CHAPTER 26

Health Demography

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WHAT IS HEALTH DEMOGRAPHY?

In their 1992 book *The Demography of Health and Health Care* (part of the Plenum Series on Demographic Methods and Population Analysis), Louis Pol and Richard Thomas declared there was neither a widely accepted definition of *health demography* nor a distinct group of professionals who called themselves health demographers (Pol and Thomas 1992). They went on to note that most of those involved with health demography were not professionally trained demographers, but epidemiologists, sociologists, and other social scientists who happened to apply demographic concepts in the health field. Pol and Thomas provided the following definition of health demography:

> Health demography is perhaps best defined as the application of the content and methods of demography to the study of health status and health behavior... Thus health demography concerns itself with the manner in which such factors as age, marital status, and income influence both the health status and health behaviors of populations and, in turn, how health-related phenomena affect demographic attributes (Pol and Thomas 1992: 1).

A decade later, there is still no organized body of professionals who call themselves health demographers. On the other hand, demographers and epidemiologists (along with sociologists, medical geographers, and even a few economists) have begun to converge on an emerging field that has been referred to as "the social determinants of population health" (Marmot and Wilkinson 1999; Berkman and Kawachi 2000; Eckersley, Dixon, and Douglas 2001). Frustrated with the progressively narrow concern for identifying "risk factors" for disease (including the search for genetic markers) as a way of understanding and addressing problems of public health, some epidemiologists have called for broadening the usual inventory of determinants of health to include

variables such as socioeconomic status, race/ethnicity, and social support, all of which are intended to capture some aspect of social organization rather than simply characteristics of individuals (Berkman and Kawachi 2000). By advocating for the movement of these classic demographic variables to a central role in research on the health of populations, these *social* epidemiologists (as they call themselves) represent one end of the potential spectrum of health demographers.

At the same time, social demographers have rediscovered the role of social context in human behavior, and "with encouragement from their sociologic neighbors, have begun to recognize the severe shortcomings of a purely variable-based view of the world" (Palloni and Morenoff 2001: 140). The foundation for a viable discipline of health demography therefore rests in the convergence of two disciplines, demography and epidemiology, particularly those branches of each that emphasize the effects of social contexts and group membership.

As will be shown below, demography and epidemiology share a common historical origin dating back to the 17th century. However, during the course of their respective development and refinement, the two fields have diverged and become specialized to the extent that few professional epidemiologists today would claim to be a demographer, and vice versa. Practitioners in each field are trained in separate and distinct programs, learn from different textbooks, resort to different analytical methods, attend separate conferences and professional meetings, and publish their work in specialized journals (with notable exceptions such as the interdisciplinary journal, *Social Science & Medicine*).

Even when they are analyzing the same phenomena in the field of population health, for instance, the mortality crisis in Eastern Europe following economic transformation, or racial and ethnic disparities in birth outcomes, epidemiologists and demographers tend to adopt different study designs, different conceptual frameworks to select and define variables, different assumptions and tests of causality—indeed, different *languages* to define the problem and describe the data. Nonetheless, there are more apparent similarities than differences between the concerns of the two disciplines when it comes to tackling the fundamental questions of population health. In this chapter we will examine the differences and contrasts between epidemiology and demography in addressing the substantive concerns of health demography. We will also point to intersections and areas of convergence between the two that hold promise for delineating health demography as a viable interdisciplinary endeavor.

HISTORICAL LINKS BETWEEN DEMOGRAPHY AND EPIDEMIOLOGY

Epidemiology and demography share a common linguistic heritage (Rockett 1999). The term *epidemiology* derives from the Greek roots *epi* (upon) and *demos* (people) and *logos* (study). Meanwhile, *demos* and another Greek root, *graphein* (to write, draw) combine to form the term demography. Given these roots, it is not surprising that the two disciplines share a common agenda centered on the study of *populations* as opposed to individuals. Interestingly, the population focus of epidemiology became progressively obscured with the rise in popularity of so-called *risk factor* epidemiology (concerned with identifying individual causes of specific diseases) in the mid-20th century and, more recently, molecular epidemiology (concerned with identifying individual genetic susceptibility to disease).

In addition to their shared linguistic heritage, however, epidemiology and demography also share a historical past. John Graunt (1620–1674) is credited with laying the groundwork for both epidemiology and demography when, for the first time, he demonstrated systematic regularity in births and deaths across age, gender, and geographical areas (Graunt 1662 [1975]). William Farr is considered to be one of the intellectual founders of both epidemiology and demography. Indeed, prior to 1850, what we recognize today as distinct and separate concerns of epidemiology and demography were very much interwoven (Susser and Bresnahan 2001). For example, questions concerning the prevalence of diseases and rates of mortality were seldom divorced from considerations of the living conditions of the population as well as changing population composition. Similarly, beginning with Malthus, questions of population change were seldom considered in isolation from the effects of such change on population health.

Susser and Bresnahan (2001) trace the rift between modern epidemiology and demography back to the identification of the tubercle bacillus (1882) and the rise of germ theory. Parallel with the ascendancy of germ theory, the focus of epidemiologists moved away from the study of populations toward the identification of specific pathogens that caused specific diseases. The separation of epidemiology from demography was further heightened during the era of chronic disease epidemiology, which stretched from the identification of cigarettes as the cause of lung cancer (circa 1950) to the end of the 20th century. As methods for investigating risk factors for individual diseases became established in modern epidemiology, ''demography gradually disappeared from epidemiology textbooks and training'' (Susser and Bresnahan 2001: 13). As for demography, ''the study of health has played a small role in demography,'' despite the shared history of the use of population-based mortality data by demographers and epidemiologists (Weinstein et. al. 2001: 312).

Despite more than a century of divergence between the disciplines, however, the concepts and methods of demography have never ceased to be relevant for those seeking to understand the determinants of population health. As Pol and Thomas (1992) argued, there is hardly an aspect of demography that does *not* have some relevance. Demographers and epidemiologists use the same tools to define and measure population health, for example, life tables, direct and indirect standardization of mortality rates, disability-adjusted life years, and so on. Other demographic variables, such as fertility, migration, and population characteristics (size, distribution), have numerous implications for population health. Finally, both epidemiologists and demographers concur that major compositional characteristics of the population, e.g., age, sex, race, socioeconomic status, religion, and family structure, are among the most fundamental determinants of population health.

