



Social Epidemiology: Social Determinants of Health in the United States: Are we Losing Ground?

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Abstract

The United States ranks in the lower tiers of OECD countries in life expectancy, and recent studies indicate that socioeconomic inequalities in health have been widening in the past decades. Over this period, many rigorous longitudinal studies have identified important social, behavioral, and environmental conditions that might reduce health disparities if we could design effective interventions and make specific policy changes to modify them. Often, however, neither our policy changes nor our interventions are as effective as we hoped they would be on the basis of findings from observational studies. Reviewed here are issues related to causal inference and potential explanations for the discrepancy between observational and experimental studies. We conclude that more attention needs to be devoted to (a) identifying the correct etiologic period within a life-course perspective and (b) understanding the dynamic interplay between interventions and the social, economic, and environmental contexts in which interventions are delivered.

THE STATE OF THE STATE: SETTING THE STAGE FOR UNDERSTANDING SOCIAL DETERMINANTS OF HEALTH IN THE UNITED STATES

The United States has consistently ranked in the bottom half or bottom third of OECD (Organisation for Economic Cooperation and Development) countries in life expectancy in the past several decades. In 2005, the United States ranked 24th among 30 OECD countries, with a life expectancy of 77.8 years (58). Recently, the United States has been confronted with a new possibility that some segments of our population may either be losing ground as compared with others or may even be experiencing absolute declines in critical indicators of health and well being (17). At a time in U.S. history (and, in fact, in a number of industrialized nations) when relative disparities in a wide range of health outcomes may be increasing, it is important to turn our attention to understanding why this is occurring and what we can do to reduce social and economic health disparities. Although social inequalities in health are persistent over time and in all countries, the magnitudes of the differences are highly variable across time and place (46, 47, 62, 72), which suggests that we can reduce health disparities if we can better understand the forces that determine the magnitude of the differences among different social, economic, and racial/ethnic groups.

A host of recent papers have documented this rise in disparities in the past several decades. Some have focused on geographic disparities without regard to social or economic conditions (17). Ezzati and colleagues, for instance, have recently shown, in an examination of U.S. county mortality data between 1961 and 1999, that after 1983, 180 counties for women and 11 counties for men experienced declines in life expectancy. In the period between 1961 and 1983, no counties experienced such declines. Although the percentage of the total population impacted by these county-level changes is small, it nonetheless may signal an important trend. Because many other counties experienced substantial improvements in life expectancy during

this period while others stagnated, county-level health disparities widened during this same time. Epidemiologists and economists have explicitly noted the rising health inequalities in the United States related to socioeconomic conditions and among racial/ethnic groups (19, 30, 36, 53, 62, 73). Meara (53) reports that life expectancy hardly changed for people with low levels of education over the 20-year period from 1981 to 2000, and among women with low levels of education, it actually declined during that period. For men and women with higher levels of education, life expectancy at age 25 improved 1.8 years for white men, 1.0 years for white women, 3.3 years for black men, and 1.6 years for black women. Harper and colleagues (30) likewise report that black/white gaps in life expectancy have been growing since the mid-1980s. Augmenting this picture, Krieger and colleagues (37) show that the widening socioeconomic and racial/ethnic relative and absolute disparities in premature mortality and infant mortality in recent decades were preceded by a narrowing of these inequities that started in the mid-1960s and extended up to 1980. Almond et al. (1) also reported shrinking black/white disparities in infant mortality in the U.S. rural south in the mid-1960s, followed by an increase. Such short-term temporal changes, including shrinking as well as widening of the magnitude of disparities, suggest strong environmental forces are at play.

Increasing inequalities in health occurring over the last several decades have been documented for a large number of European countries and for the UK as well during this period (45, 69, 76) with some variation in the extent of change and some reports of stable risks especially in Nordic countries (38). Substantial improvements among the best off in many countries serve to widen differences, leaving the poor and disadvantaged farther behind even as population means improve. An important note to understanding this pattern is to recognize the differences between relative risks and absolute risks. In some cases, as population health improves, relative risks may grow even as absolute risks decline. Krieger (37) has noted that

this association is not consistently linked and that it will be important in future studies to show both absolute and relative risks. Studies by Mackenbach (45) in European Union countries show widening relative socioeconomic inequalities occurring in a number of industrialized countries. Others have documented the difficult circumstances in former Russia and in many Eastern European countries that have experienced declines in life expectancy (70). In fact, some demographic forecasts indicate that continued improvements in life expectancy may not continue into the future as they have occurred in the past. These demographic forecasts are based on changes in behaviors related to tobacco consumption (66) and obesity (18, 39, 59, 60) as well as to rising socioeconomic inequality and patterns of immigration and social exclusion and isolation (51).

