# Editorials, Annotations, Comments, and Topics for Our Times

## Editorial: Region of Birth and Mortality among Black Americans

The paper published by Schneider, Greenberg, and Lu in this issue of the Journal[1] demonstrates that circumstances in early life—in this case, region of birth—are linked to the circulatory disease mortality of adult Black Americans. More specifically, Black men and women born in the southern region of the United States carry with them an extra risk, regardless of whether they stay in the South or migrate. Conversely, it is suggested that Blacks born in the western region of the United States carry a lower risk throughout their adult life, regardless of where they live as adults.

This study of migrants follows a number of similar studies outside the United States which have demonstrated an effect of region of birth on mortality, in particular cardiovascular mortality. Strachan, Leon, and Dodgeon[2] were able to show that interregional migrants in England and Wales differed in their mortality from ischaemic heart disease and stroke, depending on their region of birth. Those who spent their childhood and/or youth in the northwest carried on average a higher risk than those from the more prosperous southeast. This was also true when residence in later life was controlled for. Similarly, in analyzing mortality rates from ischaemic heart disease for men from relatively poor eastern Finland and those from relatively rich western Finland, Valkonen found that "being born in East Finland is a more important risk factor than is living there."[3(p80)] It should be noted that Valkonen's study is unusually strong in that it is based on the Finnish census population individually followed up in the Finnish Deaths Registry during a subsequent 5-year period, with less than 1% loss to follow-up.

Although these studies demonstrated birth-region effects, they also showed the effects of region of adult residence. In England and Wales, this was especially the case for stroke. With respect to heart disease, the authors concluded that "differences were related in almost equal measure to region of origin and region of residence in late adult life."[2(p426)] Schneider and Greenberg, unfortunately, do not formally estimate the size of effects of region of birth or region of adult residence in their study population.

Parallel with studies demonstrating effects of the region of birth, a number of studies have also been undertaken to estimate the effect of social class at birth on circulatory disease, controlling for adult social class. Thus Kaplan and Salonen,[4] using Finnish data, demonstrated such an effect on heart disease. Lynch et al.[5] failed to find such an effect and concluded that the effect of present social class was of much greater magnitude, although the authors cautioned against an interpretation that gave importance to adult experience only. Notkola et al.,[6] in their exploration of the east–west differences in Finland, concluded that it was those men who were born landless in rural eastern Finland who were especially prone to death from myocardial infarction or ischaemic heart disease, regardless of their achieved social position later in life. A study by Vågerö and Leon,[7] using Swedish census data individually linked to subsequent deaths, suggested an independent effect of childhood social class, in particular of having parents who were nonemployed, on total and ischaemic heart disease mortality.

**Editor's Note.** See related article by Schneider et al. (p 800) in this issue.

Thus, we are inevitably drawn to the conclusion that factors early in life—seemingly linked to poverty, social discrimination, or social disadvantage in general—as well as factors later in life (perhaps independently) influence circulatory disease mortality rates. The relative contribution of early and late factors is an issue of great public health importance. Whether or not the effects of those early factors are modified by later factors is an important question. It is also imperative to understand the nature of these early influences.

In discussing their results, Schneider and Greenberg write that "a plausible hypothesis for these patterns is that a factor or factors operating in prenatal environment or in early childhood influence(s) the development of circulatory disease, an explanation which is supported by the literature."[1] An extensive literature in this area ranges from studies of animal metabolism to longitudinal studies of the health consequences of cumulative life experience. However, the literature quoted as evidence is contradictory in many of its conclusions. Whatever the preferred explanation—genetic features,[8] the fetal environment,[9] the first year of life,[10] the childhood years,[11] or all of these together—the literature shows no consensus.

Consequently, a strong need exists for studies that are designed so that they can support one, and refute another, of these alternative explanations. A study that does not allow such a test should not be quoted as giving evidence in favor of one theory rather than another. Many such studies, which are justified by establishing the importance of early life circumstances, have been unable to distinguish between alternative explanations—something that has often been overlooked by subsequent authors quoting these papers. I have often gotten the impression that "camps" were being formed in this area, quoting evidence "supporting" or "being compatible with" their favorite hypotheses, but not quoting work that goes against it nor alternative hypotheses that are compatible with the same evidence. This practice may be inevitable to some extent, but in the long run it will hold us back from a deeper understanding of the problem.

Schneider and Greenberg extend previous work in this field into a new population, thereby opening up new opportunities to answer the questions put forward above. Their study's results are compatible with the hypothesis that Black mothers in the South have difficulty in providing their children with sufficient nutrition during pregnancy, due to poor living circumstances, thereby creating vulnerability to adult disease. Their study is equally compatible with the hypothesis that it is the accumulated life experience of discrimination and social disadvantage, including poor education, that makes children born in the South vulnerable to heart disease as adults. Even if these are alternative explanations, they are not mutually exclusive; they could both be true. If it is school achievement, or fetal nutrition, or some other factor, that determines vulnerability among Black Americans born in the South, this must be addressed in a study specifically designed for that purpose, as Schneider and Greenberg are well aware.

Recent work in Sweden, undertaken to test the so-called "fetal origins hypothesis," suggests that growth retardation in utero might indeed be linked to elevated blood pressure[12] and insulin resistance,[13] and thereby, the authors presumed, to elevated cardiovascular mortality. The authors also suggest that the rise in blood pressure with falling birthweight is strongly related to body mass index in adult life. The blood pressure of those who were not obese did not vary much with birthweight. The implication of this is that early and later life experience interact in important ways. Similarly, Clausen et al.[14] found that a specific genetic characteristic was linked to insulin resistance, but only among those who are obese. If interactions of these kinds are common, which is likely, it is from understanding these interactions that future leaps in knowledge will occur. The thought that the effect on cardiovascular health of early life experience can be modified, or even reversed, by later life experience is appealing. If true, it has far-reaching public health implications.