Two trends in the late 20th century—the aging of the population and the widening disparities in health across socioeconomic groups—have encouraged the gradual reengagement of the two disciplines. As the population ages, demographers have turned to health itself as an outcome, including implications for health care need and utilization. This increased concern with health as an outcome of interest to demographers has been paralleled by a renewed interest from epidemiologists in the *population*-level and social determinants of health (Berkman and Kawachi 2000; Weinstein, Hermalin, and Stoto 2001). In particular, practitioners of the emerging (or perhaps more accurately, resurgent) subdiscipline of epidemiology, who call themselves *social* epidemiologists, now acknowledge the limitations of a narrowly biomedical perspective in understanding the

determinants of population health (Berkman and Kawachi 2000). The movement of social epidemiology has brought about an expanded understanding of the determinants of population health, including variables such as socioeconomic status, family structure, and residential segregation. Health demography increasingly involves an integration of the substantive concerns, concepts, and tools developed in both social epidemiology and demography.

SUBSTANTIVE ISSUES

Social Epidemiology and Demography: A Common Agenda

A definitive program of research for health demography would be premature, given the lack of any training program, textbook, or professional association to represent such a field. Nevertheless, we have identified some basic principles that guide the practice of both demographers and social epidemiologists investigating the social determinants of health. We discuss each of these in turn:

1. Focus on population health, as opposed to individual health.
2. Recognizing the importance of contextual influences on health (including multilevel approaches to study design and analysis)
3. Adopting a life-course perspective on health.
4. Concern for integrating both biological markers and psychosocial pathways in studying the determinants of health.

The Population Health Perspective

Prior to defining what we mean by a "population perspective," a word or two is warranted on the differing approaches of demographers and epidemiologists toward studying "health." As Jack Caldwell (2001) noted:

> Demography has maintained its primary focus on population, births, and deaths. All are definable within a fairly high degree of precision, a criterion about which demographers feel strongly . . . . When demographers purport to write on health, most of their output is usually on mortality change. One reason is that these conditions cannot be defined exactly, a situation that has been worsened by WHO's all-inclusive definition of *good health*. Another reason is the source of data. If demographers work alone through censuses or surveys, they must depend upon self diagnosis or the reporting of symptoms by respondents, and such reporting is often inaccurate (22).

Caldwell's skepticism notwithstanding, a growing number of demographers have been turning their attention toward a broader definition of health beyond simply counting the dead (or living). Without going to the opposite extreme of admitting everything under the WHO definition, health demographers have increasingly turned to more complex outcomes such as cause-specific mortality, disability, self-rated health, health services use, and health behaviors. Problems remain with the accuracy of self-reported health outcomes, but in many large-scale population-based surveys, both demographers and epidemiologists have begun to complement self-reports with biological markers of physiological change (Goldman 2001). Some of these biomarkers (such as salivary cortisol measurements) have only recently become widely available.

With regard to the *focus* of analysis, Caldwell (2001) again drew a sharp distinction between demographers and epidemiologists:

> Demographic analysis tends to seek background or fundamental influences—using social and economic data—not on the individual but on entire societies.... Epidemiologic studies are the most population-based of all medical research, but nevertheless, they are not usually embedded in whole populations seen over long periods of time in their social and economic context (31).

Happily, social epidemiologists have heeded this critique. Indeed, one of the guiding principles of social epidemiology (articulated in the first textbook with that title (see Berkman and Kawachi 2000) is the recognition that individuals are embedded in societies and populations, as well as in history. Applying the population perspective to epidemiological research means asking "Why is this *population* healthy, while another is not?" As Geoffrey Rose (1994) pointed out, the answer to that question may be different from the question that is typically posed in risk factor epidemiology: "Why is this *individual* healthy (or sick), while someone else is not?" For example, the classic case-control study design in epidemiology in the 1950s and 1960s led to the demonstration that individual smokers were at 15 to 20 times the risk of developing lung cancer compared to nonsmokers. However, no case-control study could have succeeded in identifying the correct cause of lung cancer *if everyone in the population had smoked*.

The classic epidemiological approach of identifying individual risk factors falls short both conceptually and methodologically in the task of explaining population patterns and distributions of health. For example, over 300 individual risk factors have now been identified for coronary heart disease. With knowledge of these individual risk factors, epidemiologists can now predict who is at increased risk of developing a heart attack in the future. Such an approach is exemplified by the Framingham Risk Charts. However, this individually oriented approach cannot tell us why some *populations* have much lower rates of heart attack compared to others, for example, why Japanese men have much lower rates of heart attack than American males, even though they smoke twice as much. The answer to the population question requires a population perspective, including knowledge of the distribution of risk factors in the population, as well as the interactions between individual risk factors and with potential contextual influences. Lacking an explicit population perspective, risk factor epidemiology is ill equipped for investigating what Caldwell called the "background or fundamental influences" on health.

The Geographical and Contextual Perspective

Both demographers and social epidemiologists now recognize that the determinants of health operate at multiple levels or contexts. This multilevel approach to population health is crucial not only to health demography, but it is also reflected in conceptual and methodological developments in allied fields, including ecoepidemiology (Susser and Susser 1996a, 1996b; Schwartz, Susser, and Susser 1999), medical geography (Jones and Moon 1993), and medical sociology (Macintyre 2000; Macintyre and Ellaway 2000). Medical geography, in particular, has always been concerned with the role of places and localities (Jones and Moon 1993, 1987; Moon 1990; Kearns 1993). While geographical variations in population health status have been used in epidemiology for descriptive and planning purposes, geographical contexts have seldom been part of the explanation for health status and health disparities, at least until recently.

Indeed, contextual influences on population health may be place-based (e.g., the impact of residing in particular geographical localities), or they may be defined by some other extraindividual, *social organizational* characteristics (such as cultural norms, political systems, labor market structure, population dynamics) that could be spatially or nonspatially described.

