifactorial webs of causation and concomitant focus on risk brought probabilistic approaches into the centre of epidemiology. Measures of probability establish the ‘extent to which an event is likely to occur’ (Oxford Dictionaries 2015), rather than dynamics through which such events are brought about, and resultant lack of attention to mechanism has been a major cause of criticism against probabilistic risk factor or ‘black-box’ epidemiology (Greenland et al. 2004; Ng and Muntaner 2014; Susser 1998). Galea and Link (2013) argue, thus, that epidemiologists must increase their

focus on biological mechanisms of disease causation. Others (Lofters and O'Campo 2012; Ng and Muntaner 2014) promote attention to social mechanisms. Merlo et al. (2012, 2013a, b, 2014) emphasise that probabilistic and mechanistic approaches exist in a relationship of deep tension, expressed in mentioned frictions between population-level and individual-level risks, which has not been resolved or adequately confronted. Noting that much empirical work in (social) epidemiology continues to adopt a probabilistic stance (also Smith 2011), Merlo stresses that this approach remains unable to grasp heterogeneity around averages and that epidemiologists need to step up in investigation of mechanisms of disease causation while incorporating analyses of variance. Furthermore, Merlo continues, the fundamental contradiction between probabilistic and mechanistic approaches needs to be recognised as part of a current state of crisis in epidemiology, and doors should be opened towards alternative forms of medical knowledge.

Engaging social theory

Over the past decades, then, as epidemiologists have debated various aspects of their discipline, reiterated arguments have been made for further integration of macro-level determinants of health and disease and of social theory, as well as of consideration of environmental sustainability, in epidemiological study. The following will open brief discussion on three partially overlapping areas where social science has been called upon to enter into or deepen dialogues with epidemiology, for purposes of theorising structures and relationships of power. These are analyses of macro-level determinants of health and disease, of categories of human difference and of embodiment.

Macro-level determinants of health and disease: political epidemiology and critical realism

Among the many arguments made for increased attention towards macro-level determinants of health and disease in epidemiology (e.g. Commission on Social Determinants of Health 2008; Levins 2000; Theorell 2006), authors like Beckfield and Krieger (2009) and Muntaner (2013; Ng and Muntaner 2014) argue for the development of a political epidemiology informed by critical realism.

Under the heading 'Epi+demos+cracy', Beckfield and Krieger (2009) propose a research agenda under which social epidemiology and political sociology cooperate in study of how political systems and priorities affect population health and health disparities. Beckfield and Krieger note that while epidemiology has mainly been concerned with individual-level associations between social position and health, and social sciences have looked at how political and economic systems affect population well-being, or how categories used to study health inequalities are in themselves part of relations of power, these two bodies of research have rarely engaged in explicit dialogue. Pointing to resultant knowledge gaps in existing literature, Beckfield and Krieger argue that there is much to benefit from combining strengths of sociology with those of epidemiology. Although such inquiry might be sensitive, due to inevitable actualisation of values and ideologies, Beckfield and Krieger argue that it is needed, for the production of knowledge that is practically applicable in efforts towards reducing health disparities:

Power, after all, is at the heart of the matter – and the science of health inequities can no more shy away from this question than can physicists ignore gravity or physicians ignore pain (Beckfield and Krieger 2009, 169).

Muntaner (2013; Ng and Muntaner 2014) similarly argues that furthered understanding of how social production of disease can be changed requires integration of economics, politics

and sociology into epidemiology. Noting that most epidemiological study adopts a fundamentally uncritical position towards existing social structures, Muntaner argues for adoption of the sociological framework of social conflict. Based on a view of society as characterised by inequality, tension and conflicting interests, this framework enables investigation of how unequal power relations are causally linked to unequal distribution of resources and, in turn, to generation of health inequalities.

In response to calls for theory, Ng and Muntaner (2014), like O'Campo and Dunn (2012; Dunn 2012; Lofthers and O'Campo 2012), promote integration of critical realism (also Collins et al. 2015; Scambler and Scambler 2015). A central feature of this theoretical approach, as emphasised by these authors, is its focus on mechanism. In contrast to familiar epidemiological efforts to demonstrate associations between various factors and data, realist epidemiology aims to identify context-mechanism-outcome patterns and thus provide explanations of how macro-social determinants and population health are causally linked. It thus aims to explore 'relational mechanisms such as sexism, racism, heterosexism, ableism, ageism, classism' (Ng and Muntaner 2014, 32) as well as transnational division of labour and historical trajectories of exploitation, while aiming to assist the development of a 'public health imagination' (Ng and Muntaner 2014, 32f) by which personal health problems are understood to be public and political in nature. Such realist focus on mechanism implies, Dunn (2012) adds, that increasing amounts of data and/or statistical precision are not to be taken as the main or only ways to furthered knowledge. Attention should also be paid to theoretically framed analyses of causal mechanisms, resting on qualitative as well as quantitative inquiry.

