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Socioeconomic Status and Coronary Heart Disease Risk Prediction

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Achieving the Healthy People 2010 goal of reducing health disparities has been challenging. Disparity in life expectancy by socioeconomic status (SES) has been increasing.[1] Much of this disparity is attributable to higher cardiovascular, particularly coronary heart disease (CHD), mortality among persons of lower SES. Disparities in CHD mortality result from multiple factors, including early life environment and material disadvantage,[2] social and behavioral risk factors,[3] access to care,[4] and systematic underestimation of risk among persons with lower SES in clinical care. This Commentary addresses the importance of SES in CHD risk assessment.

Current risk-based intervention strategies ignore the independent contribution of SES to CHD and thus may contribute to increasing SES disparities. If CHD risk is better estimated among the poor, it could lead to increased statin use for cholesterol reduction and aspirin use for CHD prevention among this population, which may reduce CHD disparities.

Independent CHD Risk Factor

Along with age and sex, SES is a powerful determinant of health and is inversely associated with risk for CHD.[5] This risk is independent of age, sex, smoking, hypertension, diabetes, physical activity, diet, cholesterol, and body weight.[5] The risk associated with low educational attainment is roughly comparable to many traditional CHD risk factors such as high cholesterol levels.[5] These findings suggest that CHD risk assessment using Framingham risk scores (FRS) underestimates CHD risk for persons with low SES and could exacerbate disparities in preventive treatment.

Pathways

As with many CHD risk factors, the underlying pathways between SES and CHD are complex but they likely include biomedical, psychosocial, and behavioral mediators. In a study of more than 30 000 patients referred for stress testing for evaluation of suspected coronary artery disease, lower SES was independently associated with impaired functional exercise capacity and abnormal heart rate recovery, and these physiologic characteristics accounted for a major proportion of the association between lower SES and increased all-cause mortality.[16]

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Moreover, increasing evidence suggests that chronic psychosocial stress associated with low SES promotes atherosclerosis and CHD events.[6] Socioeconomic status is also likely associated with behavioral risk factors incompletely captured cross-sectionally or not measured at all. For example, persons of lower SES confront more barriers (eg, access, costs, health literacy) to modifying risk behaviors over time such as quitting smoking, improving diet, increasing physical activity, and adhering to medications.

Failure to achieve target levels for serum low-density lipoprotein cholesterol (LDL-C) through therapeutic lifestyle change in the presence of CHD risk is an indication for cholesterol-lowering medication. Given greater barriers to successful therapeutic lifestyle change, more persons with low SES may fail to achieve these targets without medication. However, bias in FRS will exclude some persons with low SES from proceeding to treatment with cholesterol-lowering medication. A similar bias exists for initiation of aspirin prophylaxis for CHD where eligibility is also based on CHD risk assessment.

CHD Risk Prediction

Risk models are assessed based on the ability of the model to appropriately classify individuals according to their risk of experiencing the event of interest during a defined follow-up period. Two key assessment components of risk models are discrimination and calibration. Discrimination refers to the ability to sort patients according to their actual risks. It reflects the relative ordering of model predictions, not actual probabilities. Calibration refers to how closely the predicted probabilities of the model match actual risk on an absolute basis. A model with good discrimination but poor calibration can typically be adjusted to improve its calibration without affecting its discrimination simply by adjusting the magnitudes of its predictions without changing their relative ordering. However, a model with poor discrimination cannot be adjusted without affecting calibration.

The clinical utility of CHD risk models has traditionally been judged primarily, and often exclusively, by discrimination. Typically, discrimination is assessed based on relatively insensitive concordance statistics, such as the area under the receiver operating curve (C statistic) used for dichotomous event indicators, or its generalization for use with survival analysis models of event time data.

Most novel CHD risk factors do not appreciably improve model discrimination because few risk factors have sufficiently high relative risk and/or population prevalence. Measures of SES are also unlikely to substantially improve model discrimination – meaning that adding SES to CHD prediction models, such as FRS, is unlikely to substantially improve CHD risk discrimination for individual patients. However, the primary goal of adding SES to CHD risk assessment is not to improve risk discrimination among individual patients, but, by improving calibration, to eliminate systematic underestimation of risk for the population of patients with low SES, thus minimizing disparity in risk-based treatment for patients. This is similar to applying the FRS to a different population, such as in the United Kingdom rather than the US population where it was developed. The FRS had to be recalibrated (in this case downward) to account for lower mean CHD risk in the UK population even though not all UK patients have lower CHD risk than US patients.