Historically, interest in geographical variations in health, and in ecological variables in particular, waned in the social sciences following the powerful demonstration of the ecological fallacy by Robinson (1950) and others. Also, in the health sciences, analyses involving ecological variables became discredited along with the emerging dominance of risk factor epidemiology (Macintyre and Ellaway 2000). For example, ecological analyses were given scant attention (a mere two mentions in the index) in seminal textbooks such as the first edition of Rothman's *Modern Epidemiology* (1986). Unfortunately, in their distaste for committing the ecological fallacy, epidemiologists threw the proverbial baby out with the bath water. Indeed, the problem was not ecology. Rather, the real issue that Robinson (1950) brought to the table was the genuine risk of making individual inferences based on ecological associations. With such distinctions not being clearly articulated, a separate fallacy, termed the *atomistic or individualistic fallacy*, has been coined to refer to the epidemiologist's tendency to assume that health was solely determined by individual risk factors and behaviors (Schwartz 1994; Diez-Roux 1998). Within the social sciences, this trend has been reversed with the renewed interest in contextual influences on health, led by medical geographers, demographers, sociologists, and social epidemiologists (Kawachi and Berkman 2003).

Health demography, as an interdisciplinary subject, should be concerned with the constitutive role of places and contexts (whether defined as neighborhoods, workplaces, political systems, or regional economies). This would entail defining "area/place effects"; identifying a typology of contextual effects; and developing a research and training agenda that emphasizes the notion of contextual heterogeneity. Indeed, health scientists pursuing what may be called the "social determinants of health" research within public health have provided a basis to develop these ideas.

To begin with, area—or place—effects refer to the health effects of variables that tell us something about the places or contexts, and not simply about the individuals who inhabit them. Macintyre (1997) provides a useful distinction for considering place-effects, referred to as collective and contextual place-effects.

A collective effect refers to aggregated group properties that exert an influence on health over and above individual characteristics, for example, living in areas with a high proportion of people who have certain individual characteristics (e.g., based on age, social class, income, or race). A contextual effect, meanwhile, reflects the broader political or institutional context, for example, the presence or absence of opportunity structures that are intrinsic to places, such as the presence of infrastructure resources or the economic and legal policies of states. These variables have also been referred to as "integral" variables.

Meanwhile, by putting the notion of contextual heterogeneity at the center of the "social determinants to health," medical geographers have provided a useful framework, drawing on multilevel methodological perspectives (Subramanian, Jones, and Duncan 2003). At the most basic level, operationalizing the idea of contextual heterogeneity requires us to distinguish compositional (individual) explanations from contextual (including collective) explanations of geographical variations in health outcomes.

The compositional explanation for area differences would ascribe the variations in health outcomes to the characteristics of individuals who reside in them. If this is true, then similar types of people (based on their age, sex, race, socioeconomic status, and so on) will experience similar health outcomes no matter where they live. On the other hand, if place makes a difference, then similar types of people can be expected to achieve different levels of health depending on where they live.

We can, and should, however, go beyond the simplistic notions of "context and composition." If places make a difference for population health outcomes, then the degree to which they may matter can be anticipated to be quite different for different types of individuals. Such contextual heterogeneity could be manifested in at least two ways. First, place variations may be greater for one population group than another. Thus, if between-context differences are greater for low (compared to high) socioeconomic status (SES) groups, this would suggest that where low SES groups reside is crucial for their health, while for high SES groups it may not really matter where they live. The second dimension related to contextual differences is an intrinsic interest in monitoring places. Thus, for instance, places that are worse off for low SES may or may not be worse off for the high SES groups, suggesting that the contextual geography of health cannot be summarized in one map; rather the map may vary depending on one's SES.

If contextual differences exist, independent of individual, compositional factors, then explaining such differences using variables that relate to contexts (not individuals) is critical to the development of causal models of contextual effects. In summary, the interest in contextual analysis lies not simply in evaluating health achievements based on "who you are in relation to where you are." Rather, it is posing the question in terms of "who you are *depends* on where you are." The methodological implications for incorporating contextual perspectives to social inequalities in health are discussed in a later section.

The Life Course Perspective

Parallel with the growing interest in the dimension of place and contexts, researchers have increasingly sought to understand the patterns of population health and health disparities along the dimension of *time*. The life course approach relates to how health status at any given age, for a given birth cohort, reflects not only contemporary conditions but also embodiment of prior living circumstances, *in utero* onward (Krieger 2000). Detailed presentations of this perspective have been articulated elsewhere (Kuh and Ben Shlomo 1997; Hertzman 1999).

Three distinct pathways have been hypothesized to be relevant to the life course approach (Hertzman 1999). First, one path includes *latent effects* in which the early life environment affects adult health independent of intervening experience. A frequently cited example is the association between markers of intrauterine development (for example, birth weight) and adult diseases, such as hypertension, coronary heart disease, and cancers of the prostate and breast (Davey Smith, Gunnell, and Ben-Shlomo 2001). However, a growing body of research indicates that factors associated with a child's early life environment, such as maternal attachment, parent-child interactions, and child abuse, have wide-ranging and lasting effects on health behaviors and physical outcomes in adulthood (Taylor, Repetti, and Seeman 1997; Felitti et al. 1998). According to Hertzman (1999):

> Specific biological factors (e.g., low birth weight) or developmental opportunities (e.g., adequate exposure to spoken language) at critical/sensitive periods in (early) life have a lifelong impact on health and well-being, regardless of subsequent life circumstances. The fact that crucial elements of emotional control, peer social skills, language development, and the understanding of relative quantity all have critical periods in the first five years of human life adds biological plausibility to the latency model (86).