Categories of human difference

Efforts, as quoted above, to investigate 'relational mechanisms such as sexism, racism, heterosexism, ableism, ageism, classism' (Ng and Muntaner 2014, 32) ideally encompass critical inquiry into how the very categories used to define such relations, within the study of health inequalities (Beckfield and Krieger 2009), can themselves be considered part of power structures. Stratifications along lines of race/ethnicity, sex/gender and class/socioeconomic position are central to (social) epidemiology's study of health disparities, as well as to conceptualisation of exposures or factors of risk. In social theory, inquiry into how difference has been constructed and negotiated in medical science along these categories forms a field in itself (e.g. Birke 2000; Johansson 2004; McClintock 1995; Merchant 1989; Stepan 1996), and corresponding analysis on epidemiological handling of such categorisation can enrich analyses of health disparities with methodological and theoretical as well as self-reflexive insight.

Pollock (2012) investigates how notions of race have been invoked and constructed in relation to heart disease through efforts like the Framingham study, while Epstein (2004, 2007) looks at recent inclusion of populations differentiated along categories of race/ethnicity and sex/gender in clinical trials, arguing that such inclusion does not in itself counteract health disparities rooted in social structure rather than biology. Shim (2000, 2002, 2005, 2010, 2014), on her part, analyses how categories of race/ethnicity, sex/gender and class are used in cardiovascular epidemiology. A wealth of studies point to cardiovascular health inequalities along these lines (e.g. AHA/ASA 2013; Dalstra et al. 2005; Kurian and Cardarelli 2007). Although epidemiologists may have differing understandings of why such disparities exist and how they should be addressed, Shim argues that in epidemiological practice they tend to be dealt with in uniform, routine or 'ritualised' ways. Shim hereby refers to standardisation, a basic way of avoiding error in epidemiological study by weighing results against a standard population. Variables like ethnicity or socioeconomic status are thus compared and adjusted against 'standard differences',

with the aim of making results representative of larger populations and/or teasing out influences from different factors. This practice tends to render health disparities as given, Shim argues, by normalising them and smoothing over the very differences that should be the focus of research (also Lofters and O'Campo 2012). Furthermore, by constructing such categories as individual-level risk factors, as in attributes of individuals and groups rather than dynamics of relationships between individuals or groups, and by modelling public health interventions accordingly, Shim asserts that epidemiology may contribute to perpetuation or even production of social inequalities.

Shim evokes the theoretical frame of intersectionality, the concept brought forward by Crenshaw (1989) and developed by theorists like McCall (2005), Nash (2008) and Choo and Ferree (2010) as a means of thinking beyond categories like ethnicity, gender, class and sexuality as separate, but rather as interacting in multi-layered ways. Intersectionality has been proposed as a theoretical framework for epidemiology and public health (Bauer 2014; Dworkin 2005; Hankivsky 2011; Iyer et al. 2008) as well as for analyses of risk (Collins et al. 2008; Hannah-Moffat and O'Malley 2007; Olofsson et al. 2014). Kapilashrami et al. (2015) suggest, for example, that such a framework can help researchers look towards social dynamics rather than social categories and thus consider structural drivers of inequalities rather than individual-level behaviours. Studies looking at how intersectionality has actually been integrated in the fields of health and risk research find limitations, however (Girtli Nygren and Olofsson 2014; Hankivsky 2012; Kapilashrami et al. 2015; Olofsson et al. 2014). Efforts have been made to integrate intersectionality into quantitative study, explicitly (e.g. Bauer 2014; Hinze et al. 2012; Veenstra 2011) or more implicitly through analyses of heterogeneity within and between social categories (Merlo et al. 2004; Mulinari et al. 2015a, 2015b). It is sometimes argued, however, that qualitative study is more suited for intersectionality studies than quantitative (Girtli Nygren and Olofsson 2014; Schultz and Mullings 2006), and integration of qualitative methods into epidemiology has been promoted for that purpose (Bauer 2014).