On the basis of national averages for an outcome such as life expectancy, it is well known that for many years the United States has not performed nearly as well as one would expect, given both our gross domestic product (GDP) and our spending on health care. The United States may be doing particularly poorly vis à vis countries we consider as quite comparable to us in GDP such as the United Kingdom. For instance, a particularly interesting paper by Banks and colleagues (2) showed that among people in similar socioeconomic positions, men and women in the United Kingdom have a considerable health advantage compared with their U.S. counterparts for a number of health outcomes ranging from biomarkers of cardiovascular and metabolic risk to self reports of chronic conditions. The prevalence of diabetes in the United States was 9.5 and 8.2 for those with the highest levels of education and income, respectively, compared with rates of 5.7 and 4.4 in U.K. counterparts (2). Even though these upper-socioeconomic status men and women have access to the best possible medical care in the United States, they still assume some risk just by living in the United States.

Although social epidemiology as a field has perhaps made its greatest advances over the past three decades, it has occurred at a time when

health inequalities have widened across many countries. This situation implores us to examine both the methodological approaches to document social determinants of health, and it challenges us to understand the drivers of social disparities in health so that we can better inform policy, improving both population health and reducing disparities. On a more positive note, even though social inequalities in health are pervasive and present in virtually all societies examined, the magnitude, both absolute and relative, varies widely across time as well as among countries or states or provinces within countries. This latter finding, along with the several policy initiatives within countries and across countries (33), implies that by identifying the forces that serve to reduce inequalities, those in public and private sectors can better implement those actions and policies in all places.

Because the trends reviewed above clearly identify that some environmental conditions increase or decrease health disparities even as many countries have experienced overall improvements in population health, the aim of this article is to explore two major paradigms that are fundamental to social epidemiology to see if they can help us understand these current trends in health. The first is related to integrating a life course perspective into planning interventions and interpreting the reasons for changing patterns of health. The second is related to Rose's paradigm (67) about the determinants of population health and understanding contextual-level influences on population health. Both these perspectives can help investigators interpret the trends in mortality in the United States and in many other countries. The two perspectives also help us to understand why observational and experimental data have so often come to different conclusions about social and behavioral risks to health and have led us to more effective interventions. It is critical to reconcile these divergent results from observational and intervention studies to improve population health and reduce health inequalities. Thus, the aim of this review is not to compile the literature on each of the major social

RCT: randomized controlled trial

determinants of health (e.g., socioeconomic conditions, social networks, discrimination, work conditions) but quite the contrary: to identify provocative and important paradigms that can help us interpret research findings so that public health as a field can move forward substantively, methodologically, and theoretically.

Life course issues have recently come to permeate thinking about a broad number of exposures in public health. It is now commonplace to think of critical or sensitive periods in exposure to risk as well as to understand dynamics related to cumulative exposure. And yet, there is little consensus about which specific exposures are related to which life course dynamics for which health outcomes. Life course perspectives have been the focus of much recent attention and have implications for study design and causal inference as well as for points of intervention. First, life course models pose interesting challenges to classical approaches to randomized experiments as well as to observational studies. Second, a review and critique of Rose's paradigm of population health indicate ways in which this paradigm is useful in interpreting patterns of health and well being and in shaping interventions. The paradigm may have limitations in helping determine for whom new interventions should be planned. This paradigm while of interest in many areas of public health has been centrally important to social epidemiologists because of the way in which Rose originally challenged us to think about the macro forces that might shape population patterns of disease prevalence and distribution of risk. Recently, this work has been scrutinized more critically by epidemiologists.