A second type of life course effect, referred to as *pathway effects*, posits that the early life environment sets individuals onto life trajectories that in turn affect health status over time. An example is the tracking of socioeconomic disadvantage from childhood through to adult life. Finally, a third type of life course effect, referred to as the *cumulative model*, posits that the intensity and duration of exposure to unfavorable environments accumulate over time and produce adverse effects on health status, in a dose-response manner. For example, the effects of poverty on physical and psychological health are much more pronounced among individuals who suffer repeated spells of economic hardship, as opposed to single episodes (Lynch, Kaplan, and Shema 1997; McDonough et al. 1997). A cumulative life course approach, therefore, lends itself to the recommendation that investigators assess wealth, permanent income, and long-term economic deprivation, as opposed to single-time measures of income or poverty (Williams and Collins 1995).

Integrating Biological Markers and Psychosocial Pathways

The life course perspective discussed in the previous section has led naturally to the search for specific biological *mechanisms* that link early life environments to physical and psychological health outcomes. Demographers and social epidemiologists alike have begun to address the possibility of mapping the linkages between early life circumstances and subsequent health outcomes by incorporating biological markers into study designs. Early life circumstances "imprint" themselves on the central nervous system via brain development and alterations in neurochemistry. In turn, because the central nervous system "talks" to a variety of other physiological systems, including the immune, hormone, and clotting systems, biological measurements of these systems can help to establish the causal chains extending from early life circumstances to differential resilience and vulnerability to disease later in life (Kelly, Hertzman, and Daniels 1997).

Incorporating biological markers into social science surveys is likely to become an important part of the health demographer's research strategy for other compelling reasons. As Goldman (2001) argues, if exposures to different social environments are causally related to health, then scientists ought to be able to demonstrate how stressful working conditions, unsafe neighborhoods, poverty, and racial discrimination, as well as a host of other exposures throughout the life course, express themselves in terms of differences in biological and physiological parameters that matter to health.

Two broad classes of biological markers are of potential interest to health demographers: primary mediators of the stress process and markers of secondary outcomes. The former class of biomarkers is exemplified by the organizing concept of "allostatic load" introduced by McEwen and others (McEwen 1998; McEwen and Seeman 1999). *Allostatic load* is defined as the "wear and tear" exacted on the body's physiological systems as a result of chronic stressors (such as living in poverty or residing in unsafe neighborhoods). So far, four primary mediators of this process have been proposed,

including cortisol, noradrenalin, epinephrine, and dihydroepiandrosterone (DHEA) (McEwen and Seeman 1999). These primary mediators have widespread influences throughout the body that potentially account for the differential resilience and vulnerability of individuals in response to adverse social circumstances. For example, cortisol is a quintessential "stress hormone" associated with activation of the hypothalamus-pituitary-adrenal (HPA) axis. The dysregulation of cortisol secretion is implicated in a wide range of disease processes, from elevated blood pressure and higher central adiposity to glucose intolerance, immune suppression, and cognitive decline (McEwen 1998). Cortisol measurement has been incorporated into surveys and study designs via 12-hour urine collections and, more recently, noninvasive and relatively inexpensive saliva specimens.

In empirical work, elevated markers of allostatic load have been linked "upstream" to adverse early life circumstances and lower socioeconomic position, as well as "downstream" to mortality risk, functional decline, and cognitive decline (Seeman et al. 1997).

In contrast to biological markers like cortisol (which represents one of the primary mediators of the allostatic process), biomarkers of *secondary outcomes* refer to those that tap into the cumulative responses to the primary mediators in various tissue/organ systems (McEwen and Seeman 1999). For example, waist-hip ratios and glycosylated hemoglobin levels both reflect the effects of sustained elevations in blood glucose and the insulin resistance that develops as a result of elevated cortisol and sympathetic nervous system activity over time. Elevated blood pressure is yet another secondary outcome resulting from the cumulative effects of allostasis. Other secondary biological markers have been proposed for the immune and clotting systems (McEwen 1998).

In sum, the substantive concerns of the emerging field of health demography have significant implications for the design of future studies conducted by both demographers and social epidemiologists. As suggested by Goldman (2001), future prospective studies should

> begin at birth, follow respondents at regular intervals throughout the life cycle, obtain detailed life histories concerning social, SES, psychological and health dimensions, consider not only the individual and family but the broader social environment, and include biological measurements along the way (134–135).

METHODOLOGICAL ISSUES

Addressing the substantive concerns of health demography requires not only novel study designs, but also departures from the traditional methods of analyzing data. In this section, we contrast the standard methodological concerns of demographers and epidemiologists, then go on to highlight multilevel statistical methodology as an emerging area of convergence between the two disciplines.

Social Epidemiology and Social Demography Contrasted

Palloni and Morenoff (2001) provide a useful summary of major methodological differences between demography and epidemiology. First, epidemiologists are taught during their training to view randomized clinical trials as the "gold standard" of evidence. No doubt this claim stems from famous instances in which observational

data (such as the apparently protective effect of beta-carotene intake on lung cancer risk) were later overturned or contradicted by clinical trial evidence (beta-carotene supplementation seems to increase the risk of lung cancer). Clinical trials are feasible, indeed desirable, where the independent variable consists of a simple "exposure" that can be exogenously manipulated (such as swallowing a beta-carotene pill). However, relatively few questions of interest to the health demographer (for example, the relationship between minority ethnic status and low birth weight) have this characteristic.

When clinical trials are not feasible, epidemiologists would still prefer study designs with tight control over potential confounding factors. For example, in examining the relationship between smoking and lung cancer, a traditionally trained epidemiologist would prefer a study design that afforded the tightest possible control over social class. Thus, the classic British Doctors' Study, one of the first to demonstrate a link between cigarette smoking and lung cancer, consisted solely of male physicians (Doll and Hill 1956). As Palloni and Morenoff (2001) have noted, "in general, the study designs favored by epidemiologists are more conducive to the identification rather than the explanation of causal factors" (143). However, the price paid by epidemiologists is their propensity to overcontrol for social and behavioral factors. The interest of the health demographer clearly cannot be focused on isolating the causal effect of cigarette smoking on lung cancer, stripped of its social context.

In contrast, social demographers, being less concerned with identifying the causal role of single risk factors, tend to "lean heavily toward study designs requiring ... representative samples, a richer stock of characteristics of individuals or social contexts" (Palloni and Morenoff 2001: 143). *Social* epidemiologists would heartily concur with these sentiments (Berkman and Kawachi 2000).

