Is the subjective cognitive decline an indirect measure of stress burden? A combined assessment of allostatic load, cortisol reactivity and perceived stress among older adults

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Background: Prolonged exposure to stress mediators, including cortisol, can compromise neural structures in a cumulative "wear and tear" process known as allostatic load (AL). Signs of AL represent a risk condition and therefore an interesting target for diseases prevention. Although several studies demonstrated that chronic stress represent a risk to cognitive impairment, the "wear and tear" signs that precede the illness are unknown. The complaint about memory (Subjective memory decline – SMD) has been considered a pre-clinical sign of cognitive impairment and therefore may represent a sign of the stress burden on the nervous system. However, there is no evidence to support this assertion. Objective: To analyze whether SMD is associated with AL indicators.

Methods: SMD using the Memory Assessment Complaint - Questionnaire (MAC-Q), Perceived Stress (PS), salivary cortisol induced by the Trier Social Stress Test (TSST) and a AL index was evaluated in 227 individuals aged ≥50 years with preserved cognitive and functional capacities. AL index was composed of mediators of the neuroendocrine, immunological, metabolic and cardiovascular systems.

Results: Controlling for confounders, MAC-Q scores were associated with PS [(95% CI = 0.093–0.261; $B = 0.177$); $p = < 0.001$], AL index [(95% CI = 0.092–0.751, $B = 0.422$); $p = 0.012$] and with cortisol concentration [(95% CI = 0.010–0.170; $B = 0.09$); $p = 0.028$]. Conclusion: The SMD was associated with AL indicators suggesting that complaint about memory may signalize the "wear and tear" of neural systems targets of the stress and therefore a risk condition to cognitive disorders.

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Modeling the age-varying link between smoking and telomere length in a large sample of US adults

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Background: Telomere length and rate of telomere shortening is recognized as a marker of cellular aging and a potential 'record' of lifetime stress exposure. Smoking is linked to shortened telomeres in a dose-dependent manner. While the link between smoking and leukocyte telomere length (LTL) has been investigated cross-sectionally and longitudinally, little is known about the potential age-dependent impact of smoking. We examined whether the association between LTL and smoking varies across age using a nationally representative sample of adults.

Methods: Smoking behavior and LTL were surveyed during the 1999–2002 NHANES data collection waves for 2,424 current and former smokers between the ages of 20–77. Pack years were calculated as number of packs per day divided by number of years smoked. Using time-varying effect modeling (TVEM), the association between LTL and pack years was estimated as a flexible, non-parametric function of age controlling for sex, race/ethnicity, income, and waist-to-height ratio.

Results: Overall,mean LTL among smokers decreased at a steady rate throughout adulthood. A significant negative association between pack years and LTL was detected specifically among smokers between the ages of 32–47 and 55–70. No significant association was found between pack years and LTL for individuals under 32, or between the ages of 47 and 55.

Discussion: These findings highlight key age periods when the link between smoking history and telomere length is most salient. Further, they demonstrate the value of TVEM in advancing our understanding of developmental trajectories of associations between telomeres and exposures across the life course.

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Associations between biological variables involved in estrogen signaling in pre- and post-menopausal women – Findings from the women 40+ healthy aging study

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Background: Low and fluctuating levels of estradiol (E2) have been associated with increased depression and anxiety in women. However, E2 replacement therapy is not always successful in reducing affective symptoms and studies point to differential signaling mediated by estrogen receptors (ERs). Increasing evidence suggests associations between levels of methylation within the estrogen receptor (ER) genes, E2, variable number tandem repeats (VNTRs) polymorphisms, and chronic stress. The aim of this study was to assess possible associations between these variables in a healthy cohort of pre- and post- menopausal women.

Method: Saliva and blood were collected from 130 women (age: 40–73 years). Methylation of targeted CpG sites located in the ER and HPA axis genes (*ERS1*, *ERS2*, *GPER*, *NR3C1*, *FKBP5*), VNTRs in the ER genes, and hormones (E2, cortisol) levels were assessed by Next Generation Sequencing (NGS), capillary electrophoresis and ELISA, respectively.

Results: Preliminary analysis ($N = 8$) suggest associations between hormones levels (E2, cortisol) and methylation of targeted CpG sites in the *NR3C1* and *ESR1*.

Discussion: Our preliminary findings point to existing associations between the assessed variables. Provided we will be successful in confirming our hypothesized links in the total sample ($N = 130$), this would shed light on the mechanisms underlying estrogen signaling in healthy women. Eventually, these findings could inform research on how differential estrogen signaling may be linked with the development of female depression and anxiety.

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