Much recent research takes a life-course perspective on adult health risks. Sometimes this means nothing more dramatic than collecting data from early life as well as from later life in cross-sectional surveys. In such a study, Brunner et al.[15] were able to demonstrate that indicators of early life experience (for instance, education and body height) as well as factors operating later in life (such as work characteristics) independently contribute to plasma fibrinogen concentration and, thereby, risk of coronary disease. A mass of evidence relating different aspects of the life course to biological and social correlates, from birth to old age, is presently appearing in medical and sociological journals. A number of ongoing longitudinal studies, starting at birth and slowly moving towards increasingly older ages, are particularly important and show promise that this trend will continue.

A more systematic discussion of this evidence would be rewarding. Regardless of whether or not we believe in one of the specific hypotheses, such as "biological programming in utero," most researchers would agree that an individual's health and social career become linked early in life. Health and social career evolve together, partly because they are influenced by the same set of social circumstances, and partly by their mutual influence on each other. To understand why there are such high levels of circulatory disease mortality among Black Americans, an elaboration of this so-called "co-evolution hypothesis"[16] would seem to be promising.

Half a century ago, Gunnar Myrdal wrote in his impressive treatise, *An American Dilemma,* on the situation of Black Americans:

> Area for area, class for class, Negroes cannot get the same advantages in the way of prevention and cure of disease that the whites can. Discrimination manifesting itself against the Negro's health is indirect as well as direct, and fits into the pattern of a vicious circle. . . . Ill health reduces the chances of economic advancement, which in turn operates to reduce the chances of getting adequate medical facilities or the knowledge necessary for personal health care.[17(p171–172)]

This was written in the context of understanding how Black migration and population changes influenced the economic and social standing of Black Americans. Migration was seen as one way of achieving economic and social advancement which, in turn, was seen as the main way to reduce mortality. The optimistic conclusion was that "any intelligent effort to reduce Negro morbidity and mortality will result in striking success."[17(p174)]

If we as researchers are to contribute to this success, we need to make more intelligent efforts, particularly in understanding which factors of early life are important for present day mortality rates, and, secondly, in identifying those factors of adult life which might modify, reduce, or even reverse risks carried from childhood. □

***Denny Vågerö***
***Department of Sociology***
***Stockholm University***
***Sweden***

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# Editorial: The Uses of Psychosocial Epidemiology in Promoting Refugee Health

An estimated 18 million people worldwide are currently refugees from low-income countries, and an additional 15 to 25 million are internally displaced within their own national borders. Less than 5% of refugees resettle in industrialized countries.[1] The rest remain for long periods in camps established by the United Nations High Commissioner for Refugees where they are assisted by nongovernmental humanitarian relief organizations. Typically, these camps are located in developing countries ill equipped to handle the complex social and economic needs of these groups. While camps may provide some safety and improved health care, refugees remain highly traumatized, deracinated individuals, often prevented by circumstance from engaging in productive labor and prohibited by edict from integrating into the surrounding society. The condition of persons internally displaced may be even more dire.

International agencies and governments now recognize that the psychological health of these groups requires programmatic attention. In this context, public health professionals with a mental health perspective have substantial expertise to offer. Psychosocial and psychiatric epidemiology, for example, offers methods for quantifying mental health burdens at the population level, identifying at-risk groups, and evaluating the efficacy of prevention and treatment. Since efficient health planning is only possible with knowledge of disease rates and program performance, epidemiology is indispensable for developing rational refugee health policies.

The universality of disease entities—a presumed *sina qua non* of epidemiologic research—has long been debated in cross-cultural mental health research.[2] However, in recent years, researchers and clinicians have developed comprehensive, phenomenological descriptions of psychiatric disorders, atheoretical as to etiology,[3] together with matching, highly structured diagnostic instruments for population-based surveys.[4] According to World Health Organization reports, these instruments exhibit acceptable cross-cultural reliability and are judged serviceable by regional psychiatrists.[5] Devins, Beiser, and colleagues have a meticulous paper in this issue[6] on the Affect Balance Scale's psychometric equivalence among different refugee groups in North America which is a welcome addition to this literature. In sum, while the role of culture in the definition and expression of disturbed behavior remains to be fully elucidated, instrumentation is now adequate for pursuing quantitative and clinical questions relevant to research and program planning.

Clinic-based research on refugees settled and seeking treatment in economically advanced nations flourished early in this new psychometric environment.[7–9] Logistically more taxing epidemiologic investigations in unselected samples are of relatively more recent vintage.[10–15] However, population-based studies on the far greater number of traumatized individuals never reaching our industrialized shores constitute just a handful of important pioneering works[16–18] and provocative "in-house" surveys by UNICEF and nongovernmental relief organizations.[19–20] As a consequence, we have no substantial body of established population-based findings on psychosocial distress and frank psychiatric disorder among these groups.

Results from a large-scale UNICEF survey in Rwanda, involving interviews with 3000 Rwandese children, dramatize the compelling claims of such crises on our professional and moral attention. This UNICEF study estimated that, during the 1994 genocidal civil war, 90% of the children had seen dead bodies or parts of bodies; a third had witnessed family members being killed; and 15% had hidden under dead bodies.[21] Given these

Editor's Note. See related article by Devins et al. (p 794) in this issue.