

# Social inequality, ethnicity and cardiovascular disease

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**Background** Epidemiological research on cardiovascular risk factors has led to important advances in prevention science by providing insights that have now resulted in substantial reductions in mortality. This research used the variation in risk among individuals as the guide to causal exposures. Large differentials remain among socio-demographic groups, however, and the causes of these differentials may be distinctly different from those observed at the individual level.

**Methods** Vital statistics and census data from the US and selected regions were used in an ecologic analysis.

**Results** In 1996 heart disease mortality in the US varied from 156/100 000 among African-American women to 51/100 000 among Asian women; similar differentials were observed for men. Income equality was correlated with heart disease mortality among the 47 largest US cities ( $r = -0.4$ ;  $P = 0.006$ ). Independent of income equality, racial segregation was also associated with risk of death from cardiovascular disease in this sample of cities.

**Conclusions** Social processes generate marked differentials in heart disease mortality among demographic groups. In the US, death rates are currently 2–3 times higher among African Americans compared to Asians. Broadly speaking, this variation results from their separate cultural legacies, based on well-recognized lifestyle factors and dietary patterns. Ecological comparisons across cities that share similar lifestyle patterns suggest that income inequality and patterns of racial discrimination are each associated with large variation in mortality in a similar manner. Racism and social inequality can be conceptualized as social causes of excess cardiovascular mortality that may not be measurable at the individual level.

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Socioeconomic influences on cardiovascular disease (CVD) are well recognized and have now been demonstrated in a wide range of settings.<sup>1–3</sup> Measures of social status, typically income and formal education, rank order the risk that individuals are likely to experience in a monotonic fashion reasonably well, however the slope of this relationship varies with the social context. In industrialized societies this relationship is currently inverse, while among developing societies higher social status is most often associated with higher risk.<sup>1,4,5</sup>

The current focus on social status as a risk factor of the same character as blood pressure, cholesterol or smoking is somewhat unsatisfying, however. First, despite the wealth of data that has been accumulated on this question, the set of inferences that follow from the gradient in socioeconomic (SES) risk is surprisingly limited. Social status describes the impact of the hierarchical arrangements on a broad set of behavioural and biological

traits, which themselves are the more proximate mediators of risk. The observation that yet another set of health outcomes can be rank ordered by social position adds nothing new to our understanding of public health. The causal pathway itself requires more detailed explication before the social status measures can have explanatory power. Second, social status is not an attribute of individuals, but a relationship among people. Attempts to understand these social processes through an exclusive focus on the individual as the unit of analysis will always be of limited value. As many investigators have noted, comparisons of the health impact of absolute levels of social status—for example, wealth—often cannot be made from one country to the next. Social status is therefore historically contingent. Some investigators argue that SES indicates the impact of one's position in the social hierarchy, relative to others, not simply the effect of position taken as an abstract measure.<sup>6,7</sup> This framework has been challenged, however, with arguments that give primary emphasis to material conditions among those at the bottom of stratified societies.<sup>8,9</sup> These controversies have served to focus greater attention on SES as a proxy for disease-promoting social

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forces. The common theme that emerges is the view that the SES-health gradient is evidence of the toxic side-effect, if you will, of the economic and ideological forces that determine how society is segmented and organized into its demographic units, whether these units are aggregated as social classes, races or ethnic groups.

The key conceptual issue that is at stake in the contest between the analyses that rely on the traditional epidemiological method and the analysis of social forces is whether or not the exposure is conceptualized as an individual trait or a social process. Even if the argument is accepted that group-level processes, and their manifestation as disease-promoting forces, are relevant to an understanding of cardiovascular risk, an obvious impediment limits its practical value for research—in CVD at least both the exposures and the outcomes are almost always more interesting and better characterized at the individual compared to the group level. In recent years, an attempt has been made to address this shortcoming by finding measures that make it possible to model group-level processes more effectively.<sup>6–11</sup> Among the most promising candidate measures is social inequality.<sup>9–11</sup>

In addition to relative social status, the public health literature has devoted enormous attention to the role of ethnicity and race.<sup>12</sup> Race is a multifaceted concept, mixing, in unknown quantities, genetic and biological attributes, social status and cultural practices. All of these quantities are hard to measure, particularly when the individual is the unit of analysis.<sup>12–15</sup> The broad scope of each of these constituent elements, the lack of precision associated with any measurement strategy, and the heterogeneity of assumptions that underlie the questions being asked, have all conspired to make this a field of research distinguished more by contentious debate than rapid progress. Some advantage might exist, therefore, in moving beyond individual measures. Unfortunately, procedures to assess racism as a broad social process have not received adequate attention.

