Figure 1.
Social determinants, for better or worse, of biological aging and health.

Family Disadvantage Accelerates and Support Decelerates Biological Aging and Illness

Dr. Ronald L. Simons and Dr. Leslie Gordon-Simons
University of Georgia

Introduction

- Dramatic growth in the number of older adults throughout the world has placed inequalities in healthy aging at the forefront of the public health agenda.
- Although traditional health risk behaviors have shown a small effect on accelerated biological aging, recent studies report that the social environment may exert a much stronger influence. Building on this research, the present study uses longitudinal assessments of family and neighborhood processes collected throughout childhood and adolescence from a sample of several hundred Black Americans to investigate the effect of cumulative adversity versus cumulative support on speed of aging and health during early adulthood (age 29).
- Though some studies have examined the effect of adversity on accelerated aging, there has been almost no research on the extent to which long-term support might decelerate aging. Further, most studies have focused on older samples, leaving the question of whether disparities in speed of aging (variations in the difference between chronological and biological age) are evident already in early adulthood.
- Finally, while often assumed, there has been no investigation of whether the effects of the social environment on metabolic/cardiovascular problems (BP, insulin levels, inflammation, etc.) and chronic illness are mediated (explained) by speed of biological aging.

Sample, Procedures & Analysis

Sample:

We used 4 waves (ages 10-18) of interview and neighborhood data collected from roughly 400 Black children and their families to predict speed of aging and health at age 29.

Procedures:

Individuals completed interviews at ages 10, 12, 15, 18, At age 29, they completed another set of interviews and blood was drawn to assess accelerated aging and metabolic/cardiovascular risk.

Measures

Speed of Aging. We utilized a recently developed gene expression index of aging developed by Peters et al. (2015). This transcriptome index consists of 1497 sites where the level of gene expression (amount of mRNA) is correlated with age. A high agreement (r = .972) was found between the discovery sample of 7074 individuals of European ancestry and a replication sample of 7909 that included various ethnic groups including African Americans, Native Americans, and Hispanics. This transcriptome index is highly correlated with age and the difference between an individual's predicted and chronological age indicates, in years, the extent to which they are experiencing accelerated or decelerated aging.

