DEMENTIA RISK IN OLDER VETERANS WITH FRAILTY: A CROSS-SECTIONAL STUDY

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Frailty, a clinical syndrome characterized by vulnerability to stressors resulting from multisystemic loss of physiological reserve, predicts future cognitive decline. However, frailty has also been proposed as a dementia risk factor, predicting future cognitive impairment. The study aim was to determine frailty in older veterans and its association with risk of dementia. Community-dwelling Veterans ≥50 years completed a mailed socio-demographic questionnaire and Self-Administered Gerocognitive Examination (SAGE), July 2019-May 2020. The information was complemented with EHR data. We calculated the CAIDE score, a validated tool predicting dementia (≥6 points= high risk 20 years later) and the 31-item VA frailty index data (frail ≥.20, non-frail ≤.20). After adjusting for socio-demographic characteristics, smoking, alcohol/substance abuse, OSA and anticholinergic use, odds ratio (OR) and 95% CI were calculated using BLR to assess the cross-sectional association between frailty and dementia risk (CAIDE ≥6 points and MCI). The survey response rate was 19.75% (1,073 of 5,432). Participants mean age was 68.38 (SD=8.49) years, 57.50% (n=617) Caucasian, 69.34% (n=744) non-Hispanic, 95.81% (n=1,028) male, and 36.72% (n=394) frail. 11.84%(n=127) screened positive for MCI and 15.38% (n=165) for dementia. 689 (75.88%) veterans were at high risk for dementia of whom 426 (61.83%) were non-frail and 263 (38.17%) were frail. Frailty was cross-sectionally associated with higher risk for dementia in older Veterans, adjusted OR:1.45 (95%CI:1.016-2.070), p=.041. The mailed screening was a feasible and practical approach to screen for dementia risk. Early identification of patients with frailty can help in the implementation of interventions aimed at preventing or delaying dementia.

DEPRESSION MEDIATES THE CAUSAL EFFECT OF FOOD INSECURITY TRANSITION ON COGNITIVE FUNCTION

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Despite substantial evidence on the relationship between food insecurity and worse cognition, few studies have examined the underlying mechanisms. This study examined whether and to what extent depression mediated the casual effect of food insecurity on cognition in midlife. Longitudinal data from the Coronary Artery Risk Development in Young Adults Study were used. The 2000 survey was used as our study's baseline, when respondents aged 33-45. We lagged the explanatory and mediator variables to set up temporality. Food insecurity transition from 2000-2005 surveys was recoded (persistent food secure, persistent food insecure, became food insecure, and became food secure). Depression was measured in 2010, while cognitive functions were measured in 2015. Causal mediation analysis examined and decomposed the direct and indirect effect. Compared to persistent food security, both persistent and became food insecure were linked to worse cognition, after adjusting demographic and cardiovascular risk factors. Further, for both exposure groups, depression had significant and strong mediation effects (natural indirect effect ranged from -0.037 to -0.018). Specifically, as respondents experienced persistent food insecurity or became food insecure, their depression increased, which, in turn, reduced their cognition. The proportion of the total effect of food insecurity transition on cognition mediated by depression ranged from 7-24% for processing speed, verbal memory, cognitive, and language functions. Persistent and became food insecure reduced cognitive function directly or indirectly through increasing depression among middle-aged adults. Interventions targeting either food insecurity or treatment of depression among food insecure individuals may have the potential to slow premature cognitive aging.

DESIGNING A GENETIC SCREEN TO IDENTIFY FACTORS REQUIRED FOR MEIOTIC NUCLEAR REJUVENATION

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During the natural cycle of life, most eukaryotic organisms grow old, age, and die. A common natural mechanism by which organisms "reset" their lifespan is through sexual reproduction; however, how this rejuvenation takes place remains unknown. My lab has found that meiosis in budding yeast, the developmental program that forms sex cells, eliminates age-induced damage. This involves the formation of a novel nuclear compartment, the Gametogenesis Uninherited Nuclear Compartment (GUNC), which acts as a trash can for accumulated age-induced damage. To understand the molecular details of this process, I worked on designing a screen for genes involved in GUNC formation. My mentor and I fused three different proteins targeted to the GUNC and a protein that is able to bind to a drug-resistance plasmid, in order to couple the inheritance of a selectable DNA marker with the elimination of age-induced damage. Initial testing of these three fusion proteins suggested that they were unable to successfully target the plasmid to the GUNC; as such, testing of additional candidate proteins is necessary. We plan to eventually use this system to identify mutations that disrupt GUNC formation and cause inheritance of the drug-resistance plasmid. By identifying and perturbing proteins involved in GUNC formation, we are hoping to be able to drive the inheritance of specific types of age-induced damage, allowing for the determination of what a symptom versus a cause of aging is.

DIABETES, OBESITY, AND OSTEOPOROSIS IN AN ETHNICALLY DIVERSE POPULATION OF WOMEN RECEIVING OSTEOPOROSIS SCREENING

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