A second area of contrast between demographers and epidemiologists is in their use of analytical methods. While both epidemiologists and demographers tend to draw on the same repertoire of regression models, the interpretation of regression coefficients in social demography draws on "complex models that, more often than not, consider simultaneous causality and incorporate networks of causal relations (simultaneous equation models), recognize latent constructs made up of multiple indicators (latent and structural equation models and models with multiple indicators), and involve nested processes (choice based models, selection models)" (Palloni and Morenoff 2001: 144). By contrast, it is still relatively rare to see such complexity acknowledged in epidemiological models:

> Odds ratios in epidemiology emerge from and are couched in the most simplistic of linear representations, with little concern for the existence of simultaneity, nested processes, sequence of causal stages, and potential differences between latent constructs and indicators (Palloni and Morenoff 2001: 144).

To be fair to epidemiologists, recent developments in causal thinking within epidemiology have begun to show serious attention to the problems of endogeneity, simultaneity, and selection, through implementation of graphic models (causal diagrams), potential-outcome (counterfactual) models, and structural-equations models (Greenland and Brumback 2002). These methods have yet to make inroads in *social* epidemiology, however. Meanwhile, a potential area of methodological convergence between social demographers and social epidemiologists is the emerging consensus that we need to move beyond single-level models if we are to address questions of social embeddedness and the role of social contexts in shaping health behaviors and health outcomes.

Multilevel Statistical Methods

Traditional epidemiological and demographic methods, while extremely useful, are not adequate for the task of implementing the population, contextual, and life course perspectives raised in the preceding section. In this section, we do not intend to repeat the well-established set of either the classic demographic methods (such as life table methodology, population projection methodology, etc.) or the classic epidemiological methods (such as case-control and cohort study designs, etc.).[1]

Analysis of data in traditional epidemiological and demographic studies tends to be carried out at a single level, be it individual or aggregate. Since they operate at a single scale, these analytical approaches are incapable of handling the substantive concerns raised in the previous sections, specifically those related to integrating geographical and temporal perspectives into understanding socioeconomic inequalities in population health (Subramanian, Jones, and Duncan 2003). While choosing to work at the aggregate level leaves the investigator prone to the charge of committing the ecological fallacy (Robinson 1950) or, more precisely, aggregation bias (Roberts and Burstein 1980), choosing to work exclusively at the individual level risks committing the individualistic fallacy (Alker 1969). Critical to overcoming these problems is the explicit recognition of multiple sources/levels of variation that contribute to population differences in health. One way to accommodate multiple sources/levels of variations is to exploit the idea of nesting, so that individuals (one source/level) are seen to be nested within contexts or places or ecologies (second source/level). Such a nested framework anticipates two related assumptions vital for the health demographic perspectives outlined earlier:

1. If we structure individuals to be nested within their contexts, we implicitly assume that individuals from one area are more alike than different.
2. If we explicitly recognize the different levels that structure the outcome, it also suggests that variances that can be attributed to different levels can be simple or complex.

While these concerns are both of substantive importance, as we argued above, there are also statistical implications. As is well recognized, single-level regression models make two assumptions that are incompatible and problematic with the contextual, life course, and multilevel perspectives that we outlined earlier. The first assumes that each individual observation is independent of the other or, in formal terms, there is no "clustering." The second relates to the "homoscedasticity" assumption, i.e., that variation around the average relationships is constant. The presence of clustered data can seriously undermine statistical significance testing. For example, the effect of cluster sampling on the actual α level of a t-test performed at a nominal α level of 0.05, with a small intraclass correlation (ρ) of 0.05 and cluster size of 10, the operating α level was found to be 0.11 (Barcikowski 1981). With large ρ and large cluster sizes, the operating α level increases rapidly. Clearly, in such situations, not taking into account the clustered nature of the data can produce spurious levels of statistical significance (Tate and Wongbundhit 1983).

[1] For an excellent discussion of the classic demographic methods, the reader is referred to the textbook by Preston and colleagues (Preston, Heuveline, and Guillot 2001); for epidemiological methods, see the textbook by Rothman and Greenland (1998), as well as Moon and colleagues (2000).

One way to deal with the clustering is to view data structures in a hierarchical manner. We refer to hierarchy as consisting of units grouped at different levels. To give an example, a health demographer may be interested in examining the effect of residential segregation (by class or race) on individual health outcomes (e.g., the risk of infant mortality or low birth weight) (Acevedo-Garcia and Lochner 2003). In this case, we can think of a two-level structure where individuals are level 1 (lower level) units that are clustered within metropolitan areas of varying degrees of residential segregation at level 2 (higher level). Although empirical studies of residential segregation and health outcomes have seldom adopted this design, data structures of this type are crucial in sorting out the compositional effects of areas from their contextual effects, as well as for determining the relative importance of the different levels for health status. In ecological studies, an observed association between racial segregation (e.g., as assessed by the index of dissimilarity for a metropolitan area) and infant mortality may be consistent with either a compositional effect (i.e., a high proportion of black residents within a metropolitan area giving rise to high infant mortality rates) or to a contextual effect (i.e., something about residential segregation *per se* resulting in high risk of infant mortality), or both.

Recent developments in multilevel statistical methods have provided a unified and realistic approach to address the issues of clustering and to model complex variance structures simultaneously at multiple levels (Bryk and Raudenbush 1992; Longford 1993; Goldstein 1995). Multilevel modeling procedures account for the clustering and dependency in the outcome by partitioning the total variance into different components. In the simplest case, the response variation can be partitioned into a lower-level component and a higher-level residual component, with the latter representing the "source" of the clustering in the response. Indeed, it is this partitioning of the variance that provides important clues regarding the levels "where the action lies."