Cumulative adversity. Five scales were standardized and

Summary of Results

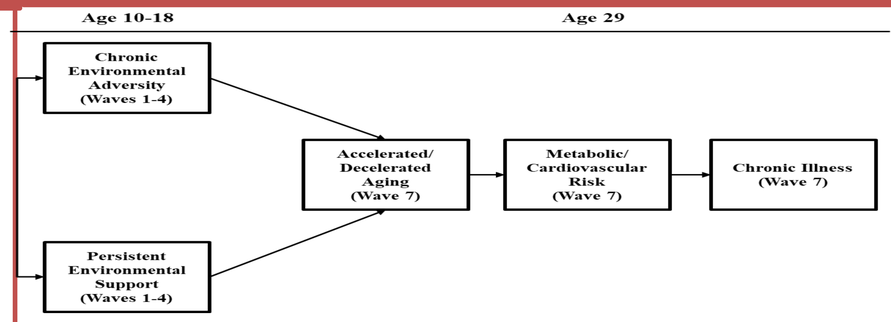

Figure 1. Theoretical Model to be Tested

- Overall, the sample was roughly half a year older biologically than their actual age. Accelerated aging was calculated by subtracting residual scores from the regression of biological age on chronological age.
- As expected, at each wave of data collection the various types of adversity were positively, and the various types of environmental support negatively, correlated with accelerated aging
- We summed across types of adversity and across types of support to form measures of chronic adversity and of persistent environmental support, respectively. The adversity measure was correlated .248 and support -.321 with accelerated aging, and each of the measures was also significantly associated with cardiometabolic risk and chronic illness.
- Figure 2 shows the results of using SEM to test major elements of the theoretical model. The fit indices indicate a good fit to the data. The model shows that both chronic adversity and persistent support continue to be related to speed of aging after controlling for the effects of the control variables. As expected adversity is associated with accelerated aging whereas support is related to decelerated aging. The graph in 3a shows that a SD increase in adversity is associated with being .739 years older than one's chronological age. In contrast, 3b indicates that a SD increase in support is associated with being .687 years younger than one's chronological age. There was no significant interaction between adversity and support. Rather, both adversity and support appear to exert independent effects on aging.
- Continuing with Figure 2, accelerated aging predicts cardiometabolic risk (β = .237) even after controlling for earlier assessments of cardiovascular risk. And, metabolic/cardiovascular risk, in turn, predicts onset of chronic illness (β = .404) even after controlling for earlier self-reported health. Importantly, the model shows no significant direct path from accelerated aging to chronic illness or from adversity and support to either metabolic/cardiovascular risk or chronic illness. Bootstrap methods indicated that the indirect effect of chronic adversity on chronic illness through both accelerated aging and metabolic/cardiovascular risk is significant and the indirect effect of persistent support on chronic illness through both accelerated aging and metabolic/cardiovascular risk is also significant.

Graphs and SEM

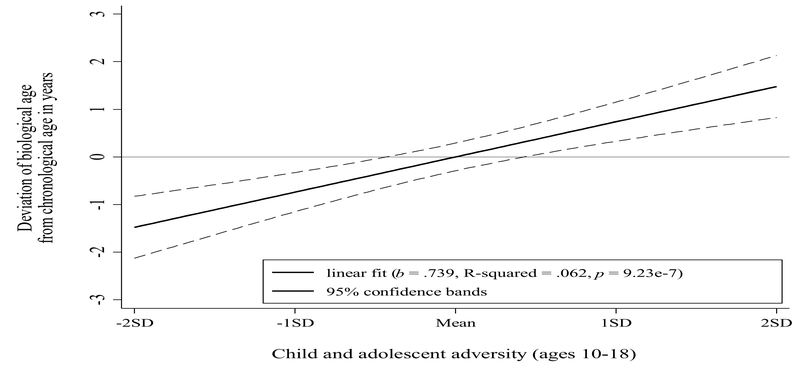

Figure 3a.
Scatter plot representing the association between child and adolescent cumulative adversity (waves 1—4) and biological aging using mRNA. The solid line displays the predicted regression line, and the dashed lines are the 95% confidence bands for the fitted line. Predicted scores represent residual biological age after controlling for chronological age.

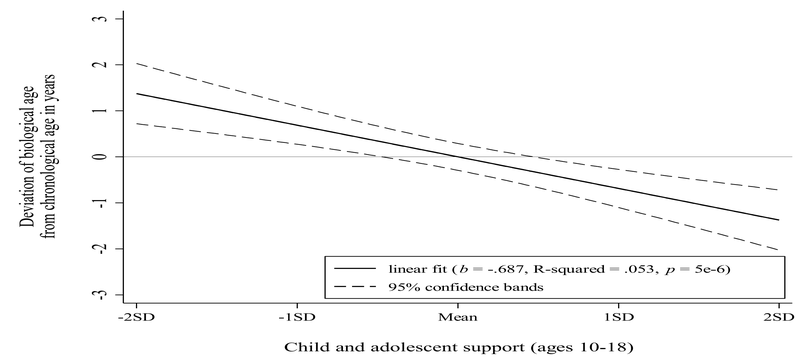

Figure 3b. Scatter plot representing the association between child and adolescent cumulative support (waves 1—4) and biological aging using mRNA. The solid line displays the predicted regression line, and the dashed lines are the 95% confidence bands for the fitted line. Predicted scores represent residual iological age after controlling for chronological age.

Discussion & Implications

- Our findings suggest that policies and programs that promote family support, decrease financial hardship, encourage school success, and decrease neighborhood disorder are likely to enhance healthy aging.
- In recent years the U.S. has largely lost its will to invest in such programs, but our results suggest that this neglect is likely to result in long-term costs due to an increased number of individuals suffering accelerated aging and early onset of chronic illness.