INTERPRETING THE RESULTS OF SOCIAL AND BEHAVIORAL INTERVENTIONS FROM A LIFE COURSE PERSPECTIVE

Over the past two decades, a series of randomized controlled experiments hoping to change social, behavioral, and psychological conditions have produced null or modest results in terms of

improving health outcomes. To be sure, some have had greater impacts than others, but on the whole, most of the investigators leading these interventions hoped the results would show much stronger effects, considering the magnitude of risks reported in observational studies. The adoption of a life course approach provides insight into why trials may have not yielded the effects anticipated on the basis of observational studies. Because life course approaches attempt to identify the etiologic period of risk, they are critical to the design of interventions. Life course issues have permeated much of epidemiology (41, 42, 49, 56, 57, 61, 64, 74). Although life course approaches were once commonplace only for developmental outcomes, epidemiologists have come to recognize that concepts related to etiologic period, latency, and sensitive periods are all relevant to epidemiology (29, 31, 65).

Although there has been some debate among epidemiologists about the value of randomized controlled trials (RCTs) as an effective study design in which to test large-scale social and behavioral interventions, experimental designs have critical advantages related to randomization. They limit the kinds of confounding and selection issues that are inherent in many observational studies. Furthermore, ultimately public health sciences are aimed at improving population health not just furthering basic sciences related to understanding the world around us. To accomplish this goal, we need interventions and/or policies that have public health impact. Using experimental approaches, whether they are full-scaled randomized trials or more quasi-experimental approaches building on natural experiments, is important in public health. Experimental approaches help to identify interventions that work and identify the critical periods in which interventions will maximize public health benefits.

To discuss issues related to life course dynamics and subsequently to understand the need to embed interventions more deeply into the social conditions in which people live, I draw on two recent RCTs related to social support and social network interventions: The two

studies are Enhancing Recovery for Coronary Heart Disease Patients (ENRICHD) and Families in Recovery from Stroke (FIRST). The results of these interventions, and in fact, their designs, serve to illustrate the issues related to life course and social context.

ENRICHD was an RCT aimed at improving social support and reducing depression in post—myocardial infarction (MI) patients (7). The primary outcome was the reduction of reinfarction and all-cause mortality. ENRICHD enrolled post-MI patients from more than 80 hospitals and 8 clinical centers across the United States ($n = 2481$). ENRICHD was based on evidence from a large number of longitudinal observational studies indicating that both depression and social support were related to survival post MI (13). Most of the studies indicated that both men and women who were depressed or were socially isolated had elevated mortality or reinfarction risks ranging from ~2 to 4 (9, 11, 14, 35, 77). Results from related clinical trials were inconsistent (23, 24, 26). The results from M-HEART, a study (22) published just before the launch of ENRICHD, showed null results but suggested a trend that women in the intervention group fared worse than women in the usual care group ($p = 0.064$).

With a follow-up of 3.4 years using an intent-to-treat analysis, there were no differences between the intervention and control groups in ENRICHD ($p = 0.89$) (7). In fact, the survival curves completely overlap when looking at the primary endpoint of reinfarction or all-cause mortality. The ENRICHD study did find reductions in the mediating conditions related to depression and low social support, suggesting the intervention was changing the conditions upon which it was designed to intervene (7). The magnitude and long-term differences were smaller than expected, however, causing concerns about the intervention's effectiveness. At six months, at the conclusion of the main intervention, however, investigators noted significant differences between the intervention and usual care (UC) groups in social support and depressive symptoms.

Subgroup Differences in Outcome: Does Social Context Influence Treatment Outcomes?

The most intriguing and controversial analysis from ENRICHD is the subgroup analysis of outcomes looking at gender and racial/ethnic differences. In prespecified analyses, statistically significant interactions between gender and the intervention revealed that men in the intervention had better outcomes than did men in the UC group. Women in the intervention group had worse outcomes than did women in the UC group. In posthoc analysis with stratification by both gender and race/ethnicity, one trend demonstrated that white men benefited from the intervention ($HR = 0.80$, $p = 0.10$), whereas other groups showed no benefit from participating in the intervention (68). Furthermore, using outcomes related specifically to cardiovascular disease, white men had a risk of 0.63 ($p = 0.004$) of reinfarction or cardiovascular mortality, and white women and black men and women experienced no benefit from being in the intervention group. See **Figure 1**. White men were more likely to be married and better educated; have the fewest chronic conditions, better ejection fractions, and less severe myocardial infarctions; and were more likely to receive thrombolytic therapy and cardiac catheterization and coronary revascularization. None of these conditions accounted for the difference in outcomes by treatment group seen in ENRICHD. However, such differences might suggest that unmeasured covariates may account for these gender and racial/ethnic differences.