Besides the technical advantages that this methodology offers, the multilevel analytical approach provides one very useful way of addressing the substantive issues outlined above (Leyland and Goldstein 2001; Subramanian, Jones, and Duncan 2003). As the name suggests, the approach anticipates that determinants of health status occur simultaneously on several levels, e.g., individuals, neighborhoods, regions, and states. Consequently, multilevel techniques are essentially about modeling heterogeneity at each of the desired levels of the conceptual model through a range of variables that tell us something about each of the levels. Importantly, these methodological and substantive perspectives are supported by a robust technical estimation process (Goldstein 1995). In the following discussion we briefly summarize the statistical nature of a basic multilevel model.[2]

Consider again our example of isolating the effect of residential segregation on low birth weight (a risk factor for infant mortality). A two-level simple multilevel model with a continuous response with a single level-1 continuous predictor treated as a fixed effect can be written as:

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + (u_{0j} + e_{0ij}) \tag{1}$$

In Equation 1, y_{ij} is a continuous response (birth weight) for individual i in metropolitan area j. The fixed parameters β_0 is the population mean (intercept), and β_1 estimates the

[2] A more detailed statistical and methodological exposition of multilevel models can be found elsewhere (Subramanian, Jones, and Duncan 2003).

overall relationship between y_{ij} and x_{ij}. The random parameter u_{0j} is the specific effect of metropolitan area j, while e_{0ij} is the residual for individual i from the metropolitan area j. It is assumed that the random parameters are independent and are normally distributed at each level. Thus, $\boldsymbol{u}_{0j}$ is distributed with a population mean of zero and a population variance of $\boldsymbol{\sigma}^2_{u0}$ (the between metropolitan population variance) and the level-1 residuals $\boldsymbol{e}_{0ij}$ are also assumed to have a mean zero and variance $\boldsymbol{\sigma}^2_{e0}$ (the between individual within metropolitan area population variance). The total variance in $\boldsymbol{y}_{ij}$, therefore, can be written as:

$$\text{Var } (\boldsymbol{y}_{ij}) = \boldsymbol{\sigma}^2_{u0} + \boldsymbol{\sigma}^2_{e0} \tag{2}$$

For the above, the ICC coefficient ($\boldsymbol{\rho}_u$) can be defined as:

$$\boldsymbol{\rho}_{\text{u}} = \boldsymbol{\sigma}^2_{u0}/\boldsymbol{\sigma}^2_{u0} + \boldsymbol{\sigma}^2_{e0} \tag{3}$$

The ICC, therefore, is the proportion of variance accounted for by the metropolitan areas. Since variance components at level 2 and level 1 are unrelated to the population mean and have similar distributional assumptions, it is possible to add the different variance components and apportion the percentage variance to each level. While this basic multilevel model allows us to disentangle the composition and contextual sources of variation, it can be easily extended to explore the contextual heterogeneity and cross-level interactions between ecological and individual variables (Subramanian, Jones, and Duncan 2003).

Any research on population health that takes context and place seriously is intrinsically multilevel and cannot be otherwise. Multilevel methods consider most data structures within a nested framework, and such nesting could be hierarchical and/or nonhierarchical. Seen this way, repeated/longitudinal analysis (whether it is people or places that are repeatedly measured), multivariate analysis (when there is more than one interrelated outcome), and cross-classified analysis (when we do not have neat hierarchical nesting) are simply special cases of a multilevel framework (Subramanian, Jones, and Duncan 2003).[3]

TRENDS AND DIFFERENTIALS IN MORTALITY AND MORBIDITY

In the previous sections, we highlighted some key substantive and methodological issues relevant to health demography research conducted at the individual (micro) level, e.g., the salience of incorporating a life course perspective, the use of biological markers in longitudinal studies, and the relevance of contextual influences on individual health. In this section we summarize the trends and differentials in mortality and morbidity at the population (macro) level. We focus the discussion on three topics: (1) the concept of the epidemiological transition (and its critique); (2) the measurement of mortality and morbidity at the population level; and (3) trends and differentials in global health status.[4]

[3] For a detailed exposure to the methodological possibilities available through a multilevel framework for routinely collected health-related data see the compilation by Leyland and Goldstein (2001).

[4] Readers are referred to chapter 9, "Infant Mortality," and chapter 10, "Adult Mortality," for more specific and detailed accounts of ongoing research in health demography.

The Epidemiological Transition

One area of common interest to both demographers and epidemiologists is to describe and forecast global patterns of health. Originally, demographers found it useful to describe stages in the "demographic transition" to refer to the change from high fertility and high mortality rates in "traditional" societies to a pattern of low fertility and low mortality rates in "modern" societies (Thompson 1929; Notestein 1945). Subsequently, Omran (1971) extended this framework to describe three stages in the "mortality transition" consisting of: (1) the age of pestilence and famine, associated with the predominance of mortality from epidemic infectious diseases, malnutrition, and complications of pregnancy and childbirth; (2) the age of receding pandemics, during which mortality fell and life expectancy increased[5]; and (3) the age of noncommunicable diseases, during which mortality came to be dominated by chronic degenerative diseases such as heart disease, stroke, and cancer. A fourth stage in the mortality transition was later added by Olshansky and Ault (1986) and by Rogers and Hackenberg (1987), describing an era of delayed mortality from degenerative diseases, as well as the resurgence of "old" infectious diseases (e.g., tuberculosis) and the emergence of new infectious diseases (e.g., HIV/AIDS). Patterns of mortality and morbidity in this fourth stage have been explained largely on the basis of individual lifestyle (Rogers and Hackenberg 1987), although this interpretation has been questioned on the grounds that it overemphasizes individual determinants of health while underplaying the importance of broader social and economic factors (Beaglehole and Bonita 1997).

Demographic and epidemiological research continues to be informed by the concept of the epidemiological transition. However, both the epidemiological transition, and the related but broader concept of the "health transition" (Caldwell 1990), have been criticized on the grounds that they are descriptive frameworks rather than true theories that yield predictions about patterns of population health (Wallace 2001). Beaglehole and Bonita (1997) provide a cogent critique of the epidemiological transition model. For example, they point out that the model fails to explain differences in mortality rates between countries and has limited ability to predict changing patterns of disease with "modernization." Contemporary examples of mortality change, such as the mortality crisis in post-Soviet Russia (McKee 2001), do not fit well into the orderly progression of stage suggested by the model. In fact, it has become evident that the various stages in the health transition can overlap within any given country and that they do not necessarily progress in a linear fashion. Moreover, the categorization of diseases into infectious and noncommunicable diseases ignores major causes of mortality and morbidity, such as traffic accidents, unintentional injuries, and violence. Finally, Beaglehole and Bonita noted a tendency to analyze the transition in isolation from the background social and economic forces that propel population-level changes in health status.

