Researchers Uncover Aging Clues Related to Biological Shield

Researchers in the United States investigated associations between cumulative social advantage (CSA) and epigenetic aging, neuroendocrine function, and systemic inflammation.

Social relationships were determinants of functional capacity, health, and longevity. Strong supportive relationships were linked to strengthened immune function, improved cognitive outcomes, and reduced risks of morbidity and mortality. However, access to relational resources was uneven, and social advantages accumulated over time, which may have contributed to health disparities throughout life.

CSA was conceptualized as a multidimensional construct that reflected sustained access to social resources across religious, familial, community, and emotional domains. It had been linked to better functional health, lower multimorbidity, and decreased mortality risk. The authors proposed that sustained social advantage would manifest in core biological systems regulating aging, including inflammatory, neuroendocrine, and epigenetic pathways.

Study

In the present study, researchers tested associations of CSA with systemic inflammation, neuroendocrine function, and epigenetic aging. They used data from two Midlife in the United States (MIDUS) cohorts: MIDUS-II and MIDUS Refresher. The MIDUS-II and Refresher cohorts were recruited during economic stability and after the financial crisis (of 2008–09), respectively.

The researchers modeled CSA as a second-order latent construct that reflected access to social resources in four domains: Community engagement, religious and faith-based support, extended emotional support, and parent-child relationship quality. Sixteen self-reported, evidence-based indicators of CSA were selected.

Religious and faith-based support was examined using three scales: Religious practice, identification, and coping. Community engagement was measured using six scales: friendship support, positive relations with others, and social integration, contribution, actualization, and acceptance. Parent-child relationship quality was measured using four scales: Paternal warmth and generosity, and maternal warmth and generosity.

Extended emotional support was assessed based on the number of hours of emotional support received from parents, children, and others. DNA methylation was processed through seven epigenetic clocks, including Horvath, Horvath2, Hannum, PhenoAge, multiple GrimAge implementations, and DunedinPACE, which captured the pace of biological aging, to quantify molecular aging.

Systemic inflammation was evaluated using serum biomarkers: C-reactive protein (CRP), E-selectin, interleukin (IL)-6, IL-8, IL-10, tumor necrosis factor (TNF)-α, and intercellular adhesion molecule (ICAM)-1. Overnight urine samples were collected, and catecholamines (epinephrine, dopamine, and norepinephrine), cortisol, and cortisone were measured to evaluate neuroendocrine function.

A confirmatory factor analysis (CFA) was performed to validate the structure of CSA. First-order latent domains were estimated from their respective indicators. Domain-specific factors were modeled to load onto a second-order latent CSA construct. Each biological outcome was regressed on this second-order construct, adjusted for sex, age, race/ethnicity, education, household income, and cohort (MIDUS-II and Refresher).

Results

The study included 2,117 individuals, with an average age of 55. Most participants were female (55 percent) and White (75 percent). CSA indicators were suitable for factor analysis, with acceptable internal consistency across the 16 indicators; the CFA supported the proposed hierarchical structure of CSA. Age was significantly associated with virtually all measures of inflammation and epigenetic aging.

Higher education was associated with slower epigenetic aging and lower systemic inflammation, while household income showed smaller and less consistent associations. Black individuals showed elevated inflammatory activity and accelerated epigenetic aging compared with White individuals. Cohort differences were minimal, while sex differences favored females in neuroendocrine and epigenetic measures, although CRP concentrations were higher among women. Higher CSA showed consistent associations with more favorable biological profiles.

All DNA methylation clocks were negatively associated with CSA, implying slower molecular aging among those with greater social advantage. In particular, GrimAge and DunedinPACE clocks showed the most robust effects after false discovery rate correction. CSA was also negatively associated with cytokines and vascular adhesion markers, with IL-6 showing the strongest and statistically significant association after false discovery rate correction. However, CSA was not associated with any urinary marker.

Conclusion

Taken together, CSA was associated with lower systemic inflammation and slower biological aging. The findings supported the idea that sustained access to diverse social resources became embedded in physiological systems that regulated biological aging. The study's limitations included its cross-sectional design, residual confounding, and the use of overnight urine samples. Future studies were encouraged to explore pathways that may be modified through intervention and clarify how different dimensions of social integration influenced the molecular architecture of aging.

Source:

https://www.news-medical.net/news/20251012/Could-belonging-be-a-biological-shield-Researchers-uncover-aging-clues.aspx