lower degree of familial aggregation; 3) there was no evidence of cross-aggregation between mood disorders and migraine.

Conclusion: Our data confirm familial aggregation of bipolar-I disorder, unipolar depression and migraine. The finding of an increased risk of migraine with aura among relatives of probands with unipolar depression alone could indicate partially shared etiological factors underlying unipolar depression and subtypes of migraine.

SS-10-05

Somatic health and illness and depressive symptomatology: Data from a population health survey

M. Kovacs, M. Kopp. *Semmelweis University, Budapest, Hungary*

Objective: To investigate the rate and associations of self-reported physical health and illnesses and depressive symptomatology, and also their impact on everyday psychosocial functioning and quality of life.

Methods: Data were obtained from the Hungarostudy 2002, a national representative health survey of the adult (18+) Hungarian population (Kopp et al., 2002, N=12668). Depressive symptomatology was measured by the shortened version of the Beck Depression Inventory (BDI). Participants signed their main illness caused the highest problems in the previous 12 months. General quality of life was measured by the WHO Well-being Index, and the Illness Intrusiveness Scale showed the extent of impairment in psychosocial functioning caused by the main illness.

Results: BDI scores were negatively correlated with the WHO Well-being, the self-assessed general health state, and positively with the Illness Intrusiveness scores. 69.6% of those with BDI above 19 reported decreased or missing well-being, this rate was 20.8% in those with BDI below 19 (OR: 8.7, 95%CI: 7.8-9.8). Depression was the main cause of daily limitations in 1.7% of the whole sample (2.3% in women and 0.8% in men). However this rate was much lower than the rate of those who signed musculosceletal (18.0%), or cardiovascular diseases (13.4%), regarding the extent of impairment, those with depression had the highest Illness Intrusiveness mean scores in both sexes, in all age groups.

Conclusion: Although depression was only the seventh most frequent cause of daily limitation, it caused the highest negative impact of everyday functioning, preceding all the other major illness groups, and significant decrease in the general well-being.

Tuesday, April 5, 2005

SS-16. Section symposium: Biological rhythm in psychiatry

Chairperson(s): Manfred Ackenheil (München, Germany), J.P. Macher (France)
16.15 - 17.45, Gasteig - Room 0.131

SS-16-01

Chronobiology and mood disorders

A. Wirz-Justice. *Universitätsklinik Psychiatrie Center für Chronobiologie, Basel, Switzerland*

Chronobiological abnormalities in mood disorders are well known to clinicians, however, the novel, non-pharmacological therapies that have arisen from biological rhythm research are not yet in general practice. The circadian clock in the suprachiasmatic nucleus drives the 24-hour pattern in psychological, physiological, neuroendocrine and biochemical functions, including the sleep-wake cycle. The main zeitgeber for the circadian clock is light, a treatment that has been most successfully applied in winter depression, but newly, also as an adjuvant to SSRIs in non-seasonal major depression. Bright light exposure rapidly increases serotonin turnover, thus acting on similar mechanisms as antidepressant drugs. The pineal hormone melatonin transduces the length of night into a "nighttime" signal. Exogenous administration of melatonin can resynchronise disturbed sleep-wake rhythms (such as in the blind); in depression, sleep quality is improved without any effect on mood. In spite of much research into circadian rhythms in affective disorders, there is no consensus as to which part of the clock mechanism has gone awry. When desynchrony occurs between rhythms, shifts and decrements of mood can be measured even in healthy subjects. Sleep deprivation or shifting sleep timing can improve depression. Chronotherapeutics - light, melatonin, (partial) sleep deprivation or sleep advance - have the disadvantage of not being patentable, not being in "easy-to-take" pill form, and not being promoted as simple everyday adjunct treatment strategies. They do have the advantage of appealing to the zeitgeist - patients would rather not take drugs - while being well-tested and neurobiologically active treatments.

SS-16-02

Role of melatonin in the circadian system, melatonin and circadian organization of functions, melatonin and biological rhythms

P. Pevet. *Institut Federatif des Neurosciences de Strasbourg, Strasbourg, France*

The temporal organization of living organisms relies on clock(s) that generate rhythms and are capable for being entrained to environmental factors. Such clocks convey circadian information to the rest of the organism via nervous and/or endocrine pathways. Melatonin (Mel) secretion by the pineal during the night is under control of the circadian clock. The Mel rhythm is thus an efferent hormonal signal from the clock which can be used as a circadian mediator to any structure than can "read" it. Moreover, the duration of the nocturnal Mel secretion, which is proportional to the length of the night, allows the brain to integrate the photoperiod. Mel appears thus, to convey photic informations that are used for both circadian and seasonal organization.In Mammals, even if the presence of Mel receptors in the suprachiasmatic nucleus of many species indicates an hormonal feed-back on the clock, it was concluded that Mel has a very limited role in circadian organization. The Mel rhythm, however, is only one of the efferent signals of the clock and the little effect of pinealectomy on circadian organization could be explained by the integration of the circadian signal through other clock outputs. This does not preclude an important role for Mel in circadian organization. Indeed, i) subtle desynchrony of several physiological functions have been described after pinealectomy, ii) re-entrainment rate of the activity rhythm is modified in presence or absence of Mel after a phase-shift of the L/D cycle, iii) Mel administration in the SCN induces an increase in the amplitude of clock oscillations. Moreover, through involvement of Mel receptors within the clock, exogenous Mel can be used as a pharmacological tool to manipulate circadian processes (Chronobiotic effect). In rodents, Mel entrains

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free-running circadian rhythms but only when the administration time coincides with the light/dark transition (e.g. onset of activity in nocturnal mammals, offset of activity in diurnal ones) and the phase angle difference depends on the duration of the Mel signal. The molecular mechanisms underlying these effects of exogenous Mel on the clock rhythmicity are not clarified yet. Contrary to what has been described for photic or non-photic cues, clock gene mRNA's are not the initial targets for Mel action on the SCN.