Given the vexed nature of epidemiological research on social status and race, it is hardly surprising that attempts to examine their interactions are subject to even more limitations. At the same time, it is equally true that common threads connect social status and race as analytical categories, and reinforce the importance of social processes. Very few societies have a more disparate public health record by social status and race for cardiovascular diseases than does the US. In this presentation, therefore, a variety of examples taken from the US experience are used to explore how the social hierarchy in general, and inequality and race/ethnicity in particular, influence the distribution of risk for cardiovascular diseases.

## Methods

Several data sources were used to model the group variation at several levels. National vital statistics data from the National Center for Health Statistics (NHCS) were accessed to obtain mortality rates for the major demographic groups. The largest US cities ( $N = 47$ ) were characterized from a data-sharing exercise initiated by the Chicago Department of Health (CDOH). (These data were made available by S Whitman, PhD, CDOH.) Finally, a data set involving 280 US metropolitan areas was used to model residential segregation; these data were provided by the NCHS as derived from the 1990 census. Black:white

residential segregation was measured with the Gini coefficient. Bias is known to exist in the coding of death certificates for various racial/ethnic groups, with rubrics for coronary heart disease (CHD) used less often, and those for non-specific forms of heart disease used more frequently, among African Americans.<sup>3</sup> To reduce this bias, ethnic/racial comparisons were based on 'diseases of heart', International Classification of Diseases, Ninth Revision (ICD-9) codes 390–398, 402, 404–429. In some instances data were also aggregated as 'Cardiovascular diseases', codes 390–459. Age-adjustment was made to the 1940 standard. The entire age range was included.

Racial/ethnic categories correspond to the definitions used in US federal sources. Ecologic analyses were conducted using the metropolitan statistical areas as defined by the US Bureau and Census.

## Results

At the outset, given the interest in characterizing the experience of large sociodemographic groups, the overall mortality pattern is summarized. The rates of heart disease among the racial and ethnic groups in the US vary widely, with blacks having rates two and a half to three fold greater than Asians, depending on gender (Table 1).<sup>16</sup> It has been recognized for many years, of course, that hypertension is the primary pathway leading to higher CVD risk among blacks. It is immediately apparent from these data, however, that the anticipated inverse gradient between SES and CVD is not reproduced at the group level—Hispanics have similar levels of income to blacks and Asians are not better off economically than whites. Other aspects of the historical experience of these groups exert crucial influences, not the least of which are the duration of residence in the US and their respective food cultures. In the context of this discussion, these patterns serve to further demonstrate that measures of social status are historically contingent.

Attempts to explore group-level causal processes based on racial categories fall prey to a major conceptual dilemma, namely the potential confounding of biological (i.e. genetic) and social factors. Although plausible arguments can be made against a biological determinist view of racial patterns of common disease,<sup>17</sup> there is no body of evidence that allows this debate to be resolved on empirical grounds. However, social equality—in economic terms—can be modelled directly at the population level using a variety of methods. The per cent of income received by the less affluent half of the population functions as an intuitive and robust measure. Within the 47 largest US cities income equality has a moderately strong negative correlation with heart disease (Table 2;  $r = -0.41$ ,  $P = 0.006$ ). This relationship is diminished in strength when the analyses are restricted to specific race-gender groups, however, findings among sub-groups are

**Table 1** Heart disease mortality, United States, by gender and race/ethnic group, 1996. (Age-adjusted, per 100 0000)

Ethnic group	Men	Women
Black	243	156
White	175	95
Hispanic	118	65
Asian	98	51

Source: NCHS.

