

residents, subsidized housing residents were more likely to be women (66% vs. 55%, $p < 0.01$), racial/ethnic minorities (50% vs. 18%, $p < 0.01$), and to lack a high school diploma (50% vs. 20%, $p < 0.01$). They also had poorer health status, including higher rates of self-reported functional impairment (difficulty with 2 or more ADLs; 16% vs. 10%, $p < 0.01$), probable dementia (15% vs. 8%, $p < 0.01$), and frailty using the three-level Fried frailty index (55% vs. 26%, $p < 0.01$). Subsidized housing residents also had higher rates of hospitalization (29% vs. 22%, $p < 0.01$), move to a higher level of care (4% vs. 3%, $p < 0.01$), and death (10% vs. 7%, $p < 0.01$) compared to community-residing peers. These findings will help inform targeted interventions to improve aging in place for this vulnerable population.

SESSION 1310 (POSTER)

BIOLOGY OF AGING

ANTI-AGING PROTEIN CD9 AFFECTS AGE-RELATED HEART FAILURE

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The CD9 is transmembrane protein that plays a critical role in many cellular processes including aging associated cardiac pathologies. The heart function declines in the aged population. Ageing is strongly associated with many age-related conditions such as increased risk of heart failure. If aging can be prevented slowed down or even reversed, heart failure and other signs of aging could be controlled or even cured. It is unknown whether CD9 is cardioprotective. The objective of this study is to investigate whether a decline CD9 levels contributes to aging-related heart failure. Our data shows that CD9-deficient aged mice develop cardiac abnormalities and pathological cardiac hypertrophy, Cardioprotection by CD9 in old mice is followed by the downregulation of SIRT6 in the heart, and CD9 overexpressed exosomes ameliorates cardiac pathologies in treated mice and improves their long-term survival. Additionally, the serum level of CD9 decreased significantly in aged mice. CD9 overexpressed exosomes are cardioprotective and improve cardiac function in aged mice. These exosomes mediate their paracrine effects by attenuating, blood pressure, heart beat, reactive oxygen species and fibrosis. Remarkably, CD9 overexpression reversed fibrosis associated brain natriuretic peptide (BNP), Sirt6, and galectin 3 (Gal-3). These results provide a new perspective on the pathogenesis of cardiomyopathies and open new avenues for treatment of the disease.

THE ROLE OF POLYGENIC SCORE AND COGNITIVE ACTIVITIES ON COGNITIVE FUNCTIONING OF OLDER ADULTS

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Purpose of study: This study investigated whether and to what extent genetics for cognition and engagement in cognitive activities are related to trajectories of cognitive functioning in older adulthood. Furthermore, we explored whether engaging in cognitive activities could moderate the

effect of genetic traits on cognitive functioning in general and across different dimensions: fluid and crystallized intelligence. Design and Methods: Growth curve models were estimated using the sample of 3,129 individuals aged 50 or older (10,000 observations) in the U.S. from 2000-2012 waves of the Health and Retirement Study. Polygenic score for general cognition (PGS) was used to measure genetic traits for cognition, and the number of hours spent per week on each of nine cognitive activities was used to measure individuals' level of the engagement in cognitive activities. Results: PGS for cognition, reading books, using a computer, and playing cards/games/solving puzzles had positive effects on cognitive functioning. The positive effect of PGS on cognitive functioning was reduced from excessive TV watching. The positive effect of PGS on cognitive functioning was strengthened by spending more hours reading papers/magazines. The measure of fluid, rather than crystallized intelligence, appeared to drive these results. Conclusion: Findings suggests that while genetic factors predict cognitive functioning, engaging in different types of cognitive activities could yield different cognitive functioning trajectories in later life. Practical implications are that older adults should be more selective when choosing their leisure activities to promote cognitive health.

DETERIORATING HEALTH AMONG OLDER ADULTS AND CORTISOL: LONGITUDINAL EVIDENCE FROM THE MIDUS STUDY

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Various mental and physical health conditions common among older adults have been linked to cortisol dysregulation (i.e., blunting of daily cortisol patterns) in predominantly cross-sectional studies. Researchers have suggested that cortisol dysregulation interferes with regulatory functions throughout the body and brain, disrupting multiple biological systems, and contributing to the development or progression of negative health outcomes over time. Prospective studies are needed to investigate the causal direction of cortisol dysregulation and poor health outcomes. This study examined whether diurnal cortisol patterns predicted subsequent health deterioration using longitudinal data from the National Survey of Midlife in the US (MIDUS). Analysis was restricted to 1,336 participants who provided salivary cortisol (4 samples/day for 4 days) and health data in MIDUS II (2004-2009) and updated health data in MIDUS III (2013-2014) (mean age=56, 45% male, 94% White). We simultaneously modeled multiple measures of diurnal cortisol patterns and their relationships to changes in mental (depressive symptomology) and physical (self-rated physical health, functional limitations, and number of new chronic health conditions) health from MIDUS II to III. All indicators of physical health deterioration were associated with cortisol, though not all measures demonstrated relationships in the expected direction. Mental health change over time was unrelated to cortisol. Older age was also associated with increased functional limitations and more new chronic conditions but improvements in mental health over time. Findings suggest that diurnal cortisol patterns contribute to physical health deterioration over time, independent of age-related decline, but not mental health changes in later life.