A second much smaller RCT designed to improve functioning in stroke patients was based on social network intervention models (27). This trial was conducted with 291 patients from 8 Boston-area hospitals and rehabilitation centers. The intervention was based on family systems and cognitive behavioral therapy. It aimed to enlarge social networks, improve social support, and increase recovery efficacy and effective problem solving. The primary outcome was functional independence

ENRICHD:

Enhancing Recovery in Coronary Heart Disease Patients

UC: usual care

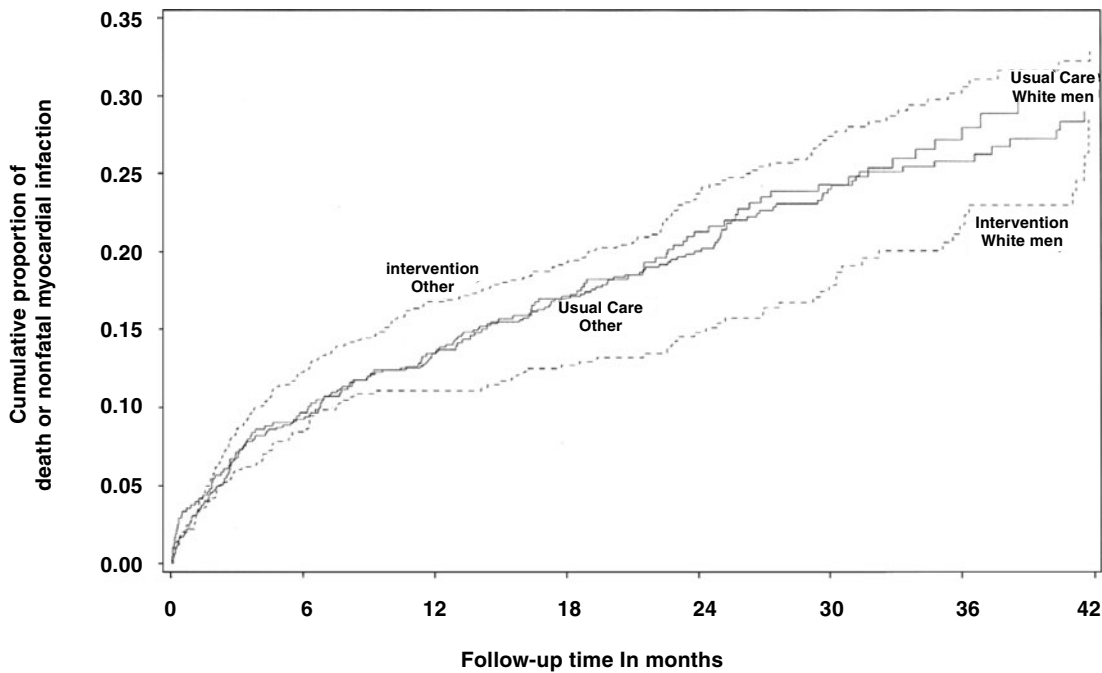


Figure 1

ENRICHD cumulative all-cause mortality and nonfatal MI by gender, race/ethnicity, and treatment group (69).

at six months poststroke. The results of this trial showed no difference between intervention and UC in the primary endpoint at either three or six months, using an intention-to-treat approach (28). An examination of prespecified subgroups revealed that those who were not depressed and had little cognitive impairment, more minor strokes, and fewer preexisting chronic conditions tended to benefit from the intervention. Those who were frailer tended to do better in the UC group compared with the intervention group. Post-hoc subgroup differences were very revealing in this study as well. A frailty summary score based on the above conditions (16) showed that among nonfrail participants, those in the intervention group did better than did those in the control in functional outcomes ($p = 0.001$) and they had lower mortality rates ($p = 0.03$). Among those who were frail, those in the UC group tended to do better in terms of functional outcomes and mortality risk. See **Figure 2**.