**Table 2** Correlation between income equality<sup>a</sup> and heart disease mortality, 1990, US big cities

	Correlation (N = 47)	P
Total population	-0.41	0.006
White men	-0.28	0.063
White women	-0.20	0.196
Black men	-0.55	0.002
Black women	-0.27	0.093

<sup>a</sup> Per cent of income to bottom half of the distribution.

difficult to interpret when social processes are the exposure of interest.

One of the most powerful social processes at work in large cities in the US over the last several decades has been the creation of enormous geographical differentials, in the form of marginalized and impoverished neighbourhoods. This process is most advanced in the largest cities. Isolating the US cities with the most income equality and the least income equality reinforces the view that size has become a factor in the emerging pattern of disparities in heart disease (Table 3). This analysis also helps allay concern that the association between SES and heart disease is not being confounded by geographical regions (i.e. the traditionally backward South versus the more developed West and North.). A modest association exists with population size—cities having a population over one million experience an average heart disease death rate of 185/100 000, compared to 166/100 000 among cities of population less than one million.

Despite the enormous descriptive literature that exists on US racial differentials the analytical approaches have been very limited. In general, investigators attempt to use a limited set of risk factors as proxies for group-level risk.<sup>14</sup> The results derived from this method of analysis are highly susceptible to residual confounding, however.<sup>13-16</sup> While average levels of classic risk factors, most notably hypertension, clearly account for a proportion of black-white differences in cardiovascular risk, many other factors that can influence health are distributed differentially between those two groups. In effect, racial/ethnic

comparisons are subject to one of the classic epidemiological errors, the ecologic fallacy. Modelling social processes at the group level, while leaving open the intervening mechanisms, escapes the risk of the ecologic fallacy. One of the most powerful mechanisms through which racism is enforced in the US has been residential segregation. Using that indicator measure, ecologic analyses of metropolitan areas in the US demonstrate that both racism and economic inequality are consistently associated with variation in risk of CVD (Figure 1).

## Discussion

The primary intent of this presentation has been to highlight the extent to which abstract social processes condition cardiovascular risk. While social factors have been widely studied in cardiovascular epidemiology, the focus has been almost exclusively on the attributes of individuals and how social factors might engender the physical effects that are being measured. In an analysis restricted only to groups, the inference follows that social inequality and racism are *bona fide* causes in their own right, and they are even worthy of being named in prevention strategies.<sup>18</sup> Just as identification of cigarette smoking as a cause reduces the need to obtain exhaustive information about mechanism, knowing that social processes are potentially modifiable components of a pathway to disease should warrant their designation as a cause and an opportunity for intervention.<sup>19</sup>

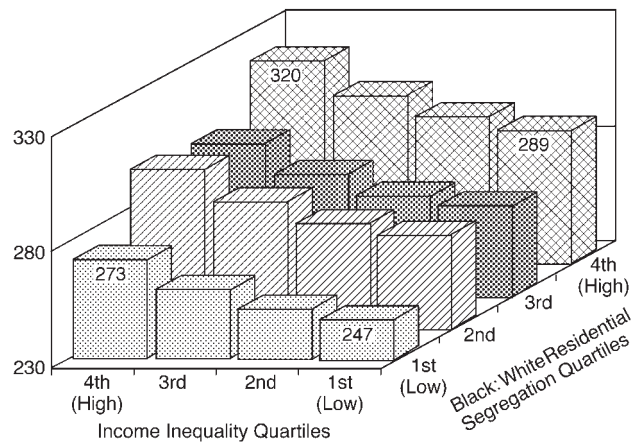
The observation that group membership based on social status or race/ethnicity influences cardiovascular risk is hardly new, and evidence exists that these differentials may be increasing.<sup>2,3,20</sup> The purpose of this analysis, however, was not to reiterate that point but to demonstrate how the underlying exposure leading to group differences can be conceptualized as a social process. Because most epidemiological research requires individual-level outcomes as the basis for causal inferences, the focus on group-level effects has been relatively neglected. While it is true that the individual is the most fundamental unit at which health can be measured, the life circumstances of that individual are determined at higher levels of social organization. Social processes involve relationships among people, not isolated acts of individuals; causality, in this approach, is located

