

Background and Aims: Both insomnia and sleep duration have previously been linked with a range of adverse outcomes, but no studies have explored their relative effect on subsequent work disability. The aim of the present study was to investigate the contribution of insomnia and sleep duration to later short and long-term work disability.

Methods: Data on insomnia, sleep duration and potential confounders were gathered from 7849 working persons (40–44 years). The outcome was award of disability pension 18–48 months later, as registered in the National Insurance Administration.

Results: Insomnia was a strong predictor of both short- and long-term work disability, and this effect remained significant in the fully adjustment model. Reduced or excessive sleep duration was significantly associated with subsequent work disability in the fully adjusted model; only in the crude model did sleeping less than 5.5 hours predict work disability.

Conclusions: The present study provides further evidence that insomnia is a strong and independent risk factor work subsequent work disability, while at the same time ruling out that this association is caused by a reduced or excessive sleep duration.

Symposium: Addiction and psychiatric comorbidity: Rule or exception?

S03.01

Cannabis and psychosis

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This contribution will explore UK-based research developments in substance misuse and mental illness over the last 20 years. The main body of work revolves around research largely, but not only, funded by the Department of Health from the late 1990s. Early research revolved around alcohol, especially alcoholic hallucinosis: the relationship with schizophrenia-like illness was examined and the conclusion at that time on the basis of twin, family and clinical studies, was that very few cases did develop into schizophrenia. More recently, large general population epidemiological, and medium or small scale clinical studies, have been undertaken on the relationship between substance misuse (sometime specifically on cannabis) and the later development of mental illness especially psychosis. The presentation will aim to draw parallels with the current debate around the link between cannabis and psychosis and urges caution in too rapid an assertion that cannabis is necessarily 'causal' and the clinical and policy implications of the misinterpretation of evidence. A proposal will be put forward that the genesis of psychotic illness in alcohol misuse is revisited using more sophisticated research methodologies. Given the changing landscape of substance use in the UK, particularly the fashion of polysubstance use and the recognition that this may be associated with psychotic illness, other drugs that are associated with psychotic illness should be similarly investigated to determine whether there is a common mechanism that might throw light on understanding the relationship between substance use and psychotic illness or schizophrenia.

S03.02

Cocaine addiction and psychotic disorders

E.J. Franzek, I.C. Elsenaar. *Bouman Kliniek, Rotterdam, The Netherlands*

In order to create hypotheses about the relationship between the effects of cocaine and the development of psychotic symptoms we conducted a pilot study with 55 patients. All patients were personally investigated on their current psychopathology. The 55 patients (more than 80% were males) were distributed in 5 diagnostic subgroups:

- 1) Addiction without a further AS I diagnosis (n=10)
- 2) Addiction and psychotic disorder related to cocaine (n=8)
- 3) Addiction, psychotic disorder related to cocaine and another AS I disorder (n=10)
- 4) Addiction and a schizophrenic spectrum disorder (not schizophrenia) (n=10)
- 5) Addiction and schizophrenia (n=17)

We investigated if psychotic symptoms occurred during cocaine use, if these symptoms depended on the dosage of cocaine and if similar symptoms also occurred without cocaine but during stress.

Hypotheses: Schizophrenic patients receiving neuroleptics respond completely differently to cocaine use than all other groups including the schizophrenia spectrum group (without schizophrenia). When using cocaine the schizophrenic patients did not experience new psychotic symptoms, moreover, many of them reported being less bothered by delusions and hallucinations compared to times without cocaine. In all other groups positive psychotic symptoms occurred dependent on the dosage of cocaine, in some of them similar symptoms were triggered only by stress. The symptoms that are triggered by cocaine have a dosage dependent hierarchical structure: (1) mistrust, (2) plus delusions of reference with fear, (3) plus delusions of persecution with anxiety or panic and illusions, (4) plus hallucinations like threatening voices and noises, (5) plus disorganized behavior.

S03.03

Addiction and add

B.J.M. van de Wetering. *Bouman GGZ, Rotterdam, The Netherlands*

Attention Deficit Disorder is a well recognized developmental disorder in children. The disorder has been found to persist in adulthood in about 50% of the cases with an estimated adult prevalence of about 3.5%. ADD in adulthood is characterized by high rates of one or more concurrent psychiatric disorders like mood disorders, anxiety disorders and personality disorders.

In the last decade interest has increased in the co-occurrence of ADD and substance use disorders (SUD). Approximately 15–25% of ADD patients have a lifetime diagnosis of SUD involving a wide range of legal and illegal substances. Given the different spectrum of ADD symptomatology as compared to the childhood clinical picture, the high rates of psychiatric co-morbidity and the interference of substances with ADD symptoms and course, diagnosis and treatment of these patients constitute a real challenge.

Recent clinical research is directed at unravelling specific risk factors for developing SUD during the course of ADD in later life. Early diagnosis and adequate treatment seem to diminish this risk. Childhood conduct disorder and increased impulsivity are considered to imply an increased risk for developing not only SUD but also criminality in (early) adulthood. Clinical implications of these findings will be discussed during this presentation.

S03.04

The link between PTSD and substance misuse

L.M. Najavits. *Harvard Medical School, Boston, MA, USA*

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 hyporeactivity 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 being 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.