Population Health: Challenges for Science and Society

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The emphasis on risk factor intervention at the individual level has predominated in efforts to reduce mortality and promote health. Interest in social and other nonmedical interventions, particularly socioeconomic status (SES) influences, has increased in recent years. This article focuses on the interaction of social structure and socioeconomic status with other influences in complex pathways to affect health, and their contribution to health disparities. It examines both social class as an explanation of health differences and competing hypotheses concerning prenatal and early nutrition and cognitive capacity. Although education is associated with income, wealth, occupation, and other SES indicators and may not be the most important SES determinant, it influences a variety of pathways to health outcomes and offers strategic leverage for intervention because of social and political consensus on its value beyond health.

Keywords: Population health, social class, socioeconomic status, infant mortality, nutrition, cognitive capacity, education.

THERE HAS LONG BEEN A DIALECTICAL TENSION between the view that health and disease is most fundamentally shaped by broad social, cultural, and environmental factors and the view that specific pathogens and risk factors are most important to understanding and combating illness and death (Bloom 2002; Brandt 2007; Dubos 1959; Grob 2002; Rothstein 2003; Waitzkin 1983). In the last half century an individualized and medicalized perspective focusing on risk factor identification and intervention has dominated (Rothstein

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2003). More recently, however, an emerging revitalization of a broader perspective on the determinants of population health and its potential value in helping solve existing and future health problems has gained traction. This perspective focuses on "upstream factors" such as cultural and social structures, socioeconomic status in one's family of birth and throughout the life course, and social and environmental factors, typically described as nonmedical determinants, in contrast to "downstream factors" more proximate to disease processes such as blood pressure, cholesterol, diet, exercise, and genetic risks.

Health and disease are widely understood as being influenced by many factors, and contending perspectives are not necessarily incompatible. Nevertheless, the relative focus on one or another of these perspectives affects research funding and the research agenda, the types of interventions developed, and the social policies seeking to promote health and prevent disease. The challenge for science and society is to make upstream and nonmedical determinants more central to how the public, policymakers, and scientists conceptualize health challenges and how to make such influences a more important consideration in the nation's research and health agenda. Although its range is broad, this article concentrates on the importance of socioeconomic factors and their links with other important determinants.

Researchers and analysts disagree about the relative importance of various socioeconomic indicators for health such as education and income and wealth relative to one another (Davey Smith et al. 1998), and uncertainty remains. Here I look at education, not because I believe it is more important than other components of social class, but because it has a strategic importance from a policy standpoint. Education affects many health and disease pathways, and because there is public and political consensus about its value, it allows health initiatives that might face larger barriers if pursued through other means such as income redistribution.

Nonmedical Determinants

The population health approach has the potential to address risk factors common to a range of diseases and other health problems. It transcends an individualized medical approach by examining how broader social and policy changes like the regulation of foods, drugs, and smoking; income

and educational policies; health system innovations; "sin" taxes; and improved environmental standards affect behavior, health experiences, and disease and mortality outcomes. Particular social and environmental influences such as social class are often associated with a variety of conditions across bodily systems and specific etiologies. Thus, thinking in broad terms about the causes of ill health may offer cost-effective opportunities in the long run (Bunker, Gomby, and Kehrer 1989; Marmot and Wilkinson 1999).

Advances in medicine and therapeutics have no doubt contributed impressively to the extension of life and its quality in the past fifty years (Cutler 2004; Cutler, Rosen, and Vijan 2006). But medical interventions are only one of many determinants of health. Although traditional public health measures, including safe water, better sanitation, immunization, food safety, and adequate nutrition still are important determinants, they often are taken for granted in developed societies. A broad view suggests that other significant influences are inherent in the social stratification of populations, the integrity of governmental processes, economic and cultural arrangements, available technologies and modes of production, and the impact of the environment itself (Benjamin 1965; Dorn 1959; Kunitz 2007; Mechanic 1978).

While recognizing the multiplicity of factors that contribute over the life course to chronic disease and mortality, analyses of mortality often center on specific determinants that appear amenable to intervention. Accordingly, the role and the importance of any single factor for any particular outcome are likely to depend on time, place, life stage, and the social context. In contrast, social class has more pervasive effects across time and circumstances.