**Table 3** Heart disease and income equality, 1990, US big cities

City	Income equality <sup>a</sup>	Heart disease death rate <sup>b</sup>
<b>Most equality:</b>		
Virginia Beach	23	140
San Jose	23	125
Columbus	21	169
Milwaukee	20	165
Indianapolis	19	157
<b>All cities: Mean, SD</b>	<b>15.3 ± 1.7</b>	<b>185 ± 36</b>
<b>Least equality:</b>		
New York	13	188
Los Angeles	13	209
Miami	13	171
New Orleans	12	189
Atlanta	10	211

<sup>a</sup> Per cent of income to bottom half of the distribution.

<sup>b</sup> (/100 000).



**Figure 1** Income inequality, black:white residential segregation and cardiovascular disease mortality, 280 US metropolitan areas, 1990

in historically contingent economic and cultural patterns, like wage inequality and racism, not a gene, a trait or a choice that can be assigned to an individual.

One of the most contentious areas of debate over inequality in the US involves the inter-relationship between race and class. Some observers view racial inequality as a manifestation of socioeconomic disadvantage, while others draw attention to the unique origins of anti-black racism in American history and its persistent power to marginalize and stigmatize people of African heritage.<sup>21</sup> An alternative argument could be made, however, that social inequality in the US is an emergent phenomenon, reflecting the combined influence of race and social class. A metaphor can suggest the nature of this argument. The Rio Negro and the Solimoes join at Manaus in northwest Brazil to form the Amazon proper, and they continue side by side for many miles before the brown and the clear waters begin to mix. Before and after their separate identity is lost, however, they flow in a single riverbed, moving as a single force. In this view, racism and class differentiation are mutually reinforcing social processes, and together they organize the process of economic exploitation. This mutually dependent relationship between the ideology of racism and the hierarchical social structure is nothing more than the particular form taken by the exploitative relationships governing wage labour in the US. The confused discourse that surrounds the debate over race and class perhaps reflects its proximity more than its uniqueness. Few would argue, for example, that it is possible to understand Spain and their conquests without Catholicism, or Britain and the empire without the belief in innate European superiority. The social identity of all members of US society, in competing ways, is defined by race, and race in turn provides much of the meaning inherent in social status. While drawn from different social dimensions, they cannot be separated one from the other, without destroying their meaning, anymore than gender can be understood separately from the concrete arrangements of power and hierarchy that prevail in a particular society. In public health, therefore, statistical procedures designed to apportion the effects of inequality to race or class are inadequate when used in isolation. Mathematical evidence of separate or independent effects is not what is at stake, since the causal argument is derived from social theory and therefore must be based on a wholly different set of assumptions and constraints. While it must be acknowledged that this view rests on a number of judgments, it serves to raise the question as to whether or not current analytical approaches take account adequately of the historical context of inequality in the US. Social epidemiology must be both critical of society, since it hopes to identify the sources of ill health, and guided by a theory of social organization, since it is the social forces that create the conditions of life for the population being studied. These requirements cannot be met by applying methods derived from the analysis of risk factors at the individual level.

Conventional epidemiology may eschew social processes as an explanation in part because their impact on individuals will be impossible to assess on a scale commensurate with physical attributes. In fact, this analytical strategy is not an attempt to deepen our understanding of how SES translates into individual risk. Just as the scope of any biological problem can be defined at different levels of complexity—the molecule, the cell, the organism, the population—the nature of the explanations given

at these levels must take different forms. While all these explanations must be based on the analysis of quantifiable, testable hypotheses, social process explanations will be devoid of meaning for individuals considered in isolation. Social processes are no less appropriate as an explanation of differential health status among populations than is evolution as an explanation of how populations of organisms are differentiated. Both are inferences made from observational data about the relationships among groups and attempt to model causal processes by examining their effects of populations of organisms. In both instances while it is difficult, if not impossible, to measure the impact on individuals, the impacts are nonetheless profound.

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