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Persistent Social Patterning of Cardiovascular Risk Rethinking the Familiar

Ana V. Diez Roux, MD, PhD

There is abundant evidence that cardiovascular disease is strongly patterned by socioeconomic position.¹ Contrary to the stereotypical image of the wealthy but stressed executive who dies of a heart attack, people in the lowest socioeconomic strata, whether defined by income, education, or occupation, are consistently at greater risk of cardiovascular disease, at least in industrialized countries. Interestingly, the increased risk is not limited to the very poor but appears to decrease in quite a remarkable graded fashion, as socioeconomic resources increase.^{2,3} This graded relationship, which is observed for virtually all indicators of socioeconomic position, is striking given the crudeness with which social factors are usually measured in epidemiological and clinical studies, and the fact that these factors are necessarily very distal to the biological processes that lead to the development of atherosclerosis and the precipitation of clinical events. The strength and persistence of these patterns suggest a pervasive influence of social context on the body and on the cardiovascular system in particular. However, like many things that we are used to seeing over and over again, with time they become invisible and their significance is forgotten.

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In this issue of *Circulation*, Tonne et al⁴ report a graded, inverse association between neighborhood deprivation and long-term survival after an acute myocardial infarction (AMI). Although there have been numerous reports of the socioeconomic patterning of cardiovascular mortality, few have investigated whether these mortality differences result from differences in the incidence of disease or from differences in survival of individuals after a clinical event. A great strength of the article by Tonne et al is the large sample size of relatively well-characterized events with complete follow-up information. A limitation discussed by the authors is exclusion of sudden deaths and in-hospital deaths. The most likely scenario is that the inclusion of these cases would have resulted in similar or stronger associations than the ones they report. Differences in survival across socioeconomic indicators are large: The authors report that patients living in the top 20% of a neighborhood

deprivation summary score have a 55% greater risk of death over the follow-up than those in the lowest 20%.

An obvious question is whether the large socioeconomic differences in survival reported by Tonne et al could result from differences in the distribution of severity of disease and other established prognostic risk factors. This type of question is raised frequently in analyses reporting socioeconomic differences in health outcomes, the implication often being that if these differences are the result of known risk factors, then they are somehow less important or less interesting. Tonne et al report that socioeconomic differences in survival are slightly reduced but remain substantial after controlling for a set of prognostic and cardiovascular risk factors measured at baseline. This result is not surprising, as it is the norm in studies that examine socioeconomic differences before and after regression adjustment for risk factors. Is it possible that measurement error resulted in residual confounding? Is it possible that other omitted risk factors or changes in risk factors over time explain these differences? Of course it is, and Tonne and collaborators go to great length, as we all do, to acknowledge these possibilities.

Answering the question of whether a set of known risk factors explains socioeconomic differences (of differences across any groups) is fraught with complexity. One problem is the almost inevitable presence of measurement error in the risk factors investigated. A less-frequently recognized methodological problem relates to the validity of estimates of residual associations after controlling for factors that may be in the causal pathway leading from the exposure to the outcome.⁵ Another important source of complexity is that many of these factors are likely to interact in causing the outcome, and these interactions usually are ignored in these types of analyses. For these reasons, simple interpretations of the comparison of adjusted and unadjusted estimates, such as "x% of the difference is explained by standard risk factors," are probably grossly inaccurate. In fact, if factors interact, as they are likely to do given the systems they form part of, then this type of partitioning becomes rather uninformative.

This does not mean that the study of what factors explain or mediate socioeconomic differences is uninteresting. To the contrary, it could shed light on disease etiology by helping to identify unknown causes and may suggest effective interventions to prevent disease. Understanding whether known factors account for differences reported in epidemiological studies will probably require approaches that are different from the ones most of us use today. A key methodological challenge is the need for methods that allow us to examine complex interrelations between factors over time. For these reasons, the simple age- and sex-adjusted estimates reported by Tonne et al are probably more meaningful than the fully adjusted estimates. Perhaps more fundamentally, the presence of differences in risk factors across levels of neighborhood deprivation (shown in

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Table 3 of the Tonne et al article) begs the question of why these differences arose in the first place. Thus, the question shifts from “Is there any difference after we control for risk factors?” to “Are the risk factors we know about also patterned by socioeconomic position?” and “What processes explain this socioeconomic patterning?” In the case of the data reported by Tonne et al, why is it that AMI cases from disadvantaged neighborhoods are more likely to present with congestive heart failure and have a history of diabetes and previous AMI? What other risk factors for post-AMI mortality are associated with socioeconomic position and why are they patterned in this way?