Social Class and Population Health

Within theories of social stratification, social class characterizes how persons and groups relate to one another in terms of social standing, resources, authority, influence and power, and deference. People experience social class as a unitary whole, but with the exception of some studies of smaller communities, empirical research largely uses, as proxies, measures of socioeconomic status (SES) like income, education, occupation, wealth, and residence. Although these indicators are intercorrelated, each also is associated with distinct pathways through which it exercises

influence. Thus empirical studies find that such indicators as education, income, and occupation have independent effects beyond their joint influences (Kessler 1982; Kitagawa and Hauser 1973; Lahelma et al. 2004) and that the relative effects of each indicator vary depending on the outcomes in question.

Social Class as a Fundamental Cause

Bruce Link and Jo Phelan studied socioeconomic factors as "fundamental causes" of morbidity and mortality (Link and Phelan 1995, 2005), explaining the concept of fundamental cause as follows:

> A fundamental cause involves access to resources, resources that help individuals avoid diseases and their negative consequences through a variety of mechanisms. Thus, even if one effectively modifies intervening mechanisms or eradicates some diseases, an association between a fundamental cause and disease will reemerge. As such, fundamental causes can defy efforts to eliminate their effects when attempts to do so focus solely on the mechanisms that happen to link them to disease in a particular situation. (1995, p. 81)

They contend that it is money, knowledge, beneficial social networks, and power and prestige, all associated with SES, that allow people to protect themselves from adversities and to take positive action to prevent or ameliorate a wide range of threats to health. These advantages allow people to lead a healthful life, to identify and avoid many dangers, and to have access to the latest biomedical technologies and services and a range of other helpful people, information, and resources.

If their view is to be more than just an interesting argument, it must generate implications that can be both tested and refuted. Link and Phelan extrapolate that if social class provides resources that protect against disease and death, its effects should be linked to the preventability of varying causes of death. Using the National Longitudinal Mortality Study, a prospective data set linking samples of the Current Population Surveys to the National Death Index, and physicians' ratings of the preventability of various causes of mortality, they found that the mortality relationship with education and income was much stronger in the case of highly preventable conditions, for example, cerebrovascular disease, chronic obstructive pulmonary disease, and cancer of the trachea, bronchus, and lung, in contrast to low-preventable conditions, such as

cancer of the pancreas, breast, and prostate or multiple sclerosis (Phelan et al. 2004).

Research on disparities further illuminates implications of the fundamental cause perspective.

The Relationship of Fundamental Causes to Racial Disparities

Eliminating health disparities is one of the major overarching goals of America's health objectives for the year 2010 (U.S. Department of Health and Human Services 2000). There are significant disparities across all indices of social stratification and in relation to a great variety of outcome measures, including the prevalence of disease, access to treatment, quality of care, and mortality (Smedley, Stith, and Nelson 2003). Disparities by race are of particular concern because they typically are large and relate to America's legacy of slavery, racism, and discrimination. As the nation focuses on racial disparities, much of the policy discussion, and its representation in the public arena and the media, has centered on the lack of progress in eliminating the relative high proportion of black to white deaths (disparity ratios).

An important implication of the fundamental cause hypothesis is that many advances in biomedical knowledge and technology will increase the health disparities between people of higher and lower socioeconomic status. To the extent that knowledge, money, social connections, and influence make it easier for more advantaged persons to gain access to valuable information and services to prevent or treat illness, they inevitably increase the disparities in outcomes. After the introduction of a new biomedical advance, the socially advantaged gain the most at first, but as the knowledge and interventions disseminate more widely, these gaps tend to close. But then more disparities are created by still more advances in knowledge and technology. In the following case example of infant mortality, I illustrate how the provision of resources not previously available to the Southern black population closed the black-white gap in essential resources for preventing infant mortality consistent with the expectations suggested by the fundamental cause hypothesis.

A Case Example: Infant Mortality

Since the middle 1960s, there have been large improvements in the United States in infant mortality rates for both blacks and whites, but as

often noted, the percentage of disparity between black and white infant mortality more than doubled between 1950 and 2004 (see table 1). Ironically, this occurred during a time period when many more additional black lives were saved per one thousand live births than white lives were.

The meaning of disparity ratios is often misunderstood, so a slight detour on this issue may be helpful before proceeding. Disparity ratios, odds ratios, and other such statistical measures derived from very different base rates often are difficult to interpret for policy purposes. As mortality or other measured events decline, decreases in base rates to relatively low levels can result in large percentage disparities in comparisons among more and less advantaged groups (Scanlan 1991, 2006). A recent World Health Organization publication shows how relative gaps in deaths can increase while absolute gaps close (Whitehead and Dahlgren 2006, table 1, p. 30). Understanding absolute changes in events among various groups usually is more informative for policy understanding than are changes in disparity ratios alone.