The results of these two trials, also coupled with the results from M-HEART, consistently demonstrated overall null results from the trials when using an intent-to-treat analysis. The intervention may have had more positive results in some subgroups, whereas in other subgroups, those in the UC group did better. Some investigators suggest that frailer, less healthy participants actually fared worse in the intervention group when compared with the UC group. We are left with a stark contrast between the observational studies, which indicate that those with low levels of social support and weak networks and high levels of depression were at substantial risk, and the results of the trials, which showed null results in intention-to-treat analysis. In post hoc analyses, there is some indication that the interventions may have been harmful in some subgroups. Only a few types of hypotheses can explain the discrepancy between the findings from the observational studies, which almost uniformly show strong ties

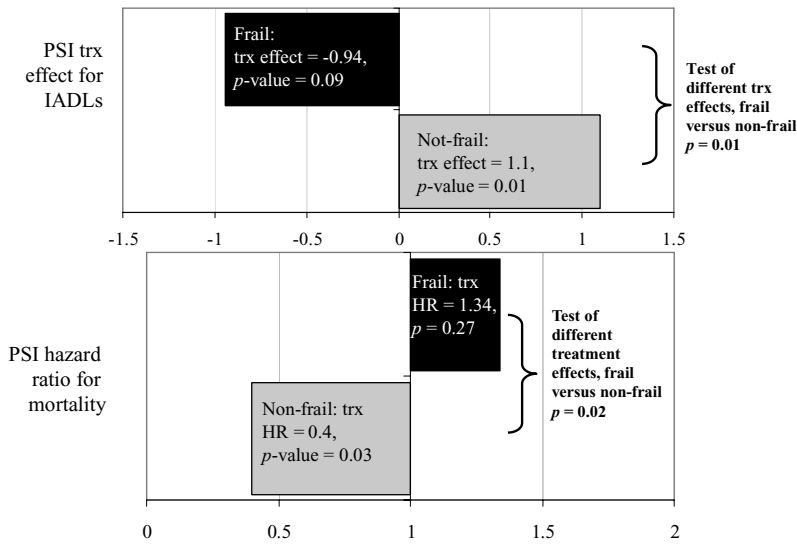


Figure 2
FIRST: differential treatment effects by frailty (16).

between these exposures and health outcomes, and the results of the RCT,s which show much weaker results. The first explanation is that the interventions failed to change the exposure or, to be more specific, did not change the exposure sufficiently or during the correct etiologic period. The second explanation posits that the exposure is causally related to the outcomes and that the intervention changed the exposure but treatment effects were heterogeneous. The third explanation proposes that the exposures are not causally related to the outcomes and the observational findings are the result of confounding by some unmeasured variable or reverse causation. The contrasts between findings from observational studies and randomized clinical trials are growing, and discrepancies in findings have been observed for a number of recent interventions. Of particular interest is the possibility that observational studies rarely help us identify the etiologic period clearly enough to know when to intervene. Thus, many interventions may be targeted at a population when their period of etiologic risk has largely passed.

Most observational studies identify a risk factor at one period in time. In such studies, it is impossible to identify the etiologic period

of risk. For some exposures, especially those related to tobacco consumption and selected cardiovascular risk factors, longitudinal information on exposures over long time periods does exist, and epidemiologists can start to identify etiologic periods of risk with some accuracy. In social epidemiology, we are just starting this research endeavor. Life course models identify where exposures may have the most important impacts. Three distinct trajectories have been linked with each of the most common life course models (6, 31, 40, 65). The first life course model that has been dominant in developmental studies is related to critical or sensitive periods in which early childhood or even prenatal exposures shape subsequent outcomes that may or may not be evident for years. In this model, early exposures shape subsequent outcomes independently of later experiences or changes in exposure. The exposure may not lead to obvious outcomes until later life owing to some latency period. In the second life course model, exposures throughout life have a cumulative effect. In such cases, there do not appear to be sensitive periods, but rather, it is the exposures over many years that have the largest impacts. In the final life course model, early exposures may shape

