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Sleep disorders and sleep deprivation have been linked to markers of biological aging, including methylation change and increases in white blood cell and neutrophil counts. However, little is known regarding the association of sleep duration with biological markers of aging. We investigated links of self-reported sleep duration with biological aging markers in 615 participants in the Baltimore Longitudinal Study of Aging (BLSA) aged ≥50 years (mean = 71.0 ± 11.2, 49.6% women, 68.8% white) with data on self-reported sleep duration in hours (i.e., ≤6 (n=131), >6 to 7 (n=234), >7 (n=250)), demographics, and genetic and methylation data (mDNA). Our aging biomarker outcomes were four epigenetic clocks (Horvath, Hannum, PhenoAge, and GrimAge), mDNA-estimated PAI1, and estimated granulocyte count. After adjustment for age, sex, and race, compared to those sleeping ≤6 hours, those reporting >7 hours of sleep had faster biological aging according to Hannum age-acceleration, PhenoAge, GrimAge, mDNA-estimated PAI1, and granulocyte count. In addition, sleep duration interacted with age, such that compared to individuals reporting ≤6 hours of sleep, individuals reporting >6 to 7 hours showed lower GrimAge with increasing age, and with sex, such that males with longer sleep duration (>6 to 7 and >7 hours) showed a lower granulocyte count compared to females. Findings suggest that both short and long sleep duration are associated with and may contribute to accelerated aging. Prospective studies in larger samples are needed to examine whether changes in sleep duration precede changes in aging biomarkers.

LONGITUDINAL COURSE OF GDF15 LEVELS BEFORE ACUTE HOSPITALIZATION AND DEATH IN THE GENERAL POPULATION

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Growth differentiation 15 (GDF15) is a potential novel biomarker of biological aging. To separate the effects of chronological age and birth cohort from biological age, longitudinal studies investigating associations of GDF15 levels with adverse health outcomes are needed. We investigated changes in GDF15 levels over 10 years in an age-stratified sample of the general population and their relation to the risk of acute hospitalization and death. Serum levels of GDF15 were measured three times in 5-year intervals in 2176 participants aged 30, 40, 50, or 60 years from the Danish population-based DAN-MONICA cohort. We assessed the association of single and repeated GDF15 measurements with the risk of non-traumatic acute hospitalizations. We tested whether changes in GDF15 levels over 10 years differed according to the frequency of hospitalizations within 2 years, or survival within 20 years, after the last GDF15 measurement. The change in GDF15 levels over time was dependent on age and sex. Higher GDF15 levels and a greater increase in GDF15 levels were associated with an increased risk of acute hospitalization in adjusted Cox regression analyses. Participants with more frequent admissions within 2 years, and those who died within 20 years, after the last GDF15 measurement already had elevated GDF15 levels at baseline and experienced greater increases in GDF15 levels during the study. The change in GDF15 levels was associated with changes in C-reactive protein and biomarkers of kidney, liver, and cardiac function. Monitoring of GDF15 starting in middle-age could be valuable for the prediction of adverse health outcomes.

METABOLIC SYNDROME AND NEUROCOGNITIVE FUNCTION AMONG OLDER HISPANICS/LATINOS WITH HIV

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Neurocognitive impairment is prevalent among persons with HIV (PWH), particularly among Hispanics/Latinos/as/x (henceforth Hispanics). We examined disparities in HIV-associated neurocognitive function between older Hispanic and non-Hispanic White PWH, and the potential role of metabolic syndrome (MetS) in explaining these disparities. Participants included 116 community-dwelling PWH ages 50-75, who were enrolled in a cohort study in southern California (58 Hispanic [53% Spanish-speaking] and 58 age-comparable non-Hispanic White; Overall group: Age: M=57.9, SD=5.7; Education: M=13, SD=3.4; 83% male, 58% AIDS, 94% on antiretroviral therapy [ART], 4% detectable plasma RNA). A global neurocognition score was derived from T-Scores on a comprehensive neurocognitive battery, with separate demographic adjustments for English and Spanish-speakers. MetS was ascertained via standard criteria that considered central obesity, elevated triglycerides, low high-density lipoprotein cholesterol, and elevated fasting glucose, as well as current medical treatment for these conditions. Covariates examined included sociodemographic, psychiatric, substance use and HIV-disease characteristics. Hispanics had higher rates of MetS (56%) than non-Hispanic Whites (37%; $p<.05$). A stepwise regression model on global neurocognition including ethnicity and covariates that differed between ethnic groups, selected only Hispanic ethnicity as a significant predictor (B=-3.82, SE=1.27, $p<.01$). A comparable model also including MetS showed that both Hispanic ethnicity (B=-3.39, SE=1.31, p=.01) and MetS (B=-2.73, SE=1.31, p=.04), were significantly associated with worse global neurocognition. Findings indicate that MetS does not fully explain disparities in neurocognitive function between Hispanic and non-Hispanic White older PWH, but rather is an independent predictor of neurocognitive function along with Hispanic ethnicity.