

EPV0675

Pathogenetic substantiation of depressive disorders in old age as factors of premature brain aging

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Introduction: A systems approach to understanding late-onset depression points to its association with cardiovascular disease, cerebrovascular disease, tissue and organ trophic disorders, cognitive impairment, anergy, and decreased lifespan. These features are consistent with progressive aging

Objectives: literature analysis

Methods: general scientific method

Results: **Cellular senescence** is characterized by irreversible cell growth arrest. Characteristic features of senescent cells include increased cell size, accumulation of β -galactosidase and lipofuscin in the cytoplasm, accumulation of DNA damage foci, condensed heterochromatin regions, shortened telomeres, and increased expression of cell cycle regulatory markers. Telomere attrition has been linked to depression. Individuals with MDD have shorter white blood cell length compared to subjects without a history of depression. Telomere attrition is also associated with more unstable IL-6 levels and oxidative stress markers in major depressive disorder. Postmortem studies of the brain have revealed that severe depression leads to depletion of various areas of the cerebral cortex and oligodendrocytes. According to the authors, there is a link between severe depressive disorders and cellular aging of the brain. **Mitochondrial dysfunction.** Mitochondrial dysfunction in depression and aging includes genomic instability, defects in biogenesis, electron transport chain and mitochondrial scavenging, nutrient regulation mechanisms, proteostasis. In late depression, the number of mitochondrial DNA copies decreases. Increased production of ROS is a marker of mitochondrial dysfunction. In contrast, depressed individuals have elevated lipid peroxidation markers and an imbalance between oxidative stress and antioxidant stress markers. MtDNA oxidation and fragmentation are consequences of elevated OS, with high levels of fragmented mtDNA being associated with more severe depressive episodes and also a biomarker of aging and tissue damage. In patients with late-onset depression, ccf mtDNA has been found to correlate with chronic levels of IL-6, a master regulator of inflammation and a major component of SASP.

Conclusions: Total scores assess indicators of mitochondrial dysfunction, telomere length, cognitive tests, and structural changes on MRI in women with late depression higher than in elderly men.

Disclosure of Interest: None Declared

EPV0676

Hair cortisol concentration in early pregnancy on postpartum depressive symptoms

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Introduction: Knowledge of the prenatal predictors of postpartum depressive symptoms is of fundamental importance in preventing them.

Objectives: This study aimed to examine whether first trimester maternal hair cortisol influenced maternal postpartum depressive symptoms.

Methods: The women ($N = 75$) were tested twice: in the first trimester of pregnancy and within three months after delivery. In the first trimester, they had hair samples taken and were examined using a sociodemographic survey and questionnaires: the Edinburgh Postnatal Depression Scale (EPDS), the Perceived Stress Scale (PSS-10), and the Zimbardo Time Perspective Inventory (ZTPI). After delivery, women completed a survey about the course of delivery and their child's health, EPDS, PSS-10, and ZTPI.

Results: A multiple hierarchical regression analysis was conducted to identify the predictors of postpartum depressive symptoms. In the first step, presence of maternal chronic illness and birth length were added, since these variables were associated with postpartum stress in our study. In the second step, we added the scores on depressive symptoms (EPDS) and stress (PSS-10) in the first trimester. After including hair cortisol concentration in the third step, depressive symptoms, stress, and hair cortisol concentration in the first trimester turned out to be predictors of postpartum depressive symptoms. The results showed that higher levels of depressive symptoms and stress and a lower level of hair cortisol concentration in the first trimester were associated with higher levels of postpartum depressive symptoms. We conducted mediation analyses to test time perspective as a mediator in the relationship of hair cortisol concentration in the first trimester to postpartum depressive symptoms. The results suggested that fatalistic time perspective mediated the relationship between hair cortisol concentration in the first trimester and postpartum depressive symptoms. In other words, women with lower hair cortisol concentration in the first trimester were more likely to develop postpartum depressive symptoms, and this relationship was explained by fatalistic time perspective.

Conclusions: The results of our study indicate that women with very low hair cortisol concentration in the first trimester are at greater risk for postpartum depression. These are the first data to show that low level of cortisol in the first trimester can be a predictor of maternal postpartum depressive symptoms. The results of our study suggest that examining hair samples in the first trimester of pregnancy may be a valuable method for identifying women at risk of developing postpartum depression.

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EPV0677

Mindfulness-Based Interventions for Difficult-to-Treat Major Depressive Disorder: A Systematic Review and Meta-Analysis

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