In summary, according to Beaglehole and Bonita (1997), "although the health transition theory is a useful descriptive tool, it remains a blunt instrument with only limited predictive power" (10). Health demographers therefore face a challenge in further developing and refining the construct into a theory that is testable and

[5] In Western Europe and North America, the second stage of the mortality transition has been dated from the beginning of the 18th century until the early 20th century, with the 1918 to 1920 influenza pandemic being the last major pandemic (Mackenbach 1994).

applicable to both developing and industrialized countries, as well as historical and contemporary contexts.

Measurement of Mortality and Morbidity

Mortality and cause-of-death statistics based on death certificates continue to be the mainstay of health assessment in both demography and epidemiology. However, those outside the field often overlook the complexities of mortality data collection. As Sir Austin Bradford Hill (1984) remarked:

> In making comparisons between death rates from different causes of death at different times or between one country and another, it must be realized that one is dealing with material which the distinguished American statistician Raymond Pearl long ago described as, 'fundamentally of a dubious character,' though of vital importance in public health work (259).

It still remains the case that complete cause-specific death registration data are routinely available for only a minority of the world's countries. Less than one-third of the global population is covered by national vital registration systems, and there is wide regional variation in coverage, ranging from 80% population coverage in the European region to less than 5% coverage in the Eastern Mediterranean and African regions of the World Health Organization (Bonita and Mathers 2003). On the other hand, recent improvements in sample registration systems and surveys have improved coverage, especially for under-five child mortality and maternal mortality. For example, data collection on child mortality has improved with cross-country surveys such as the Demographic and Health Surveys (DHS) and the Multiple Indicator Cluster Survey (MICS) program of UNICEF. Bonita and Mathers (2003) estimate that national vital registration systems together with sample registration data currently cover about 74% of global mortality. Survey data and indirect demographic techniques provide information on child and adult mortality for the remaining 26% of estimated global mortality.

The assessment of morbidity is even more complex than the measurement of mortality, since it must frequently rely on the self-reports of symptoms and illnesses by survey respondents. For instance, it is widely recognized that even the self-report of the commonly used single item on general health perception (''How would you rate your overall health? Excellent, Very Good, Good, Fair, or Poor?'') can be biased. The problem is related to unmeasured differences in expectations and norms for health, based on culture, educational attainment, age, gender, and other respondent characteristics. In developing countries in particular, the socioeconomic gradient in poor self-rated health often runs in the ''wrong'' direction, with more educated groups reporting higher levels of morbidity, even though their objective health status (e.g., as measured by mortality rates) is clearly superior to lower socioeconomic groups (Sen 2002).

The accuracy of self-reported morbidity in surveys can be improved by validating specific diagnoses against medical records and pathology reports. Obviously, these procedures are time-consuming and expensive and limit the size of the survey. An alternative is to use data from hospitals or clinics or even restrict survey respondents to health professionals (such as in the Harvard Nurses' Health Study). However, as pointed out by Caldwell (2001), such approaches go ''far toward destroying the concept of a *population*, which is basic to the way demographers see the world'' (22–23; emphasis in the original).

In recent years, the World Health Organization has pioneered cross-country survey-based approaches to measuring disability. Based on the International Classification

of Functioning, Disability and Health (ICF), the WHO carried out a Multi-Country Survey Study in over 60 countries during 2000–2001. A health module was administered to assess six core domains of health status, including pain, affect, cognition, mobility, self-care, and usual activities (including household and work-related activities) (Üstün et al. 2001). The WHO survey included case vignettes and some used selected measures to calibrate respondents' self-assessments of their health. Statistical methods were developed to correct potential biases in self-reported data (Murray et al. 2002b). Over half were household interview surveys, two were telephone surveys, and the remainder postal surveys. The results of the WHO Multi-Country Survey for the first time provide measures of disability prevalence and health status that are comparable across a broad set of countries (Bonita and Mathers 2003). It represents an important step toward standardizing the measurement of morbidity and disability across populations.

Finally, the World Health Organization has taken the lead in refining summary measures of population health status that combine measures of survival and morbidity into a single metric (Murray et al. 2002a). Two classes of measures have received particular attention: the disability-adjusted life-year (DALY), which is a "health gap" measure that combines time lost due to premature mortality with time lived with disability and healthy life expectancies (HALE), which measures the equivalent number of years of life lived in full health extrapolated from comparable cross-national data (World Health Organization 2000). Both sets of measures have been widely debated. In particular, critics have pointed out the data demands and complexity of making the calculations that involve numerous assumptions (Almeida et al. 2001). The weighting of disability states as well as the social values implicit in the weighting of life years at different ages has been similarly contested. Such debates notwithstanding, health demographers (and policy planners) increasingly recognize the limitations of assessing population health status through measures of mortality or life expectancy *alone* (i.e., ignoring morbidity).

Global Trends and Differentials in Mortality and Morbidity

Improvements in global health status, as measured by gains in life expectancy, have been accompanied by widening differentials both between and within countries. Life expectancy at birth currently ranges from 81.4 years for women in the established market economies of Western Europe, North America, Japan, Australia, and New Zealand, down to 48.1 years for men in sub-Saharan Africa (Bonita and Mathers 2003). While mortality rates have declined markedly for specific causes of death (such as coronary heart disease) in wealthy countries, other regions of the world have witnessed equally spectacular reversals in life expectancy. For example, between 1991 and 1994, life expectancy at birth in the former Soviet republics fell by 4 years for males and by 2.3 years for females (McKee 2001). Between 1994 and 1998, life expectancy for Russian men improved, but declined significantly again over the next three years (Bonita and Mathers 2003). Worldwide, about 37 million people are currently living with HIV/AIDS, of whom 95% reside in developing countries. The impact of HIV/AIDS has been catastrophic in sub-Saharan Africa, where between 2000 and 2005, the United Nations Development Program has projected that the decline in life expectancy due to the disease will amount to 34 years in Botswana, 26 years in Zimbabwe, 19 years in South Africa, and 17 years in Kenya (United Nations Development Program 2002).