Treatment Disparity

Use of statins and aspirin for CHD prevention are based on the estimation of absolute CHD risk. High-risk, poor patients are less likely to receive statins or aspirin than more affluent patients.[4] Underestimation of risk for persons with low SES may further exacerbate underuse of these interventions among these patients. Although the effect of lower LDL-C treatment threshold for persons with low SES has not been studied by a randomized trial, this approach

is supported by the finding of little variation in the relative risk reduction associated with statin treatment across moderate to high ranges of LDL-C levels.[9] Use of aspirin prophylaxis involves weighing reduction in cardiovascular risk against risks of gastrointestinal and cerebral hemorrhage. If both risk of CHD and adverse events are associated with low SES, some of the benefits of accounting for SES might be mitigated, but reliable data are lacking.

Bias in Risk Assessment

The extent that FRS underestimates risk for poor patients in a US sample has not been examined. Brindle et al examined the accuracy of FRS among a prospective cohort from West Scotland.[10] The FRS underpredicted risk to a greater extent among those persons with low SES. Based on FRS, individuals living in communities with the lowest income had only a 3% higher estimated risk than those living in the wealthiest communities in contrast to more than a 41% higher observed risk between these groups. In another Scottish study, ASSIGN (a cardiovascular risk score similar to FRS, but includes SES and family history) showed better calibration for prediction of cardiovascular disease by SES than did FRS.[11] The model showed minimally improved model discrimination compared with FRS, but appropriately predicted 18% more events than did FRS (298 v 253), among those in the fifth lowest SES. Another cardiovascular risk score, QRISK, also more closely approximates risk among persons at low SES[12] despite having only slight improvement in discrimination relative to the FRS. These studies suggest that including social risk in CHD risk prediction may mitigate underestimation of risk for those at low SES to varying degrees, but it does not substantially improve model discrimination. Data from the United States are needed to examine this question as well as assess the potential risks associated with this strategy including effects of potential drug overtreatment.

Choice of SES Measure

ASSIGN and QRISK use area-based SES measures called deprivation indices, such as the Scottish Index of Multiple Deprivation and the Townsend Index.[11,12] Both indices are based on composite measures of impoverishment of residents within postal codes. In the United States, area-based measures of SES have been shown to be nearly comparable to individual measures in capturing morbidity risks for many individuals associated with SES.[13] Zip codes have been used widely in research as a proxy for SES when individual measures are not available. Patients' addresses can be linked to the sociodemographic characteristics such as census tracts or block groups to yield more refined estimates of SES, but this process is more challenging to implement.

Individual measures of SES, such as educational level, can be collected directly from patients. Most patients are willing to provide their educational level, and the education-CHD relationship is less subject to reverse causality than is income or occupation. Household income also provides potentially useful information particularly related to financial barriers, but it is more sensitive to gather. Use of occupation measure excludes persons who are not formally employed or retired, and is not easily converted into an ordinal scale. Type of insurance provides a proxy for family impoverishment, but SES associations differ by state, category, and gender. On balance, education may be the optimal SES measure, but area-based measures (based on geocoding addresses and linking them to census data) obviate additional data collection and may capture relevant additional community-level factors. In theory, area-based measures could be used not only to calibrate risk through estimation of SES, but also by directly capturing local CHD incidence (assuming availability of reliable data). Either individual or area-based measures could be used with an electronic health record and automatically incorporated into CHD risk assessment using a decision support tool.

Next Steps and Barriers

Assessing potential SES-related bias in CHD risk tools in US populations represents an important priority. Data are also needed for the optimal and most feasible measures of SES in various settings. Translational research is needed to determine how to best incorporate SES into the decision support for CHD risk assessment at the point of care. In the United Kingdom, SES derived from the Townsend deprivation score has been integrated into electronic medical records for more than 32 million patients, facilitating its use with risk scoring systems such as QRISK2.[14]

Adding SES to risk assessment is potentially relevant to other CHD prevention decisions, including target goals for treating hypertension. More generally, this approach could be used to address any health disparity for which SES predicts risk and treatment decisions are guided by the estimation of that risk. Research is required to quantify the potential benefits and harms of including SES in CHD risk assessment for treatment decisions.

Perhaps the greatest obstacle to translating SES into clinical practice is physician reluctanceto use SES in this context. The reasons for this are uncertain, but may include uncertainty regarding the exact causal pathways between SES and CHD, exclusive focus on individual behavioral and physiological rather than social risk factors, aversion to considering nonmodifiable risk factors, and challenges in measuring SES. Yet, similar uncertainty exists for the pathway between men and CHD. The recognition of the role of social risk is increasing, as shown by the recent World Health Organization report.[17] Other nonmodifiable demographic factors currently represent the cornerstone of CHD assessment; age and sex provide most of the discriminatory power for CHD risk prediction.[15] Currently, risk factors such as family history that are potentially less accurately reported than patient educational level are used to assess CHD risk. An additional concern may be that addressing the health consequences of low SES may appear to avoid addressing fundamental causes. Strategies addressing both causes and consequences of SES as a risk measure are needed to minimize SES-related disparities.

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