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Can Being Born Preterm or with a Socioeconomic Disadvantage in Early Childhood Accelerate Biologic Aging?

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The association of adverse childhood experiences and subsequent increased risks of adult diseases (e.g. hypertension, cardiovascular disease, and diabetes) years later have been well-documented in various longitudinal studies.

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The association of adverse childhood experiences and subsequent increased risks of adult diseases (e.g. hypertension, cardiovascular disease, and diabetes) years later have been well-documented in various longitudinal studies. What role might accelerated aging be playing in connecting an early period of stress with worsened adult health morbidities? This week we are releasing two studies and commentaries that focus on looking at accelerated aging due to stressors in early infancy or childhood. Both studies use DNA-methylation measures or “epigenetic clocks” as biomarkers of the aging process.

The first of these studies, by Raffington et al (10.1542/peds.2020-024406), reports on 600 children and adolescents who are part of a larger Texas Twin Project, looking at whether family-level and neighborhood-level disadvantages for some children affect the pace of their aging using a new epigenetic clock. The authors found that those children living in more disadvantaged neighborhoods showed a faster pace of aging than those who did not.

A second study by Lieshout et al (10.1542/peds.2020-001230) focused on infants born at extremely low birth weight (ELBW). In this study, 45 ELBW infants (born at less than 1,000 grams) who survived and reached adulthood (30-35 years of age) were compared to 49 normal birth weight (NBW) controls also now adults. A different epigenetic clock was used to measure DNA-methylation in this study than in the Raffington et al study and, interestingly enough, males born ELBW had a significantly older epigenetic age than males born NBW. In contrast, females born ELBW were not found to have advanced epigenetic age compared than their control counterparts.

What is going on to cause this accelerated aging in association with early socioeconomic disadvantage or males born ELBW? To answer this question and to provide further insight on accelerated biologic aging and its ramifications, we invited two commentaries. One by Dr. Daniel Notterman from the Department of Molecular Biology at Princeton (10.1542/peds.2021-049939) focuses on the Raffington findings and provides a superb description of DNA-methylation and how epigenetic clocks measure the aging process. Dr. Notterman reminds us that seeing an uptick in the amount of DNA-methylation in association with accelerated aging is just that— an association or biomarker but is not necessarily causal, and that the associations found in these studies need to be confirmed in future studies comparing these clocks to other markers of accelerated aging as telomere length attrition, mitochondrial function, and structure or accumulation of specific cellular proteins. Finding such correlates would further the association of accelerated aging as perhaps playing a role in the pathophysiology of earlier-onset chronic disease.

The second commentary by epidemiologist Dr. Pam Factor-Litvak from Columbia University (10.1542/peds.2020-038158), focuses on the results of the Lieshout et al study and why males but not females born ELBW had accelerated aging. Her focus is on prevention of stressors in the neonatal period as well as why we observe differences by sex. Dr. Factor-Litwak explains that because estrogen may mitigate accelerated aging, perhaps we may still see such changes in females but not until their estrogen levels decreased such as during their menopausal transition (again suggesting a need for further research). With both studies and commentaries, preventive strategies to mitigate stressors that can accelerate aging are suggested. If you want to better understand the aging process and how it might be accelerated by adverse experiences during infancy and childhood, check out both studies and commentaries.

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