Table 1 shows black and white infant mortality rates at five-year intervals from 1950 to 2004. Differences in black-white infant mortality substantially reflect differences in parental education and family income but also represent racial discrimination or other differences specifically attributable to race beyond those attributable to SES. Unfortunately, national trend data that would allow us to examine the relative contributions of SES and race are not available. The most remarkable fact in the table, although commonly taken for granted, is the large reduction in infant mortality between 1950 and 2004 as a result of improved living conditions, advances in medical technology, and access to care. There also has been an increase in the black-white disparity ratio (from 64 percent in 1950 to 142 percent in 2004) even during intervals when the number of black lives saved per one thousand live births was higher than the number of white lives saved, for example, in 1975, 1980, 1985, 1995, and 2000. This should alert policymakers that tracking disparity ratios alone can be misleading. Comparing 1950 with 2004, the number of deaths per one thousand live births decreased by 21.1 for whites and 30.1 for blacks. Nevertheless, the relatively high black infant mortality rate remains an important cause of concern, so it is essential to understand and develop appropriate interventions.

Improvement in the United States in African Americans' health and welfare has been attributed to three major interrelated trends. First was

TABLE 1
U.S. Black-White Infant Mortality, 1950–2004

	White	Absolute Reduction per 1,000 Live Births for Previous 5-Year Interval	Black	Absolute Reduction per 1,000 Live Births for Previous 5-Year Interval	Disparity (%)
1950	26.8	—	43.9	—	64
1955	23.6	3.2	43.1	0.8	83
1960	22.9	0.7	44.3	(+1.2)	93
1965	21.5	1.4	41.7	2.6	94
1970	17.8	3.7	32.6	9.1	83
1975	14.2	3.6	26.2	6.4	85
1980	10.9	3.3	22.2	4.0	104
1985	9.2	1.7	18.6	3.6	102
1990	7.6	1.6	18.0	0.6	137
1995	6.3	1.3	15.1	2.9	140
2000	5.7	0.6	14.1	1.0	147
2004	5.7	0	13.8	0.3	142
Total Absolute Reduction Rate per 1,000 Live Births, 1950–2004		21.1		30.1	

Sources: For 1950–1975, adapted from National Center for Health Statistics 1978, table 25, p. 176; and for 1980–2004, adapted from National Center for Health Statistics 2006, table 22, p. 166.

the large migration of blacks from south to north between 1940 and 1970 and their urbanization; second, a high and sustained rate of economic growth during this period; and third, and particularly relevant to this discussion, the civil rights movement, which opened access to major educational and medical institutions (Jaynes and Williams 1989; Smelser, Wilson, and Mitchell 2001). Economic downturns after 1973 complicate the picture, but it is clear that many influences at the macro level interacted to affect life chances and health. Table 1 shows that from 1965 into the 1980s, black infant mortality compared with white infant mortality fell substantially.

The years following 1965 saw substantial social legislation and civil rights advances that improved occupational, educational, and medical access for blacks. Specifying pathways to health from this broad constellation of changes associated with the "War on Poverty" and civil rights is difficult because of the complexity of interactions among these nonmedical and medical determinants encompassing new educational and job opportunities, nutritional programs and access to medical care, and an increased sense of hopefulness and personal empowerment (LaVeist 1993). Between 1960 and 1980 the black-white inequality in the amount of schooling was greatly reduced (Jaynes and Williams 1989), and by 1980, the black-white gap in median years of schooling closed to less than one-half year. These changes increased health resources and opportunities for the black population, particularly in the South, who before these developments had restricted access to essential medical resources to prevent infant mortality and protect infant health.

A major impediment to black infant health in the South was the exclusion of blacks from segregated public hospitals. In 1963, a U.S. appeals court invalidated a "separate but equal" clause in funding for hospital construction, followed by Title VI of the 1964 Civil Rights Act and implementation of Medicare in 1966, requiring that hospitals eligible for federal reimbursement not discriminate (D.B. Smith 1999). This had a dramatic effect, particularly in the rural South, in offering access to hospitals previously unavailable to many black patients and providing access to treatment for gastroenteritis, influenza, and pneumonia, the major causes of black infant deaths (Almond, Chay, and Greenstone 2003). In Mississippi, for example, black postneonatal mortality fell 50 percent between 1965 and 1971. This period, 1965 to 1975, saw not only a large absolute drop in black infant deaths but also a reduction in the disparity between black and white infant deaths. Opening public

hospitals to blacks helped equalize a vital health resource, thereby minimizing the white resource advantage.

