In this workshop I will present data from our research group and others pertaining to the biological mechanisms underlying accelerated cellular aging in PTSD. Most, but not all, studies have found that PTSD is associated with shorter mean leukocyte telomere length, an indicator of accelerated cellular aging. Mitochondrial dysfunction has been implicated in PTSD and our research group found evidence of a "u-shaped" relationship between PTSD symptom severity and mitochondrial DNA copy number. For what concerns immunity, we have recently found that PTSD subjects have increased blood levels of pro-inflammatory markers, a more senescent and dysfunctional profile of NK cells and impaired synthesis of nitric oxide. Finally, I will discuss the possibility of accelerated *epigenetic* aging in combat-exposed individuals with and without PTSD, using DNA methylation data.

Disclosure of interest The author has not supplied his declaration of competing interest.

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Symposium: How to Integrate Stress - (Epi) Genetics and Imaging and What Does It Tell Us

S038

Stress Hormone System and Epigenetics in Depression

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Background Exposure to early life adversity (ELA) has been identified as a major risk factor in the development of major depressive disorder (MDD). It is hypothesized that a mediating mechanism may be environmentally induced alterations in gene function. In our REDEEM (Research in depression: endocrinology, epigenetics and neuroimaging) project we are examining possible epigenetic difference in some previously investigated target genes relevant to depression. To this end, methylation of the following genes were measured: NR3C1 (HPA axis), SLC6A4 (serotonin neurotransmitter function), and CD3ε (T cell receptor gene). We also looked at possible trans-generational transmission of epigenetic markers in a mother-baby sample.

Methods DNA was isolated from depressed patients and controls and babies and a portion of the above genes, encompassing our regions of interest, were amplified by PCR. Percentage methylation levels were measured by pyrosequencing. mRNA was also measured for some gene products to see if function was related to methylation. HPA axis function was measured with serial saliva samples throughout the day.

Results to date: Methylation was increased in the CD3ε promoter in depressed subjects relative to controls. In the total group, those exposed to ELA had significantly increased methylation at this site. Levels of CD3ε mRNA levels were inversely related to methylation. There were some relationships between maternal ELA and baby methylation at the sites examined.

Conclusions Consistent with an allostatic model of ELA damage, our findings suggest an alteration in epigenetic function in acquired immunity and the HPA axis, mediated by ELA. Findings will be discussed.

Disclosure of interest The authors have not supplied their declaration of competing interest.

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S039

Stress and the serotonergic system, observations from pet imaging

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Introduction Stress response and the neuroendocrinologic factors through which it is mediated are disturbed in anxiety and in affective disorders. While acute stress is thought to result in hypothalamus-pituitary-adrenal- (HPA) axis hyperactivity (Varghese 2001), chronic stress may result in decreased HPA-response (Booji 2013). Antidepressant treatment, on the other hand, is thought to realign HPA–axis activity (Schüle 2007).

On the other hand, dysregulation within the serotonergic neurotransmitter system is understood as a central moderator in the pathophysiology of affective and anxiety disorders. Serotonergic transmission both regulates- and is regulated by- glucocortocoids. Cortisol results in an increase in serotonin synthesis and release while serotonergic transmission is thought to downregulate HPA-axis activity (Lanfumey, 2008). Positron emission tomography (PET) studies have demonstrated the link between the serotonergic system and the HPA-axis in humans in vivo. For example, a negative correlation between cortisol and $5HT_{1A}$ receptor levels in various brain regions has been shown (Lanzenberger, 2010). SERT expression, on the other hand, was shown using PET to be positively related to HPA-axis reactivity (Frokjaer 2013).

Methods n.a.

Aims Available literature on interactions between the HPA-axis and the serotonergic system will be discussed with a focus on data acquired via PET studies.

Results n.a.

Conclusions The interaction between the serotonergic system and the HPA-axis is likely bilateral and may be understood as a neurobiological link by which stress may foster the development of depression and anxiety.

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S040

Association of stress hormone system, epigenetics and imaging

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