in concentrations of markers such as cortisol, dehydroepiandrosterone (DHEA), and its sulfate form DHEA-S. Studies investigating either basal alterations in hormone levels, or HPA axis reactivity or changes due to psychotherapeutic treatment, are reviewed.

Methods: Various literature databases were screened and a snowball search system was used for the identification of relevant studies. Additionally, we searched for unpublished data. Both control group designs and single group designs were suitable for inclusion. With regard to the PTSD group, studies examining subjects with clinical PTSD were eligible for inclusion. All groups had to be diagnosed with standardized diagnostic criteria. With regard to biomarker assessment, studies using single as well as multiple time points of measurement were included. Moreover, studies applying hormone assessment in urine, blood, saliva and hair and studies measuring DHEA and DHEA-S in blood and saliva were eligible. A rating of the primary studies was conducted regarding study quality and risk of bias and concerning hormone assessment. For effect size estimation, studies were combined in separate data sets according to their design.

Results and conclusions: Data analysis is still in progress. Results will be presented and discussed critically.

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Anxiolytic neuropeptides in posttraumatic stress disorder (PTSD) – Evidence from patients and various animal models

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Background: Anxiolytic neuropeptides such as oxytocin and the neuropeptides S and Y increasingly emerge as promising novel drug targets for affective and stress related disorders. To establish them as drugs for human use, their regulation and functions remain to be fully unraveled.

Methods: First, we quantified the expression of oxytocin, NPS and NPY and of their receptors in peripheral blood of PTSD patients versus healthy controls both at baseline and during social stress test exposure as well as in trunk blood and different brain regions of mice suffering from a PTSD-like syndrome which was treated with fluoxetine versus placebo and, second, in stressed versus unstressed *NPY* KO and *Fkbp5* KO mice versus wildtype. Third, we tested the efficacy of intranasal oxytocin versus placebo on PTSD symptoms elicited by trauma script exposure in PTSD patients and analyzed its cardiac effects.

Results: First, blood oxytocin levels were reduced in PTSD patients versus controls in the morning and PTSD-like mice versus unstressed mice in trunk blood–fluoxetine rescued both mouse behavioral symptoms and reduced blood oxytocin levels. Second, PTSD patients lack the stress-induced decrease in peripheral OTXR mRNA levels observed both in controls and in the hippocampus of wildtype forced swim-stressed mice. Third, intranasal oxytocin reduced provoked PTSD symptoms in PTSD patients despite exerting sympathomimetic effects.

Conclusions: All three neuropeptides and some of their receptors are modulated by stress. In particular oxytocin and its receptor seem to play a central role in the maintenance of PTSD symptoms both in patients and in mice.

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PTSD-related shifts in HPA axis, DHEA levels in chernobyl nuclear plunt clean-up workers

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Background: Chernobyl nuclear power plant liquidators suffer from posttraumatic stress syndrome (PTSD). Dysregulation of the hypothalamic pituitary adrenal axis (HPA) has been implicated in PTSD. Diverse hormonal abnormalities are common in Chernobyl clean-up workers. However, the association between HPA alterations and PTSD in these patients has not previously been explored.

Methods: 87 Chernobyl nuclear power plant male liquidators and a healthy male control group were employed. HPA status was studied by determining early-morning ACTH and cortisol using the radio immune assay. Furthermore, we used the radio immune assay for DHEA plasma levels estimation.

Results: Overall analysis of the PTSD group identified decreased plasma cortisol and lipid/protein oxidation products relative to the control individuals as well to the plasma control individuals. PTSD-related liquidators displayed lower levels of DHEA in comparison to healthy controls, but higher levels in trauma compared with control. Low plasma cortisol levels were associated with elevation of antioxidant enzymes activities. The extent of free radical oxidation was diminished in the PTSD group but not in the trauma control individuals, in which similar plasma levels of lipid/protein oxidation products were determined.

Conclusions: Our results suggest the PTSD-related liquidators exhibited disturbance in HPA axis and in DHEA level.

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Neural and endocrine stress reactions in healthy women with and without severe early life trauma

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Background: Posttraumatic stress disorder (PTSD) patients with severe childhood trauma exhibit a disturbed fronto-limbic network. Additionally, PTSD and trauma-exposure has been associated with alterations of endocrine and autonomic stress systems. Interestingly, some studies found beneficial effects of cortisol administration on cognition in PTSD. However, it is largely unclear whether alterations in stress-systems are mainly PTSD associated