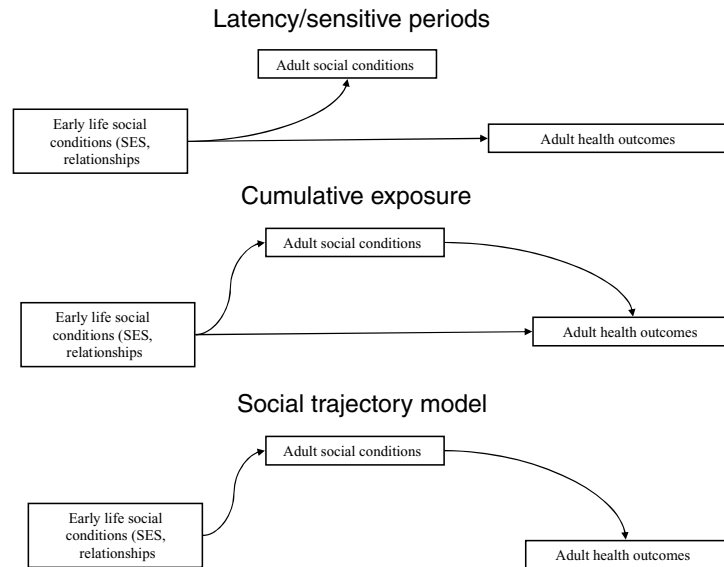


Figure 3 Three life course models of disease: latency, cumulative exposures, and social trajectories.

opportunities or barriers to critical exposures in later life, which are themselves the critical exposure linked to disease outcomes. This latter model is often called a social trajectory. In the next section, each of these three models is discussed in greater depth (all three models are shown in **Figure 3**).

Sensitive Periods and Latency: Childhood Origins of Adult Health

Developmentalists interested in early development and childhood have focused for decades on the importance of early life exposures in shaping cognition and brain function (71). Over the past two decades, epidemiologists have come to understand the early origins of diseases, often focusing on fetal origins, which evidence suggests shapes patterns of metabolic function related to diabetes and other health outcomes (3, 4). The causal pathway invoked in this trajectory can be seen in the top diagram of **Figure 3**. According to this causal diagram, early life conditions (in this case social conditions of interest) become embodied immediately and may go on to influence either adult social conditions or

adult health outcomes. In this model, there is only a causal link between early exposure and subsequent adult disease, with no pathway leading from adult social conditions to adult health. In this case of early embodiment, intervention in adulthood cannot offset the harm incurred in childhood.

Which types of exposures and outcomes are linked to this trajectory? Many examples relate to cognitive and brain development in both animals and humans. Of interest to us, though, are exposures related to both social experiences and health outcomes. Examples here are harder to find, but some interesting studies do exist. For instance, with regard to cognitive function in old age, Meaney has produced a host of studies related to nurturing experiences in early postnatal life in rats (10, 12, 15, 52, 55, 63, 75, 78). In these experiments, rats were randomized to handling and not handling postnatally. Rats randomized to both groups were very similar to each other at earlier ages, but by midlife and at older ages, the nonhandled rats developed significant cognitive impairment and simultaneously had higher levels of corticosterone. More recent studies by Meaney and colleagues show

differences in epigenetic processes, leading to behavioral outcomes (20, 21).

Cumulative Exposure Over the Life Course

Many epidemiologists interested in life course issues hypothesize that most adult disease is not likely the result of early childhood or prenatal exposure but rather as the result of a lifetime of accumulated exposures (43, 44). Such a model can relate to early exposures and simultaneously to adult exposures because it is the impact of cumulative exposures across the life course that takes a toll at older ages. Early experiences may produce some independent impact on outcomes but that is not the central issue in this model. In this model, the etiologic period is long and covers decades of an individual's life, starting either in early childhood or in adulthood. In this model, we see that even if early experiences set people up for adult experiences, it is the cumulative impact that is critical. Of central importance to the development of effective interventions is the understanding that intervention in adulthood can offset some of the harm caused by the exposures. This process is illustrated in the middle diagram of **Figure 3**, in which causal arrows go from the adult experience/exposure to the health outcome, even though a causal arrow extends directly from the early exposure to adult health outcomes. Ben-Shlomo & Kuh (6) as well as Lynch & Davey Smith (43) offer fuller reviews of life course models in epidemiology. They point out that in risk models of accumulative exposure there can be both independent and uncorrelated insults as well as correlated insults with risk clustering or chains of risk.

One of the risk factors we know the most about is tobacco consumption. We put tobacco consumption into this life course model to understand effective points of intervention. For instance, almost all smokers start to smoke as adolescents. If we had a goal of preventing tobacco consumption, our goal would most likely be to stop adolescents from starting

to smoke because quitting is very difficult. Once people start smoking, the effects on specific disease outcomes are varied. Quitting after the diagnosis of lung cancer does not change the prognosis related to lung cancer because the cumulative impact has already taken its toll to produce disease. However, quitting after a diagnosis of heart disease can alter prognosis because quitting produces an immediate effect that soon alters cardiovascular function. In these two cases, there are different etiologic periods, and because epidemiologists have studied tobacco exposure so well for so long, we have a good understanding of the differential impacts that exposures have on specific disease outcomes. Unfortunately, data on social exposures is much more limited.