SS-16-03

Sleep deprivation and antidepressant treatment

U. Voderholzer, M. Berger, D. Riemann. *University of Freiburg, Freiburg, Germany*

Acute sleep deprivation (SD) for one night improves mood in about 60% of depressed patients the day after. In this respect, among all types of antidepressant treatments, SD elicits the fastest results. The main limitation, however, is the transient nature of the effect, since the majority of the improved patients experience a relapse after the next night of sleep. A variety of studies focussed on strategies to avoid relapsing and additionally treated the patients with light therapy, lithium, or other drugs. A further strategy has been to advance the sleep period to an unphysiological time. Several studies showed that a phase advance of the sleep period, over a course of either six or three nights consistently stabilized the antidepressant effect of SD in about 60% of those patients who responded positively to SD (1,3). Up to now, only one study also included a control group which participated in a phase delay protocol after SD instead of a phase-advance protocol (2). Significantly more patients relapsed in the phase-delay protocol, supporting the hypothesis that sleeping at certain phases of the circadian rhythm, i.e., especially late in the night and in the morning, has depressogenic effects. The major limitation of the phase advance studies is, that the effect has been shown over a period of one week; further studies have to be included a follow up over the course of four to six weeks.

References

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SS-16-04

Sleep-wake rhythm disturbances in major depression and primary insomnia: A study of sleep microstructure

L. Staner. *FORENAP, Rouffach, France*

Objective: A close relationship between the regulation of sleep and the regulation of mood has been suggested by several studies showing that insomnia and depression are two closely linked clinical entities. In the present study we investigate whether a same or a different mechanism is operating in the sleep onset disturbances of primary insomniacs (PI) and major depressive insomniacs (MDI).

Methods: For this purpose, the time course of EEG power density during the period preceding sleep onset and during the first non REM period was examined in three age and gender matched groups of 10 women and 11 men (MDI, PI and healthy controls - HC).

Results: In contrast to HC and MDI, PI did not experience a gradual decrease of their alpha and beta1 power during the sleep onset period and had a lower delta activity in the 5 minutes preceding sleep onset. Compared to the 2 other groups, MDI exhibit less dynamic changes in slow wave activity during the first non REM period.

Conclusion: The present results suggest that increased wake propensity (Process W) may mainly be implicated in PI whereas a lower sleep pressure (low Process S) is related to MDI. The paper will be discussed in light of ongoing studies on sleep microstructure in 2 different model of sleep disturbances: post-nap sleep and sleep after a transient tryptophan depletion paradigm

SS-16-05

Treatment of seasonal affective disorder

K. V. Danilenko. *Centre for Chronobiology Institute of Internal Medicine, Novosibirsk, Russia*

Bright light is the treatment of choice for winter seasonal affective disorder (SAD). An accepted algorithm is to begin treatment with 10'000 lux fluorescent light for 30 minutes daily upon awakening. Although response usually occurs within a week, treatment should last longer for a stable response, sometimes for the entire winter. Meta-analyses reveal that light has antidepressant action beyond its placebo effect. Dawn simulation is a low intensity form of applying the light signal during sleep. The pathophysiology of SAD is not yet clear, nor is known the mechanism by which light is antidepressant. Beyond direct neurobiological effects (e.g. to rapidly increase serotonin turnover) morning light phase advances circadian rhythms - which tend to be phase delayed in SAD - and the advance is correlated with mood improvement. Regular exposure to outdoor light is also therapeutic. Other treatment modalities have been studied much less than light therapy. Pharmacological approaches (fluoxetine, bupropion, reboxetine, agomelatine, some others) provide clinically equivalent results as light, though not as rapidly and with more side effects. High-density negative air ionisation is surprisingly effective compared to (placebo) low-ion density administration. Physical exercise and behavioural therapy have been the focus of a few positive studies. Sleep deprivation is effective in some SAD patients, similar to the response in non-seasonal depression. Evening melatonin administration is not clearly antidepressant. Tests of a morning carbohydrate-rich diet have also been ineffective, and hypericum, a herb which increases light sensitivity, is an interesting possible adjunct at present under investigation.

Sunday, April 3, 2005

W-06. Workshop: Neuropsychological and neuroanatomical correlates of affective-cognitive interaction in major depression

Chairperson(s): Georg Northoff (Magdeburg, Germany), Heinz Boeker (Zürich, Switzerland)
16.15 - 17.45, Holiday Inn - Room 5

G. Northoff. *Universität Magdeburg Klinik für Psychiatrie, Magdeburg, Germany*

Objective: The symposium aims at demonstrating the neuropsychological and neuroanatomical correlates of abnormal affective-cognitive interaction in depression. Therefore, different studies in functional imaging and neuropsychology investigating

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