dimension, if they document for instance, that vulnerable individuals might develop SAD-like symptoms when they are placed in a light deficient environment, regardless of the time of the year.

ACTIVITY MEASUREMENTS IN SAD

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Reduced psychomotor activity has been shown to be a symptom in SAD from which nearly all patients suffer. Activity measurement by means of wrist actometers provides an easy method to record physical activity objectively.

We recorded continously for at least seven days motor activity on the non-dominant wrist of SAD patients in their normal living conditions (Actometers by ZAK, Kirchdorf a. Inn).

Quantitative analysis of the recordings showed large interindividual differences in the total amount of activity, which can be attributed to primarily different individual motoric levels and different amounts of socially enforced activity in winter.

In addition activity measurement helped us to assess compliance in outdoor patients in therapy studies that include bright light treatment or the performance of physical exercise at fixed time, thus improving the reliability of results.

Giving the temporal structure of activity, actometry is on the other hand a valuable tool to study the chronobiological implications of SAD. As the temporal pattern of rest and activity periods in the actogram allows better estimation on sleep onset, sleep duration and awakening time, actometry gives a reliable measure of hypersomnia, another common symptom in SAD.

Combined with devices that record body temperature actometry provides data which give insight into the changes of the circadian system of SAD patients during depression and bright light therapy. Our data on SAD patients show phase delay as well as phase advance relationships during depression which are normalized after bright light therapy.

INVOLVEMENT OF THE SEROTONERGIC SYSTEM IN THE PATHOPHYSIOLOGY OF SAD AND LIGHT THERAPY

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Based on published reports in the literature there is some evidence that the neurotransmitter serotonin (5-hydroxytryptamine, 5-HT) plays a fundamental role in the pathophysiology of seasonal affective disorder (SAD) and probably also in the mechanisms of action of light therapy (LT). Patients suffering from SAD differ from healthy controls in 5-HT mediated hormonal and behavioral responses to 5-HT challenges. The atypical symptoms, which represent the core syndrome of SAD, have an important relationship to the 5-HT system and selective 5-HT reuptake inhibitors have been shown to be effective for the treatment of SAD. There is some evidence that the mechanism of action of LT depends on serotonergic mechanisms. With the use of the acute tryptophan depletion technique in double-blind, balanced, placebo-controlled crossover designs we investigated the role of 5-HT in SAD and also in the mechanism of action of LT. It emerged that tryptophan depletion induced an exacerbation of the depressive symptomatology in drug-free patients with SAD who were in a stable remission from depression by the usage of LT. No significant effects on mood were observed during the control testing. Preliminary results from a study with untreated depressed SAD patients revealed no significant deterioration of the depressive syndrome after ingestion of the tryptophan-free amino acid drink (tryptophan depletion). Conclusively, the current results from the literature and our own findings support the hypothesis that a 5-HT dysfunction contributes to the pathobiology of SAD and that the functional integrity of the brain 5-HT system is important for the maintainance of the antidepressant effect of LT.

UPDATE ON CLINICAL ASPECTS OF SAD AND LIGHT THERAPY

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In the 11 years since seasonal affective disorder (SAD) was first described and light therapy was first shown to be effective for the depressive symptoms of this condition, much progress has been made in the clinical aspects of SAD and its treatment. Although SAD was initially presented as a homogeneous disorder, evidence is emerging to suggest that premenopausal women with this condition may constitute a specific subgroup. Premenopausal female SAD patients have been found to differ from their male counterparts on certain biological parameters, e.g. low plasma prolactin levels in young women vs. high levels in men. Puberty and the menopause appear to alter the prevalence of the condition in women, whereas the prevalence in men tends to remain more constant across the life cycle. Women with SAD differ from male SAD patients in that they tend to sleep and crave carbohydrates more and gain more weight than men during the winter. Originally, SAD patients were treated with 2500 lux of artificial light. More recent innovations have included: (1) more intense (10,000 lux) light sources; (2) dawn simulators; and (3) portable head-mounted Light Visors. The relative and/or additive merits of these different approaches will be discussed. One recent comparison between fluoxetine and light therapy suggests that these treatments may be equally effective as antidepressants. In addition, they may have additive or synergistic effects if used in combination, a strategy that may reduce side-effects. The mechanism of action of light therapy in SAD is not yet fully understood, although multiple lines of evidence are converging on brain serotonergic systems as a likely site of action. All these areas will be more fully reviewed during the presentation.

THE BIOLOGY OF SEASONAL AFFECTIVE DISORDER: ARE THERE WINTER SUMMER DIFFERENCES?

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Evidence is growing that patients with seasonal affective disorder have marked biological changes during the winter depressive syndrome compared with the summer, and compared with normal groups during the winter. Prolactin and some behavioural responses to m-CPP are increased in SAD and are reduced in summer and after treatment with phototherapy. This may suggest a presynaptic 5HT reduction in association with the syndrome.

Light sensitivity is increased during SAD in winter compared with the same individuals in summer and compared to normal controls in winter. Light sensitivity is reduced in SAD but not in normals by 5HT reuptake inhibition using fluoxetine, suggesting a possible link between 5HT and light sensitivity.

However, non seasonal depressed patients also show seasonal variations between winter and summer in relevant neuroendocrine variables such as cortisol levels and DST. There are also seasonal variations in the normal population. The evidence that circadian and other biological parameters of SAD vary seasonally in relation to the syndrome will be discussed.