were much less than those of the control drug, amitriptyline, at a dose of 75 mg. In three of the ten studies there was evidence that paroxetine could cause slight psychomotor enhancement indicated, for example, by increased threshold on critical flicker fusion test.

In summary; No adverse effects of paroxetine are apparent at the dose of 20 mg./day, although minor impairments can be identified at 40 mg./day. An overview of the data indicates that at the standard therapeutic dose of 20 mg./day, paroxetine has no psychomotor or behavioural toxicity.

SOCIAL FACTORS IN SUICIDE

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Background: The study objective was to investigate the age-related variation of social factors in suicide.

Method: Age-related variation in marital status, living arrangements, activity in working life and social interaction factors were investigated in an entire 12-month suicide population in Finland (N = 1.067); the findings in suicide were compared with appropriate census data.

Results: Several social factors varied across age groups among suicides, with some age-related sex differences. Compared with the general population, the suicides were more commonly never married (especially males aged 30–39 years), divorced, and widowed (especially females aged 60–69 years); living alone was more frequent among the suicides, as was living with parents among male suicides aged 25–39 years. A history of psychiatric hospitalization was especially common among young male suicides who had never married or were living with parents. Living alone was particularly frequent among middle-aged male suicides who had misused alcohol.

Conclusions: While most of the age-related variation in social factors found in suicide seems to parallel the natural variation of these factors in the general population, the results suggest that some social findings in suicide might be related to the victims' psychopathology and excessive alcohol use.

TREATMENT OF MODERATELY OR SEVERELY DEPRESSED PATIENTS WITH NEW ANTIDEPRESSANT MIRTAZAPINE

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Antidepressant treatment is recommended as the first-line therapy for moderately or severely depressed patients. Mirtazapine is a potent antagonist of α_2 adrenoceptors, 5-HT_2 and 5-HT_3 serotonin receptors, while it does not block 5-HT_{1A} receptors. Its antagonism of pre-synaptic α_2 adrenoceptors is the mechanism whereby mirtazapine enhances the release of noradrenaline. The enhanced release of noradrenaline causes stimulation of 5-HT cell firing and 5-HT release through activation of α_1 adrenoceptors on serotonergic soma and/or dendrites. Hence mirtazapine enhances both noradrenergic and serotonergic neurotransmission, and it can be best described as noradrenergic and specific serotonergic antidepressant (NaSSA). This mode of action may be accounted for its high efficacy in the treatment of depressed patients, including severely depressed (17-item HAMD scores at baseline $\geq$ 25). To assess the efficacy of mirtazapine in the treatment of patients with a DSM III diagnosis of a Major Depressive Episode (single or recurrent), an analysis was performed on pooled data from subgroups of moderately or severely depressed patients participating either in the placebo- or amitriptyline-controlled studies of mirtazapine. The patients were stratified according to their total 17-item HAMD scores at baseline: scores of 18–24 were indicative of moderate depression; at least 25 of severe depression. In the subgroup present with moderate depression, significantly larger reduction from baseline were present in the mirtazapine group compared to placebo group ($p \leq 0.01$). Matching results were obtained in the analysis of the severely depressed patients: reductions from baseline during treatment with mirtazapine were statistically and clinically significantly larger than with placebo ($p \leq 0.01$). In pooled data analysis comparing mirtazapine with amitriptyline, equivalent reductions from baseline were found both for the moderately depressed group and severely depressed group of patients. These results demonstrate that mirtazapine is effective in the treatment of both moderately and severely depressed patients.

NEUROENDOCRINOLOGICAL REACTION TO THE TRYPTOPHAN-DEPLETION-TEST IN PATIENTS WITH SEASONAL AFFECTIVE DISORDER WHO RESPONDED TO LIGHT THERAPY

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Some studies describe hormonal dysregulations during episodes of depression, which disappear with remission. Further investigations were able to describe a reduction of brain serotonin activity. Tryptophan-Depletion (TD) induced by ingestion of a tryptophan-free amino acid drink lowers serotonergic function and has been shown to induce symptoms of depression. Therefore we studied hormonal and psychometric reactions to TD in a double-blind placebo-controlled balanced cross-over design in 12 drug-free patients with seasonal affective disorder (SAD). Patients were in stable remission induced by light therapy. Blood samples were obtained one day and 30 minutes before as well as 5 and 7 hours after TD. After TD we found an significant increase in Hamilton Score ($p < 0.01$) and a significant decrease of total ($p < 0.001$) and free tryptophan ($p < 0.001$). During TD and placebo mean plasma concentration of prolactin raised statistically non-significant, while TD was tended to be combined with higher concentrations. Cortisol plasma concentration fell statistically significant during TD (8 a.m.:2 pm $p < 0.05$; 8 a.m.:4 p.m. $p < 0.05$) and tryptophan administration TD (8 a.m.:2 pm $p < 0.005$; 8 a.m.:4 p.m. $p < 0.005$). Concentrations were statistically higher in TD compared to placebo (2 p.m. $p < 0.05$; 4 p.m. $p < 0.001$). Changes of TSH, T3 and T4 were of no clear relation with regard to TD or control testing. Conclusively our results indicate that TD might influence neurohormonal systems as well as the serotonergic system. Moreover during TD we were able to describe a coincidence of depressive symptoms, a decrease in plasma cortisol level and a raise in prolactin concentration.

PROLACTIN SECRETION IN DEPRESSIVE ILLNESS AND HEALTHY CONTROLS AS A RESPONSE TO THE CITALOPRAM-CHALLENGE-TEST (CCT)

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The Citalopram-Challenge-Test (CCT) is one approach to investigate the reagibility of the serotonergic neurotransmitter system, which is thought to be downregulated in depression. Citalopram, a substance inhibiting serotonin reuptake, leads to an immediate secretion of prolactin in normals. Our study is designed to describe differences in prolactin and cortisol to the CCT. 12 patients, meeting criteria vor

https://doi.org/10.1016/0924-9338(96)89083-3 Published online by Cambridge University Press