Although many of the improvements in infant mortality were closely related to living conditions, appropriate health behavior and parenting skills, and access to basic health care, more recent advances in medical technology have had an important role, especially high-risk obstetrics and neonatal intensive care, and pulmonary surfactant therapy for the treatment of respiratory distress syndrome in preterm infants. Following the introduction of surfactant therapy, deaths from this cause declined substantially (Frisbie et al. 2004).

Infant postnatal deaths are typically seen as more amenable to social influences than are those occurring in the first twenty-eight days of life. As the number of infant deaths has fallen, postnatal deaths have become a lower proportion of all infant deaths, accounting for approximately one-third of such deaths. The single major cause of postnatal mortality is sudden infant death syndrome (SIDS), which accounts for approximately 8 percent of infant deaths among both whites and blacks but occurs about twice as often among blacks as among whites (Centers for Disease Control/National Center for Health Statistics 2005). Having infants sleep on their side or back was seen initially as an effective intervention and believed to account for substantial reduction in SIDS rates. Now, supine positioning is believed to be most effective in reducing SIDS risk (Malloy 1998; Wise 2003). As a result of public education, many fewer infants in all groups sleep in a prone position, but African Americans and mothers with less education have been most likely to retain or readopt the prone position over time and are least likely to use the supine position (Lesko et al. 1998; Pollack and Frohna 2002; Willinger et al. 1998). The specific factors accounting for these differences—whether in access to information, comprehension, or other barriers—remain unclear.

The general rule that the advantaged gain most with new advances has two exceptions that suggest possible ways of closing race and SES disparities. First, if interventions are structured so they have effects independent of the motivation, resources, or actions of individuals, they are less likely to be a source of disparities. Such interventions as fluoridating water supplies, removing lead from the environment, or requiring built-in safety features in vehicles tend to have these characteristics. Second, interventions can be developed that focus on disadvantage so as to provide more equality for persons with fewer resources (Mechanic

2002). Interventions like preschool enrichment, income support, early child nutritional programs, and additional educational opportunities are examples.

The Population Health Context: Social Structure and Culture

Initiatives at the population level are commonly based on the premise that wise social policies and interventions can substantially improve health and welfare and may have more pervasive effects than do individual medical interventions. But demonstrating such effects is difficult given the multiplicity of relevant factors and possible alternative interpretations. Some skepticism is important because commonly accepted interpretations of health trends—for example, the dramatic fall in cardiovascular disease beginning around 1960 attributed to changes in health behavior and new medical technology—on closer examination do not fit the data in any convincing way (Rothstein 2003; Stallones 1980). To be plausible, the changes in risk factors and the new medical technologies had to precede the downturn in mortality by a significant amount of time, but these changes occurred much later. Also, as Rothstein notes,

> The millions of persons affected by the coronary heart disease pandemic lived in two dozen countries with different social and economic structures, customs, traditions, diets, and work and recreational activities. Such diversity could never produce identical patterns of personal behavior that materialized and diminished simultaneously in all of the countries. (Rothstein 2003, p. 344)

After analyzing extensive data, Rothstein concludes that "changes in personal risk factors were not responsible for the decline" (p. 358), noting, in contrast, the clear evidence linking SES to coronary heart disease rates. Other studies found that education is particularly closely associated with coronary heart disease mortality (Davey Smith et al. 1998), an issue we will examine in more detail later.

Many efforts in public health and medicine to change health behavior in positive directions, like weight loss efforts, that often are successful in the short term either fail or have only modest effects in the longer run. They fail in part because they require continued individual awareness

and motivation, which are difficult to sustain given the structure of peoples' lives, work, and the environment. This is evident across a range of initiatives, such as improving nutrition, increasing exercise, and other positive health behaviors. Good health behaviors that flow naturally from everyday patterns of activity and experiences are more readily sustained than are artificial interventions imposed on already complex lives. Daily routines and occupations that require activities such as walking and using public transportation will be much more readily sustained than special exercise routines.

