The Foreclosure Crisis and Cardiovascular Disease

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Cardiovascular disease (CVD) epidemiology is often presented as the paradigm for an approach to prevention that focuses on identifying individual-level risk factors and intervening on them through behavioral change (eg, diet and physical activity interventions) or through the early detection and treatment of biomedical risk factors (such as high blood pressure or dyslipidemias). At times, this approach has been criticized for failing to consider the more distal causes of these individual-level risk factors (many times of a social or economic nature) that drive the distributions of the proximal risks factors, shape population-level differences in cardiovascular risk, and present large potential opportunities for CVD prevention.

In parallel to the risk factor approach, a long tradition in CVD epidemiology has documented the social patterning of CVD and the impact of a number of distal factors, including social class, work environments, and neighborhood environments, on cardiovascular events and risk factors. The article by Arcaya et al in this issue of Circulation on the links between foreclosures and blood pressure falls squarely within the latter tradition that aims to place CVD within social and economic contexts.

Spurred perhaps in part by recent economic circumstances in the United States and worldwide, a growing body of work has attempted to document the health consequences of economic recessions or exposures to specific conditions (such as job loss, financial stress, or experiences of losing a home) that may be more common during recessions. As in other areas of health research, clearly specifying the causal question of interest is key. Precisely specifying the causal question is particularly important in investigating the health effects of distal economic processes because the total population health impact of economic recessions (which may involve unfavorable effects for those experiencing unemployment counterbalanced or even exceeded by favorable effects associated with shorter work hours, lower levels of unhealthy consumption patterns, and lower levels of air pollution for the population as a whole) may be very different from the individual-level health consequences of job loss, home loss, or financial strain.2–7 In the former case, the question refers to the health impacts of accelerations or decelerations of the economy (as indexed by economic indicators such as unemployment), whereas in the latter case, the causal question pertains to the causal effects of directly experiencing unemployment, job loss, or financial strain. Ambiguity in specifying the causal question is at the root of many debates on the causal impact of economic recessions on health.

The causal question investigated by Arcaya et al in this issue of Circulation is narrowly defined as the individual-level effects on systolic blood pressure of living near vacant foreclosed properties over the past year. As described by the authors, a number of processes, including effects of foreclosures on various aspects of neighborhood environments (declines in nearby property values, degradation of the neighborhood environment, residential turnover, changes to safety levels, and changes in retail and built environments with potential consequences for behaviors linked to blood pressure), could explain the effects of living near recently foreclosed properties on health. However, the authors have gone to great lengths to adjust for various other metrics of neighborhood-level foreclosure levels (including foreclosures within 1 km of the residences, as well as counts of foreclosures within 100–200 m, 200–300 m, 300–400 m, and 400–500 m) to control for “neighborhood distress” linked to foreclosures. This suggests that their causal question is actually about the effects of proximity to foreclosed properties on blood pressure that are independent of the possible effects of foreclosures on neighborhood conditions.

Indeed, Arcaya et al report that statistically significant associations of blood pressure with foreclosures within 100 m were present only after controlling for these additional neighborhood variables. This is rather surprising if one believes that foreclosures are spatially correlated and that neighborhood foreclosure levels may impact blood pressure through the neighborhood mechanisms described by the authors. The authors interpret these findings as supporting “a relative deprivation understanding of how socioeconomic exposures affect health.” The analytical approach used suggests that the authors’ implicit causal question is actually about whether close proximity to foreclosed properties operates through a relative comparison (and stress mediated) mechanism, because they have estimated the effects of nearby foreclosures after conditioning on foreclosures in the broader neighborhood. This in itself is an interesting question, although if indeed the interest is in the stress-mediated effects of foreclosure, it is not clear why the household’s individual experience of foreclosure was not considered in the analyses (as another source of foreclosure-related stress or as a confounder of the stress-mediated effects of nearby foreclosures).

A key strength of the work is the creative use of longitudinal data from the Framingham Offspring study linked to data on foreclosures from Massachusetts deed data. As in other

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Editorial
observational studies, a key challenge in drawing causal inferences is the possibility of confounding by poorly measured or unmeasured factors. Because of the longitudinal nature of the data, both time-invariant and time-varying confounders could play a role. The authors adjusted for race, income, and education by including these variables in the regression models, but residual confounding remains a possibility because individuals residing near foreclosed properties may be different from those who do not live near foreclosed properties in ways that could be relevant to blood pressure. Given that repeat measures for each person appear to be available, the use of fixed effects for each person, which would allow estimates of associations to be based solely on within-person variation (in contrast to both between- and within-person variation as in the modeling approach used), could have potentially strengthened inferences by tightly controlling for measured and unmeasured time-invariant individual-level confounders.

A second challenge faced by the authors was the very low prevalence of exposure (only 2.3% of measurement occasions showed foreclosures within 100 m). This very low prevalence of exposure also limits the conclusions that can be drawn from the thoughtful sensitivity analyses presented in the report, including contrasting different types of foreclosures, as well as attempting to determine whether foreclosures are simply a marker for other neighborhood or street conditions by contrasting associations of systolic blood pressure with foreclosures that occurred before and after the blood pressure measurement. The very low prevalence of the exposure would also have made the more informative individual-level fixed-effects models (which derive estimates solely based on within-person variability in foreclosure experience and blood pressure) highly inefficient. It may also explain why it was not possible to detect associations of foreclosures with dichotomous hypertension.

The authors attempted to investigate the possible mediating roles of alcohol use and weight gain on the associations they observed, but as they note, their ability to draw firm conclusions was limited by the data they had and the analytical approach they used. Another potential mediating factor not explored in the article is the use of and compliance with blood pressure medications, given that the experience of stress could affect treatment. Medication use was simply adjusted for in the analysis, a common but not entirely satisfactory approach because medication use is itself a consequence of having high blood pressure. Despite these limitations, the association of nearby foreclosures with blood pressure reported by Arcaya et al is intriguing and worthy of further exploration.

To many audiences, including clinicians and the general public, the finding that experiences of economic conditions such as foreclosure may be related to higher levels of systolic blood pressure raises the question, “So what?” Surely, we do not need to show an effect of foreclosures on blood pressure (or any aspect of health) to argue that reducing foreclosures in an important policy goal. It also seems naïve (and verging on patronizing) to suggest that we need to counsel persons experiencing foreclosures (or in this case, experiencing foreclosures near their homes) not to feel stressed, drink, or put on weight. Moreover, why should clinicians or epidemiologists care about what may at first glance appear to be a rather esoteric link?

In my view, there are 2 important reasons. One is that studying the impact of exposures such as experiences of foreclosure on blood pressure may enhance etiologic understanding by helping us assess the elusive effects of environmental factors (including stressors) on CVD (to the extent, of course, that foreclosure can serve as an instrument for stress or other environmental exposures). The second reason is that studies like those by Arcaya et al shed light on the fact that a broad range of distal factors, including many affected by policies that have no intended health benefits (including various aspects of social, economic, and housing policies), may have pervasive health effects (including CVD effects) that operate through a range of interrelated and possibly small effects that ultimately drive major patterns and trends in the health of populations. Understanding these drivers and acting on them may be as important or more important to CVD prevention than traditional strategies linked to behavioral change or risk factor detection and control.

Disclosures

None.

References

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