According to the World Health Organization, about 56 million deaths occurred worldwide in 2000, of which 10.9 million (20%) were deaths among children aged less than five years of age (WHO 2001). Of the under-five child deaths in the world, 99.3% occurred in developing countries. Developing countries also share a disproportionate burden of premature deaths at young adult ages (15 to 59 years). Just over 30% of all deaths in developing countries occur at these ages, compared with 15% in richer countries. By contrast, 70% of deaths in developed countries occur beyond age 70 (Bonita and Mathers 2003).

Contrary to the linear progression from infectious diseases to noncommunicable diseases implied by the epidemiological transition, many countries confront a so-called "double burden" of diseases, with high prevalence of both old and new infectious diseases in addition to emerging epidemics of chronic noncommunicable diseases such as heart disease, stroke, diabetes, and cancer. Of the 56 million deaths worldwide in 2000, 32.8 million (or 59%) were due to noncommunicable diseases, which killed twice as many people as infectious, maternal, perinatal, and nutritional causes combined (17.8 million, or 31% of all deaths). Injuries killed an additional 5.1 million people in 2000, or about one-tenth of the world's total deaths (Bonita and Mathers 2003).

There is enormous heterogeneity in health status even within developing countries. In China (which accounts for one-sixth of the world's population), fewer than 10% of all deaths occur before age five, compared with 40% in Africa (accounting for one-tenth of the global population). Conversely, 45% of deaths in China occur beyond age 70, compared with only 10% in Africa (Bonita and Mathers 2003).

Within individual countries also, differentials in mortality have been recorded since the very beginning of vital records registration. In several countries, such as the United Kingdom (Drever and Bunting 1997) and the United States (Pappas et al. 1993), these differentials have not only persisted in spite of technological advances in medicine and the rising standard of living, but they also seem to have widened in recent decades.

The magnitude of the health differentials is striking, even in wealthy countries. Within the United States, for example, a black male born in the District of Columbia can expect to live 57.9 years, lower than the life expectancy of the male citizens of Ghana (58.3 years), Bangladesh (58.1 years), or Bolivia (59.8 years). By contrast, an Asian American woman born in Westchester County, New York, can expect to live on average for 90.3 years (Murray et al. 1998).

The challenge for health demographers, then, is not only to refine methods for documenting and monitoring population health, but also to develop new theories and conceptual models to account for the causes of health variations both within and between countries.

NEXT STEPS

In this chapter, we have attempted to set forth what we view as the key conceptual, substantive, and methodological challenges to the advancement of health demography as an interdisciplinary science. We have argued, along with others (Susser and Bresnahan 2001), that despite nearly two centuries of divergence and specialization, demographers and epidemiologists are poised on the brink of a major bridging across disciplinary boundaries, concerns, and methods. Indeed, such convergence was the theme of a recently edited issue of the *Annals of the New York Academy of Sciences*

(Weinstein, Hermalin, and Stoto 2001). Demographers are already assimilating the techniques and measures that used to belong in the domain of medical epidemiologists, such as the assessment of specific disease diagnoses in population surveys or the collection of biological specimens in longitudinal studies (Goldman 2001). For their part, epidemiologists, especially *social* epidemiologists, have begun to move toward population-based, as opposed to clinical, samples, and to incorporate an expanded understanding of the determinants of health in their work, which includes not just the traditional "risk factors" (such as genetic susceptibility or lifestyle behaviors) but, in addition, *social* determinants such as socioeconomic status, social support, and neighborhood contexts (Berkman and Kawachi 2000; Kawachi and Berkman 2003).

The cross-fertilization of demography and epidemiology is happening at a crucial stage in the evolution of human health. New threats and challenges to global health, such as the AIDS pandemic, the worldwide aging of the population, and the widening economic gulf between rich and poor countries associated with globalization, demand analytical approaches and strategic responses that are simultaneously rooted in the historical concerns of demographers with whole *populations*, as well as the more individual and biological focus of epidemiologists.

At the population (macro) level, health demographers will continue to be engaged by improving systems of measuring, monitoring, and forecasting mortality and morbidity. Much work remains to be carried out in refining the theory of health transition, to turn it into a genuine theory capable of yielding testable predictions about patterns and trends in population health. The measurement of morbidity still lags behind the measurement of mortality for all but the most economically advanced societies. Quantitative techniques for summary indices of health status (combining morbidity measures with survival) are still in their relative infancy, in part because of lack of data as well as lack of agreement about how to weight different health states and life years at different stages of the life span.

At the individual (micro) level, health demographers will continue to be challenged by the task of developing better conceptual models of the determinants of health. This task involves not only expanding the repertoire of the individual determinants of health, e.g., contextual influences such as income inequality (Kawachi, Kennedy, and Wilkinson 1999) and neighborhood environments (Kawachi and Berkman 2003), but also incorporating the dimension of time (the life course) and elucidating the biological pathways and mechanisms that connect population-level forces to individual health.

The subject matter of health demography is not new. We concur with Pol and Thomas (1992), who observed that

> This emerging discipline actually represents a synthesis and reformulation of concepts and substantive data developed in a variety of other fields . . . (most obviously) the convergence of traditional demography with aspects of biostatistics and epidemiology (2).

After a long period of separation, the fields of demography and epidemiology are finally converging. The dynamic interplay between the changing size and composition of the population as well as its changing health patterns across different multilevel contexts lie at the core of the inquiry of health demography. The foundational basis for this new field not only draws on the natural and historical overlaps between epidemiology and demography (Weinstein, Hermalin, and Stoto 2001), but also on a renewed appreciation of the need to integrate research across multiple levels of analysis, from the societal and

population level down to the individual, biological, and molecular levels (Shonkoff and Phillips 2000).

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