Social and behavioral scientists, epidemiologists, and medical historians, aware of the large and persistent relationships between social class and mortality around the world (Kunitz 2007), have long been intrigued by the fact that some very poor nations and regions have maintained low mortality over extended periods of time (e.g., Costa Rica, Cuba, and Kerala [India]), whereas other, much more affluent populations have done relatively poorly (Caldwell 1986; Cooper, Kennelly, and Orduñez-Garcia 2006; Schultz 1993). Generalization based on such selective comparisons among countries varying in national development, politics, culture, and geographic circumstances must be considered cautiously, and even skeptically, but nevertheless can provide useful insights.

Three factors beyond sufficient subsistence resources stand out. Educational attainment, whatever the context, is associated with advantages in longevity both within and across nations. Kerala in India is illustrative. Despite a low per capita income, Kerala achieved an impressively high level of literacy among men and women, in the vicinity of 90 percent (Sen 1992), and relatively low mortality. But Kerala has a long history of public policy relating to education, community medical care, public support of food distribution, and public and political activism. As in other examples given, with complex interrelationships between education and other nonmedical determinants, it is difficult to separate education and literacy specifically from these other influences.

A second and related factor, promoting better health and lower infant and child mortality, is personal empowerment, especially the education and empowerment of women (Caldwell 1986; Schultz 1993). In most societies, women are the family caretakers, preparers of meals, and monitors of family health. Poor countries with better health have better educated women and women more empowered and engaged in their communities. John Caldwell (1986) found that among the countries he studied, those with a high proportion of females in primary school in 1960 had the

lowest infant mortality rates and the highest expectation of life at birth in 1982. In contrast, those countries with a relatively high per capita income but relatively poor infant mortality usually discourage female education, contact between the sexes, and female social participation outside the household.

Female education is implicated in a wide range of family planning, fertility, child care, and other household activities and a variety of values and attitudes as well as activities outside the household. As Hannum (1998, p. 3) notes, "Compared to uneducated mothers, educated mothers attach a higher value to the health and welfare of their children; have greater decision-making power in health and other matters; are less fatalistic about disease, death, and cures; and are more likely to adopt innovative behaviors related to children's health." They also marry later, have smaller families, time births differently than do those less educated, and are more likely to work outside the household and earn a higher income and status. Thus there is no single pathway through which education functions.

A third factor that differentiates poor countries with favorable mortality rates from those with poorer health experiences is accessibility to basic primary health care services (Macinko, Starfield, and Shi 2003). Much of the impact of medical care involves basic preventive services that are cost-effective and, often, inexpensive. Countries that offer universal access to these resources and distribute them fairly have lower mortality rates and, presumably, better health.

Competing Interpretations to Social Class as "Fundamental"

Given the complex pathways through which education and other SES characteristics influence health, as well as the complex outcomes at issue, it should not be surprising that alternative perspectives linked to social class attribute primary importance to other factors. Here I address two of these perspectives, prenatal and early nutrition and cognitive capacity.

Fetal and Early Nutrition

An influential hypothesis associated with David Barker (1995) is that poor nutrition in middle to late gestation and in early life, leading to delayed fetal and infant growth, is a precursor of coronary heart disease,

stroke, high blood pressure, and type 2 diabetes in later life. Studying more than ten thousand men born in Hertfordshire, England, between 1911 and 1930, Barker found higher mortality rates for heart disease later in life among low birth weight male babies and those of low weight at one year. In a more recent example, Barker and his colleagues (2005) reported on 8,760 people born between 1934 and 1944 in Helsinki who attended child welfare clinics. The sample included 444 individuals who later died of coronary heart disease or were admitted to the hospital with this condition. Those who were later hospitalized or died from coronary heart disease were small at birth, thin at age two, but rapidly gained weight and body mass index between the ages of two and eleven. The researchers speculated that this pattern of growth is linked to insulin resistance, a precursor of coronary heart disease.

The Barker hypothesis has encouraged much research investigation and has both strong proponents and critics (Pickett 2002). Results of animal experimentation in a variety of species are consistent with the hypothesis that fetal nutrition can alter physiology so as to increase susceptibility to later disease, although there are important variations among species (Harding 2001). Also, natural experiments based on the Dutch famine in 1944/1945 (Davey Smith and Susser 2002; Susser, Hoek, and Brown 1998) and, more recently, the Chinese famine of 1959/1961 (St. Clair et al. 2005) support a link between poor fetal nutrition and some neurodevelopmental disorders. Barker (2004) has suggested a number of pathways through which early nutrition might affect varying disease states: by influencing how cells in key organs are formed; by causing differential settings of hormones and metabolism; or by introducing vulnerabilities to adverse environments in later life.