With regard to the relation between, for instance, socioeconomic disadvantage and cardiovascular disease, data from a number of studies suggest that both early life and cumulative exposures over the life course play a role in disease etiology. Evidence suggests that early exposures, even fetal exposures, play a role in shaping risk of cardiovascular disease. Differences in blood pressure, cardiovascular reactivity, and metabolic function are evident in childhood and early adulthood (5, 25, 50). Early life anthropometry is associated with adult cardiovascular disease (41). Although early events may shape risks and perhaps trajectories of risks, it is also becoming increasingly clear that accumulated exposures over the life course impact cardiovascular risk in old age. For instance, studies from Alameda county in the United States (44) and from the GAZEL study of French Gas and Electricity workers show that socioeconomic disadvantage experienced multiple times over adulthood increases the mortality risks net of early childhood experiences (54). In fact, the preponderance of evidence would suggest that sustained economic disadvantage is strongly associated with many health outcomes (42). This model of cumulative risk argues that changes in adulthood to modify risk or prevent the onset of risk will partially offset the risks set by trajectories in early childhood.

Social Trajectories of Risk

In a social trajectory model of health and disease, early life exposures impact adult exposures, which in turn directly influence disease risk. In the bottom diagram of **Figure 3**, the causal pathways indicate that early life exposures do not directly affect adult health. They influence adult social conditions, which, in turn, affect adult health. In this case, intervention in adulthood can completely offset harm incurred in childhood.

A clear example of such causal patterns relates to occupational exposures. One can imagine that early life experiences, education, and training in young adulthood place people on a trajectory to obtain certain jobs. Certain occupations, however, carry with them risks related to the physical environment (toxic exposures to chemicals or ergonomic risks) as well as to challenging and stressful conditions. These job exposures have risks related to a multitude of poor health outcomes involving cardiovascular, musculoskeletal, and cancer disease. In this model, altering the job exposure will completely offset the individual's risk. Even though specific social or other experiences in childhood may place people at risk for entering certain occupations, risks can be substantially reduced by interventions aimed at adult exposures, especially at the work site level. Parallel findings may be related to other adult exposures related to neighborhood or other adult contexts.

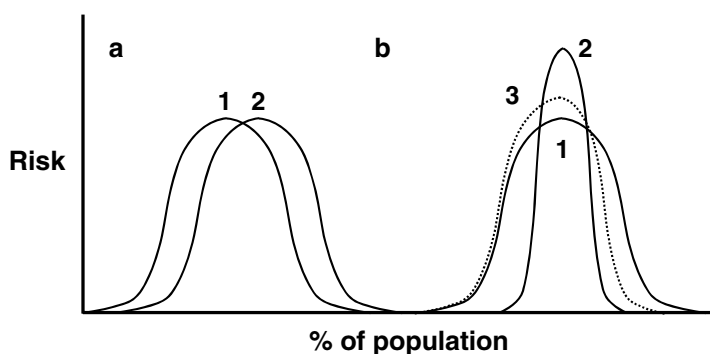


Figure 4
Variations on risk distributions based on Rose's paradigm.

ROSE REVISITED: THE DYNAMICS OF POPULATION HEALTH AND THE ROLE OF SOCIAL CONTEXT

In the mid 1980s, Geoffrey Rose, a British cardiovascular epidemiologist, started to write about population health, making a clear distinction between understanding what might produce sick individuals as opposed to what produces sick populations. By 1992, he had written a book, *The Strategy of Preventive Medicine* (67), which framed his ideas into a coherent story. "The critical insight of Rose's was that an individual's risk of illness cannot be considered in isolation from the disease risk of the population to which the individual belongs" (8). Rose makes two points, which follow from this paradigm. The first indicates that the mean of the risk distribution influences the tail of the distribution, implying that it makes little sense to change the tail without understanding that it belongs to the population. He uses a host of cardiovascular risk factors to illustrate this point. The second point demonstrates that the distribution of risk produces a "prevention paradox" in which a large number of people with a relatively modest risk may produce more cases of disease than would a small number of people at high risk. These two phenomena give rise to the need to shape prevention strategies that acknowledge the entire risk distribution and rarely target high-risk individuals or segments of the population. Most investigators have interpreted his findings to support strategies that shift the risk distribution entirely to a lower-risk position. **Figure 4a** illustrates the shift from a high-risk distribution (curve 1) to a lower-risk distribution (curve 2) without changing the shape of the curve. The paradigm is compelling and serves public health well to suggest that rather than focusing on a rather small number of individuals who are embedded in some larger population, those in public health identify the forces that shape why specific populations have particular risk distributions.

