This presentation describes current knowledge about the treatment of patients with the dual diagnosis of post-traumatic stress disorder and substance misuse, a population that is typically considered ''difficult to treat''. We will cover background on PTSD and substance misuse (including rates, the typical client, models and stages of treatment, and clinical dilemmas). Clinical interventions will also be addressed, with a focus on "Seeking Safety", an evidence-based model for PTSD and substance misuse.

Symposium: Recent advances in the understanding of adolescent conduct disorder

S59.01

Emotional processing in conduct disorder: Data from psychophysiology and neuroimaging

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Per definition adults with antisocial personality disorder have met the diagnostic criteria of conduct disorder since adolescence at least. Those who have started to show antisocial behavior before the age of ten tend to exhibit even more severe aggressive behavior throughout early adulthood. However, conduct disorder is not a homogenous category that is characterized by a steady development towards a specific antisocial disorder in adulthood. Within the category of conduct disorder there are particularly high differences in emotional processing and an individual's capability to regulate emotions. Regarding the neurobiological correlates of emotional processing in subjects with conduct disorder, psychophysiological data suggest high stability over time with the main finding of hypoarousal in resting states and autonomous hyporeagibility to emotional stimuli. Findings from functional neuroimaging are more heterogeneous probably reflecting subtypes of antisocial disorders which are subsumed under the category of conduct disorder. Developmental research in psychiatry needs a multidimensional diagnostics including a precise psychopathological characterization and subdifferentiation. Although genetic dispositions are stable, they interact with changing psychosocial factors which take influence on the developing brain in more or less vulnerable stages.

Symposium: Vulnerability factors in depression across the life span

S04.01

Genetic determinants of neurobiological vulnerability markers in depression

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Background and Aims: Neuroendocrine changes of the stress hormone system and REM sleep abnormalities are potential vulnerability markers for depression. Investigating underlying genetics provides new insights into the molecular pathways of these endophenotypes and into the pathophysiology of depression vulnerability. We selected a high-risk linkage and endophenotype approach, i.e., we investigated REM sleep abnormalities (REM density) and altered stress hormone regulation (dex/CRH test) in families with a high prevalence of major depression.

Methods: Eleven families were so far included, comprising 82 high risk family members. 32 of them were unaffected, 33 remitted, and 17 suffered from an affective disorder at the time of the investigation. Illumina Infinium Whole Genome genotyping with 100k bead chips was performed. Variance component (VC), and a quantitative trait linkage analysis (QTL) on polysomnographic (REM density) and neuroendocrine vulnerability markers (dex/CRH test) were applied.

Results: Linkage analysis (VC, QTL) revealed suggestive linkage (LOD score > 2) for altered REM density at loci of the chromosomes 2, 4, 8 and X. The linkage results on chromosomes 2 and X correspond to previous findings. No results were obtained with a classical diagnosis based linkage approach.

Conclusions: The use of quantitative vulnerability markers in a high risk family study and a SNP based whole genome approach revealed a number of loci with suggestive linkage, that were not detectable with the classical linkage approach. Our findings suggest the suitability of investigating vulnerability marker in combination with a SNP based whole genome approach in complex disorders like depression.

S04.02

Psychological vulnerability factors and neuroendocrine and sleep regulation in healthy children and adolescents

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Background and Aims: The presentation aims at summarizing current knowledge about sleep in children and adolescents and at describing possible factors influencing their sleep.

For preschoolers, there is evidence that objectively assessed (sleep-EEG, actigraphy) poor sleep is associated with increased endocrine activity; this is to say, with increased morning cortisol secretion, an associative pattern observed so far only in adults. Furthermore, poor sleep and increased cortisol secretion are associated with emotional and behavioral difficulties.

During life span, notable changes occur with respect to sleep quantity and quality. Compared to childhood, in adolescence, three prominent changes occur: First, sleep quantity declines from about 10 hours at 10 years of age to between 6.5 and 8.5 hours in older adolescents. Second, a marked shift towards a longer sleep duration and later bed time from school nights to weekend nights is observable. Third, daytime sleepiness (20%) and insomnia symptoms (25%) are common among adolescents.

Among a variety of factors affecting adolescents' sleep, we could show that negative parenting styles unfavorably influenced adolescents' sleep quality, suggesting that even 18 years old adolescents may be far away from been emotionally independent from their parents. Furthermore, the so-called weekend-shift was correlated with increased sleepiness during the week, suggesting that irregular sleep schedules may negatively influence sleep quality and daytime functioning.

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