Embodiment

As a means to conceptualise continuities and pathways between macro-social conditions and societal relationships on the one hand, and the health status of population groups on the other, Krieger (2001, 2005a, 2005b, 2011, 2012) puts forward the concept of embodiment. This notion needs to lie at the very core of (social) epidemiology, she argues, as an understanding of health disparities requires theorisation of how social structures and experiences become biologically incorporated and manifested in bodies. A central question is thus how patterns of disease distribution can be understood as 'biological expressions of social relations' (Krieger 2001, 672). Such investigation necessarily involves theory, Kreiger emphasises, and she refers to authors like Bourdieu (1984), Merleau-Ponty (1989) and Scheper-Hughes and Lock (1987).

Epidemiological interest in embodiment can be taken as synchronic with what Meloni (2014) calls the social turn in the life sciences, as well as with the vast increase of interest in the body seen within the social sciences over recent decades, springing from problematisation of previously held conceptions of the body as universally constant (e.g. Csordas 1990, 1993; Shilling 2013). While renegotiation of boundaries between the 'social' and the 'biological' may be disconcerting to natural and social scientists alike (e.g. Lock 2013; Meloni 2014), a range of scholarly efforts rise to the challenge (e.g. Ingold and Palsson 2013; Landecker and Panofsky 2013; Oyama 2000). Studies pertaining to embodiment of social determinants of health include the works of Gravlee (2009), Kuzawa and Sweet (2009) and Fausto-Sterling (2008), all looking at how social inequalities become embodied in racialised individuals and groups. The inquiry of

Fausto-Sterling et al. (2012) into social aspects of development of sex-related differences in small children also argues for applicability to studies on health disparities. Another example is the investigation by Walters et al. (2011) of the embodiment of historical trauma among Native Americans, proposing that ‘bodies don’t just tell stories, they tell histories’ (Walters et al. 2011, 179; also Hornborg 2005, 2010). The concept of embodiment provides ways, then, of moving beyond notions of disease distribution as solely reliant on behaviours and characteristics of individuals, while emphasising bodily engagement between humans and with the biophysical world and tracing connections between inner and outer realities (Krieger 2011).

Conclusion

As critical debates go on, the discipline of epidemiology continues to provide scientific underpinnings for public health interventions and clinical practices around the world. While some epidemiologists point to basic limitations in the explanatory and predictive power of risk factors as they are currently often handled (Levine 2005; Merlo 2014), many comment on persistent insufficiencies of, and difficulties in, integration of social structure and power in epidemiological study (e.g. Bauer 2014; O’Campo and Dunn 2012). Social theorists interested in health disparities and/or epidemiological knowledge production thus have many invitations to respond to.

The many (possible) interfaces between social theory and epidemiology have not been given justice in this short review. For one thing, the briefly mentioned writings on medicalisation, biopower and other social theory on risk (e.g. Abraham 2010; Beck 1992; Clarke et al. 2010; Conrad 2007; Douglas 1984; Dumit 2012; Greene 2007; Klawiter 2002; Lemke 2011; Lupton 1999; Shim 2014) show that far from only being part of the subject matter of critically oriented epidemiology, relations and dynamics of culture and power can be seen as intrinsic to the discipline’s very enactment. Here it should be noted that Shim’s (2014) ethnographic study of epidemiological knowledge production, of practices and discourses of epidemiologists in action, is one of but a few in a field that ought to be further developed. Questions of how local and global cultures intersect in creation and use of epidemiological knowledge should also be addressed, through further study of varying ways in which epidemiological findings inform public health policy in different countries (e.g. Vallgård 2008, 2010). Another area in which social theory can add to epidemiological fields of interest lies in exploration of alternative models of explanation and treatment of risk and disease sought in complementary or alternative medicines around the world (e.g. Diehl and Eisenberg 2000; Eardley et al. 2012; Fischer 2012; Hess 2004). Having said that, the areas mentioned above – analyses of macro-level determinants of health and disease, of categories of human and health difference and of embodiment – do represent arenas for potentially fruitful further collaboration and dialogue between epidemiology and social theory. In the interest of understanding and addressing health disparities in the world, we believe that such collaboration is key.

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Anne-Christine Hornborg is a Professor in History of Religions at the Centre for Theology and Religious Studies, Lund University. Hornborg has written several publications concerning indigenous worldviews, ecology and religion, healing and cultural trauma based on extensive fieldwork on Cape Breton, Canada, among the Mi'kmaq First Nation, as well as in Tonga and Peru. Hornborg has also applied theories and methods from the interdisciplinary field of Ritual Studies and from Anthropology in her examination of new ritual practices in late modern Sweden. Her discussions on neospiritual laymen therapy and coaching as products of neoliberal cosmology and the market in contemporary Swedish society has also attracted attention in a wider public.

Note

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