Others observe, however, that randomized controlled trials of maternal dietary supplements in pregnancy have had only small effects on birth weight. Moreover, even the very extreme famine conditions in Holland and China were associated with only modest observed effects. Jane Harding (2001) points out that the biology involved is complex and that maternal and fetal nutrition are separable issues depending on transfer capacity.

Prenatal and early nutrition are inevitably associated as well with a lack of resources, whether because of low income, inadequate parental education, or more unusual deprivations as occur in famines, so the causal pathways are indeed complex. It is plausible to focus on the more immediate biological precursors of birth weight and development, but more

powerful determinants in the ultimate outcomes of concern may be "upstream" in the causal pathway involving the social context of the family and particularly social class. It certainly is important to understand the relation between early nutrition and physical development, the mothers' nutritional history, uteroplacental blood flow, placental function, and fetal metabolism (Gillman 2005), but these factors are not independent of social context or social class.

Using the Helsinki data, Barker and his colleagues (2001) examined the interactions among body size at birth, growth, father's occupational status at birth, and education, occupational status, and income in adult life in relation to hospitalization or mortality from coronary heart disease. Low rates of fetal and infant growth, low educational attainment, and low occupational status each had independent associations with coronary disease. The growth of infants was retarded in families of low occupational status, and their subsequent educational attainment also was lower. The eventual educational level attained was associated with hospitalization and mortality beyond associations with poor infant growth. The researchers suggest that social class plays an important role by altering the conditions for growth and, thus, disease risk. They also speculate that deficient fetal and infant growth increases susceptibility to the effects of poor living standards.

If a child's later health is established for her partly in utero and very early in life, as Barker suggests, and is associated with her mother's health during pregnancy, which in turn is affected by early influences, then preventing prematurity and ensuring infant health are a multigenerational challenge (Lu and Halfon 2003). Potential biological pathways are suggested by work on dietary supplementation in mice, which has found that grandmaternal supplementation during midgestation affects subsequent coat color (Cropley et al. 2006). It is suggested that even grandmaternal diets may affect the incidence of diabetes and obesity (Cooney 2006). These types of possibilities require thinking about interventions much more broadly than the traditional focus on prenatal care, in which research has been unable to substantiate many of the optimistic claims (Goldenberg and Rouse 1998).

Cognitive Capacity

A specific challenge to the fundamental cause hypothesis, and a view sometimes held by physicians and others, is that the key factor

underlying the relationships between social class and disease and mortality outcomes is intelligence or cognitive ability. Barker and colleagues (2001) noted, for example, that intelligence may reflect childhood fitness related to long-term health. Gottfredson (2004) has contended this much more directly and strongly, arguing that intelligence is the fundamental cause underlying both better health outcomes and higher socioeconomic status, and she has reviewed many of the correlational data.

Multivariate studies in the United Kingdom have found that cognitive ability is indeed associated with various health outcomes. However, although it attenuates the size of associations between SES and health indicators, it does not explain them. An analysis using the Whitehall II Prospective Cohort Study and also a relative index of inequality that is weighted to account for the varying size of subgroups and adjusted for gender, cognitive ability, and various SES indicators found that cognitive ability had an effect independent of SES in only one instance: physical functioning in women. In contrast, many of the SES indicators retained independent effects when cognitive capacity was taken into account (Singh-Manoux et al. 2005). A cohort study in the west of Scotland reported similar results (Batty et al. 2006).

Link and his colleagues (2003) used two excellent major longitudinal U.S. data sources—the Wisconsin Longitudinal Study and the Health and Retirement Survey—that make it possible to examine SES and cognitive capacity in relation to mortality and ill health. They similarly found that both SES and cognitive ability are associated with mortality and other health outcomes. However, analyses from both data sources indicate that controlling for cognitive ability only modestly modifies the SES/health link, which still remains statistically significant. In contrast, controlling for SES reduces the effects of cognitive ability to nonsignificance.

The case for cognitive capacity as the crucial factor driving both social class attainment and health remains highly speculative, with no direct support. This, of course, does not make it unimportant in the chain of health influences.