Over time, however, several epidemiologists have questioned some of the assumptions Rose

made, particularly concerning risk distribution (34, 48). Although the paradigm serves as a helpful heuristic for understanding population dynamics, the paradigm might not apply in some cases exactly as it did in Rose's examples of coronary heart disease in the 1970s and 1980s. Most of Rose's examples show (a) the distribution of risk to be relatively normal with few people in the tails of risk and (b) relative risks increasing continuously. This specific distribution and relative risk give rise to the prevention paradox. Rose said the "distribution of health-related characteristics move up and down as a whole: the frequency of cases can be understood only in the context of the population's characteristics" (8, 67). For instance, in the United States, obesity has been on the rise over the past few decades, and a careful examination supports Rose's proposition that the entire risk distribution has shifted during this time. In fact, the mean has driven the tail.

However, if increasingly more people are in the tails of the distribution or if the distribution itself is not normally distributed, then Rose's strategies may not be effective, and specific high-risk strategies may become more effective. In fact, this rather straightforward empirical question has rarely been examined. Similarly if relative risks do not increase linearly or show clear threshold effects, different actions may be appropriate. In some instances, reducing population risk brings people to the formerly low risk side of the distribution into higher risk. For example, reducing overweight at the population level may produce more people with eating disorders at the very low end of the weight distribution. So, for instance, the curves in **Figure 4b** show the normal distribution again with an illustration of the optimal shift in the curve if the goal is to reduce the high-risk end of the curve but to maintain the distribution of the curve below the mean so as not to increase risk (see **Figure 3b**). Some evidence also indicates that if the risk distribution is not normally distributed or if many are in the extreme tails of risk, a population strategy of risk reduction may not always produce the same results. Rose himself acknowledged that under certain circum-

stances, high-risk strategies may be more effective on a population level than population-wide strategies would be. Certainly, from a purely hypothetical perspective, if the risk distribution is very skewed, Rose's paradigm may be more problematic. This issue at hand is whether this is actually the case with regard to some risks or if it is a purely hypothetical situation (32, 34).

Another issue also relates to the shape of the curve. For instance, considering the issue of economic inequality and health, one can imagine two curves: both normally distributed but with very different standard deviations (**Figure 4b**, curves 1 and 2). If there is something harmful about inequality itself, its relative nature, not just the absolute prevalence of poverty, then shifting the curve to the left or giving everyone the same amount of money will do little to improve population health. Reducing the standard deviation or the percentage of people in the tails of the distribution is critical to improving health. In this case, the mean may stay exactly the same, but the tails move toward the center. One could imagine population strategies that might produce a tighter risk distribution around the mean or one could employ specific high-risk strategies to pull in the tails, especially the tail with highest risk. In any case, it is time for a second critical look at the population paradigm developed by Rose and to subject the theories to empirical tests so that optimal strategies to improve population health will be developed.

CONCLUSIONS

In this article, we argue that epidemiologists and especially social epidemiologists will have to integrate a deeper understanding of (a) the etiologic period, (b) the social and environmental context in which interventions are rolled out, and (c) the potential for heterogeneous treatment effects to develop successful interventions and policies. In addition, population approaches to disease prevention, which draw heavily on Rose's strategy of preventive medicine, where entire distributions of risk move toward lower risk levels, may be

applicable much but not all of the time. A more critical look into population approaches that evaluate high-risk strategies that may reduce inequalities more effectively is necessary. Population health depends on both improving the average health expectancy of the population as well as reducing the risk inequalities within the population.

DISCLOSURE STATEMENT

The author is not aware of any biases that might be perceived as affecting the objectivity of this review.

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