The traditional indicator of socioeconomic position used in epidemiological studies has been education, and to many clinical researchers this remains the only socioeconomic indicator they even consider. The correlates of education may be different in different populations, as clearly demonstrated by striking differences in income and wealth between US whites and blacks of similar educational levels.⁶ Other person-level indicators such as income and occupation provide distinct and complementary information to education. Many different dimensions of social conditions may affect cardiovascular health. In recent years, features of the broader environmental contexts where people live and work, such as neighborhoods, have received increased attention.⁷ It has been hypothesized that physical and social characteristics of residential areas may be related to cardiovascular risk through multiple mechanisms. If relevant features of residential contexts are identified they could provide useful avenues for public health intervention.

Tonne et al use a neighborhood-level measure of socioeconomic position in their analyses, and show that neighborhood deprivation is associated with poorer survival after an MI. As the authors note, the absence of person-level information on socioeconomic indicators in their study makes it impossible to determine whether these differences across neighborhoods result from the features of the neighborhoods or to the socioeconomic characteristics of the people who live in them. Isolating the effects of neighborhood environments on survival after AMI will require data different from that available to Tonne et al. A key challenge in understanding neighborhood effects on cardiovascular risk is the development and testing of hypotheses about the specific features of neighborhood that may be relevant. This will require moving beyond aggregate socioeconomic characteristics of neighborhoods to the measurement of specific health-enhancing or health-damaging features. Tonne et al speculate on what some of the possible mechanisms linking neighborhoods to post-AMI mortality might be, but they do not have measures of these constructs. The measurement of neighborhood attributes is complex, and epidemiologists have only recently begun to develop and test measurement strategies. Despite the many methodological challenges inherent in estimating neighborhood effects, the growth of interest in this area has revitalized the notion that cardiovascular disease results from the complex interaction between the characteristics of individuals (their genes, their behaviors, their biological attributes) and the physical and social environments in which they live. Hence, strategies to prevent cardiovascular disease may need to focus not only on people but also on the environment.

Recent enthusiasm regarding the possibility of identifying the genes that play a role in cardiovascular disease further highlights the need to measure the socioenvironmental factors with which genes will undoubtedly interact. In the presence of gene-environment interaction, adequate socioenvironmental assessment may turn out to be a sine qua non for the identification of genetic effects.⁸ Thus, more sophisticated socioenvironmental measurement is likely to be a requisite for complete scientific understanding of the biological processes leading to cardiovascular disease. Perhaps more important, even if the genetic determinants can be fully elucidated, intervening at the level of the environment may turn out to be the most effective prevention strategy among those genetically predisposed to the disease.

It is sometimes argued that only factors that are modifiable should be thought of as “causes.”⁹ Regardless of whether one agrees with this definition of a cause, modifiability is a requisite for interventions on that factor to be feasible. Surely then, social conditions, which are after all of our own making, are as valid as modifiable causes of cardiovascular disease as are genetic and biological attributes. It is true that intervening on more proximal factors through medical treatments or procedures may sometimes ameliorate the deleterious consequences of adverse social circumstances. Evidence suggests, however, that striking social gradients persist even in the presence of universal health care.¹⁰ This may be because access to care and the quality of that care are not necessarily equal even in the presence of (apparent) universal access. It is also because social conditions affect health through multiple mechanisms, and blocking just one of the pathways may have only a small impact on the gradient. Tonne et al demonstrate that poverty is associated with poorer survival after an AMI. To many this will not be news, but it is in understanding and changing the familiar that the greatest challenge often lies.

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