Strategies for Prevention

The concept of fundamental causes has implications for how we think about preventing disease and mortality. Most medical and epidemiological studies regard social class as a crude proxy for more proximal

biological and behavioral factors, providing clues to the "real" causal factors. According to this view, social class is primarily important because of its associations with other factors that play a more immediate role in disease processes. Preventive efforts are then concentrated on these specific risk factors. Persons with many risk factors have a higher propensity for disease and death, but much, perhaps most, disease in populations occurs in persons in the normal range of recognized risk factors (Rose 1985). This commonly results in proposals to intervene medically at lower risk thresholds with more individuals near or at the normal range, for example, in such areas as blood pressure and cholesterol control.

The *British Medical Journal* published several articles in June 2003 suggesting a strategy for reducing cardiovascular disease and mortality with a polypill containing a statin, three hypertensives, folic acid, and aspirin, taken by everyone with preexisting cardiovascular disease and everyone over the age of fifty-five (Rodgers 2003; Wald and Law 2003). The analysis suggested that the polypill could reduce cardiovascular mortality by 80 percent (Wald and Law 2003). One implication of the risk factor approach, however, would be to have almost everyone medicated.

In a jocular response, Franco and colleagues (2004), using similar estimation methods, suggested the alternative of polymeals consisting of seven food components (chocolate, wine, fish, nuts, garlic, fruit, and vegetables) that they estimated could reduce cardiovascular disease by comparable amounts. Unlike advocates of the polypill, they had few ideas about how such a diet could realistically be achieved. In any case, the assumptions made about how risk factors are distributed in populations have an important bearing on strategies directed to populations versus those aimed at persons at substantial risk or already ill.

Manuel and colleagues (2006), for example, estimated that 4 percent of Canadians with a high baseline risk of coronary heart disease accounted for 35 percent of predicted deaths. In assessing alternative prevention strategies, they concluded that focusing on treatment with statins for those with high baseline risks would save many more lives than would a broader population health approach involving many relatively low-risk persons. Similarly, an analysis of reductions in cardiovascular disease mortality in the United States estimated that only about one-quarter of the 34 percent reduction between 1980 and 1990 was due to reducing

risk factors in the general population without coronary heart disease (primary prevention). An estimated 29 percent of the reduction came from reducing risk factors by treating the much smaller population already with coronary heart disease (secondary prevention). An additional 43 percent of the reduction was estimated to come from improvements in treatments, other than the reduction in risk factors, among persons with coronary heart disease, including the now common cardiovascular and surgical interventions, such as revascularization procedures (Hunink et al. 1997).

Optimal strategies for addressing individual diseases vary by time and place, by prevalent patterns of risk and behavior in populations, and by the available knowledge of effective treatments and interventions (Jackson, Lynch, and Harper 2006). The use of combined strategies of both primary and secondary prevention is feasible and makes much sense when effective interventions are available.

Changing a population's characteristics and conditions that affect a range of diseases is particularly useful. Such an approach concentrates resources to modify factors affecting the rate of incidence of various diseases in populations, for example, behaviors such as smoking associated with cancers, heart disease, and other illnesses or such factors as SES or social support.

Education as a Central Point of Leverage

As indicated, the components of SES, as well as many other important influences, are interrelated in complex ways over the life course and also interact in important ways with culture and social context. Moreover, efforts to establish causality in population health studies between SES indicators and health also must take account of reverse causality (Goldman 2001; J.P. Smith 1999) because health itself affects occupational attainments and earning ability. From this point of view, education is often seen as an advantageous SES indicator because for much of the population, educational attainments are established before illness sets in. Nevertheless, there is some reverse causality, sometimes beginning in early life, as the Barker studies indicate.

The evidence for the importance of education across time, place, and diverse measures of health is quite robust (Cutler and Lleras-Muney 2006; Elo and Preston 1996; Mechanic 1980, 1989, 1994; Preston and

Haines 1991). Educational attainment also has had an important place in theories of social development, modernization, and determinants of health (Mechanic 1994; Stiglitz 2006). Education could create opportunities through varied pathways including increased cognitive complexity and skill, greater knowledge, better coping capacities, and improved access to better and safer jobs and higher incomes. Education empowers individuals, builds self-esteem, and encourages civic engagement (Inkeles 1983; Inkeles and Smith 1974). It provides more personal control over work and many other aspects of one's life, as well as access to more advanced and usable knowledge. Education probably also influences deferral of gratification in constructive ways and encourages greater investment in one's own health and the health of other family members. Cultural factors, of course, have unique influences on health (Kunitz 2007), but education often interacts powerfully with these influences.

