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EDITORIAL COMMENT

Psychological Distress and Susceptibility to Cardiovascular Disease Across the Lifespan

Implications for Future Research and Clinical Practice*

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Stress and psychological distress are known powerful, modifiable risk factors for cardiovascular disease (CVD) (1–5), yet we know little about when or how the processes underlying this relationship begin. Indeed, prospective lifespan research that connects childhood experience with subsequent CVD-related outcomes to inform development of appropriate early interventions has been lacking. Using data from the 1958 British Birth Cohort Study, Winning et al. (6), in this issue of the *Journal*, thoughtfully address this gap by providing evidence suggesting that early distress, as reported in childhood, contributes independently to cardiometabolic risk (CMR) decades later in middle adulthood, even when individuals report experiencing little distress as adults. These findings are on the basis of data collected over a 45-year period from 6,714 individuals who were part of a 1-week birth cohort

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from Great Britain in 1958. Although differential attrition analysis revealed that males with lower socioeconomic status and cognitive ability scores and higher distress at age 7 were more likely to drop out of the study over time, it is important to note that this pattern of attrition suggests that the findings are conservative estimates of the distress-CMR association. When considered in the broader research literature addressing psychological impacts on the development of CVD, this paper is highly relevant with clear implications for research and clinical practice.

Several key findings reported warrant further research attention. Most importantly, their findings send a clear and important message that childhood distress, even if it remits in adulthood, may signal early CVD risk. As childhood adversity is a major contributor to early distress, these findings are consistent with the growing body of research documenting that early adversity increases the risk of CVD-related morbidity/mortality (7,8). To clarify the unique role of distress in subsequent CMR, however, future studies should include assessments of early and ongoing adversity to distinguish the effects of individual trauma and cumulative adversity over time (9) from those of distress. In other words, it would help to understand how adversity and adversity-related distress each independently contribute to subsequent CMR.

The persistent impact of childhood distress on mid-life CMR also suggests the possibility that there are sensitive periods in childhood during which some seemingly irreversible physiological, emotional, or behavioral processes are established that affect subsequent CMR. That is, perhaps there are critical windows of risk linking childhood distress and CMR that point to windows of opportunity for intervention. An important next step would be to examine the association between distress and CMR at different ages in childhood to see whether there is a specific period when the distress-CMR association is especially strong. This could help to identify optimal time points for targeting interventions. The findings further suggest that there may be sensitive periods associated with different types of CMR. For example, it appears that distress that occurs only in childhood is most strongly associated with increased heart rate

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and HbA_{1c}, whereas distress occurring only in adulthood is most strongly associated with increased triglycerides and decreased HDL. These life phase-specific findings suggest that different developmental processes and/or stress-related responses in the brain or autonomic nervous system are shaping CMR across the lifespan (10) and indicate the need for specific interventions that target different CMR markers across the lifespan. More fine-grained analyses of these data could potentially shed light on important critical windows of opportunity for interventions, whether they be pharmacological, behavioral, or familial, to minimize the impact of childhood distress on CMR. Such analyses could also shed light on potential windows of time during which support for a child's emotional development may prove especially beneficial for overall health and well-being.

The current study also begins to shed light on the interplay between psychological and physiological processes that contribute to CVD across the lifespan. To build on this work, future prospective studies should include concurrent physiological and psychological assessments over time to explore their interplay in relation to CMR markers and tease apart the mechanisms by which distress at different time points is associated with trajectories of these markers over time. Similarly, future work should examine how specific types of distress-related symptoms—anxiety, depression, panic, anger—may each uniquely affect different CMR marker trajectories over the lifespan. Having concurrent assessments of physiological and psychological response over several years would allow better examination of these relationships and once again provide much needed data to tailor appropriate secondary prevention interventions to high-risk children and their families.

These findings also have important implications for clinical practice. Given the prospective link between childhood distress and subsequent CVD risk, this study reminds us that we must seriously address our patients' psychological needs if we want to prevent CVD-related pathology. That is, we need to recognize that psychological distress is a critical indicator of risk that should be addressed before it becomes a clinically relevant physiological statistic. Indeed, persistent psychological distress should probably be considered a proxy for subclinical pathophysiology that warrants immediate intervention. Specific steps should be taken to facilitate identification of at-risk individuals and provide appropriate services for them in primary care settings. These include implementing routine screening for stress/trauma exposure and psychological distress (Patient Health Questionnaire-9, Primary Care Post-Traumatic Stress Disorder Screen), and building on-site collaborative care clinics to improve access to much needed mental health professionals (e.g., psychiatric mental health nurse practitioners, clinical social workers, primary care psychologists) who can help patients strengthen their coping self-efficacy and emotion regulation skills.

Finally, primary care clinicians need to carefully consider how we communicate with our patients. The many constraints of our current health care delivery system make it difficult to address psychological symptoms and encourage clinicians to focus instead on "managing" other known CVD risk factors: smoking, obesity, elevated cholesterol, lack of exercise. In so doing, however, we may miss the most important issue underlying these risk factors: the burden of adversity and distress affecting our patients. When considering our patients in this broader social context, telling them to lose weight, stop smoking, or eat a better diet without addressing the underlying stress or distress that may be fueling unhealthy behaviors (and lab values) may be counterproductive. Indeed, by "advising" or "directing" our patients to change their behaviors, we may undermine their trust in us and exacerbate their distress, especially if they feel stuck or unable to make the recommended changes. As an alternative to using the directive approach to patient care, we should engage in patient-centered motivational interviewing approaches that help us understand better what our patients are ready and able to do for themselves (11). Building trust by accepting where our patients are in their change process and supporting them to make the changes that they are ready to make will likely decrease the distress they experience in the visit and improve our relationship with them over time. Ultimately, this is perhaps the best, most immediate way we can apply these findings—by using more caring, compassionate approaches to patient communication.

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