Education may be particularly important to behaviors that require knowledge and cognitive capacity, such as those required to detect important symptoms early in their course, to seek information about the best sources of care and treatment, or to follow complicated medical regimens. Thus, it is not surprising that statistical controls for cognitive capacity attenuate educational effects on health more than on income and wealth effects (Batty et al. 2006; Singh-Manoux et al. 2005). In contrast, different occupations and occupational ranks expose workers to varying dangers (including pollution, toxic substances, noise, and risk of accident) and varying levels of supervision and lack of control over work associated with adverse health outcomes. Economic resources, in turn, are more supportive of good housing, sound nutrition, positive neighborhood environments, and so on.

Although there is a social gradient between the number of years of schooling and measured health outcomes, uncertainty remains as to whether added years of schooling have the largest effects on health among those with modest educations or those with education well beyond high school. In some studies, and particularly in developing countries, primary schooling that provides literacy and basic skills seems to make a large difference, but education beyond primary school is associated with better health as well (Curtin and Nelson 1999). In the American context, studies find that differences in years of schooling relative to health are substantial beyond some important threshold such as completing high school. The effects of schooling should probably not be seen as

absolute but relative to the particular health outcome in question and to educational levels in the community overall (Cutler and Lleras-Muney 2006 provide a good review). Conversely, it is plausible that income would have the largest influence until comfortable levels of subsistence are attained, whereas more years of schooling might continue to provide greater advantages in maximizing the benefits of medical knowledge and technology as well as enhanced occupational opportunities. The types of multivariate studies that allow us to examine the relative influence of income, education, or other factors cannot easily resolve the issue because such studies usually do not take account of the cultural context, which gives different meanings to the educational experience and its impact. More education, for example, without commensurate employment opportunities may not have the anticipated positive effects and, in some circumstances, may breed frustration and alienation.

Education enables many kinds of valuable behaviors relevant to health, but the way that it functions may depend on exogenous norms and values. As Kunitz observed,

> Studies of social disparities in health are often written as if behavior and culture are simply what happens when people don't have enough money or are not well enough educated to make rational choices regarding their health. They miss the ways in which communities at all income, educational and occupational levels shape behavior. (2007, p. 89)

But even while acknowledging the importance of specificity, the evidence still is impressively broad across time, place, and diverse health outcomes that education has important effects on many health indicators independent of other social class components (Cutler and Lleras-Muney 2006; Mechanic 1980, 1989).

As used in most studies, years of schooling is a crude proxy for the acquisition of skills, knowledge, and personal agency and lacks an indication of content or quality. Attending school does not necessarily ensure that children are exposed to a culture that promotes the types of skills and orientations that we believe are most important to health, and the culture of schools themselves may divert students from values associated with achievement and health (for a classic study, see Coleman 1961). In this sense, as used in health studies, schooling remains a "black box" that needs deeper investigation. Also, it is important to keep in mind

that school entry, progression, and continuation beyond the level prescribed by law remain a complex social selection process. Accordingly, we must be careful not to attribute influence to schooling that more reasonably results from the capacities, characteristics, culture, and values of families and individuals. This may be especially true in parts of the world where children are kept in school at great cost despite significant economic and other adversities. Some studies that compare localities with different mandatory educational requirements try to deal with this selectivity, but it remains a nontrivial issue for research and policy considerations.

In a recent exercise, researchers estimated the numbers of deaths that would have been averted in the United States between 1996 and 2002 if mortality among the lesser educated had been the same as among those with at least some college education (Woolf et al. 2007). They estimated 1.4 million fewer deaths, eight times more than those that might be attributable to medical advances. Because education is associated with so many other variables and pathways relevant to health outcomes, it is apparent that much education-associated mortality cannot be reduced simply by bringing everyone up to some educational standard. Nevertheless, the literature suggests that by elevating educational attainment, significant health gains are likely.

If we better understood the educational pathways to important health outcomes, we might find more efficient ways to enhance health than simply by enhancing educational opportunities and quality. What makes educational initiatives particularly strategic points of policy leverage is that even if their impact is much less than we think, the interventions themselves are highly valued and important on their own terms and strongly endorsed across the social and political spectrum. Thus, education is the component of social class most easily modified. While other social class–related initiatives relating to income security, housing, occupational safety and other working conditions, and the like have considerable importance for health, they quite typically face ideological and political resistance and social divisions that make policy difficult. This should not preclude efforts for change. But the strong coalition around education provides leverage to promote important health goals in uncontroversial ways while we continue to increase our understanding of pathways to health and build public support for additional population health initiatives.

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