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Relationship between clonidine test and suicidal behavior

Pitchot, W., Gonzalez Moreno, A., Ansseau, M. and Von Frenckell, R.
Psychiatric Unit, C.H.U. du Sart Tilman, B-4000 Liège, Belgium

Key words: Clonidine test; Depression; Suicidal behavior

The current main neurochemical theories of the biological correlates of suicidal behavior involve serotonergic and to a lesser extent dopaminergic systems (Pitchot et al., 1992). Few data are available about the possible implication of the noradrenergic function. In the present study, we assessed the growth hormone (GH) response to clonidine, a specific α2-adrenergic agonist, in 16 DSM-III-R major depressive inpatients with a history of suicide attempts, compared to 16 age- and gender-matched major depressive inpatients without history of suicidal behavior. Mean GH peak responses to clonidine were significantly lower in he group of suicide attempters than in the control group: 2.88 ± 2.76 ng/ml vs 7.63 ± 7.95 ng/ml ($t = 225$, $df = 1.30$, $p < 0.05$). Therefore, these results suggest that a blunted GH response to clonidine could be a biological correlate of suicidal behavior.

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Growth hormone response to apomorphine test in retarded vs agitated depressed patients

Pitchot, W., Gonzalez Moreno, A., Ansseau, M. and Von Frenckell, R.
Psychiatric Unit, C.H.U. du Sart Tilman, B-4000 Liège, Belgium

Key words: Apomorphine; Dopamine; Psychomotor retardation

Several lines of evidence suggest a role for dopamine in the pathophysiology of depression (Willner, 1985). Data from cerebrospinal fluid studies are consistent with the hypothesis of a relationship between impaired dopamine activity and psychomotor retardation. The aim of the present study was to assess the dopamine function at the postsynaptic level, with the apomorphine test (0.5 mg s.c.), in retarded depressed patients. Twelve inpatients meeting RDC for a retarded major depressive disorder were matched for gender, age, and, in the case of women, menopausal status with 12 RDC agitated depressed patients. Mean growth hormone peak responses to apomorphine were significantly lower in the group of retarded

depressed patients than in the control group: 4.08 ± 2.79 ng/ml vs 17.38 ± 13.57 ng/ml ($t = 3.32$, $df = 1.22$ $p < 0.005$). These results confirm that an impaired dopamine activity in depressed patients is more related to certain aspects of symptomatology, particularly motor retardation, rather than to the diagnosis of depression.

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Abstract withdrawn.

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Chronic treatment with antidepressant drugs enhances the excitatory effect of quinpirole on hippocampal neurons in vitro

Bijak, M.
Polish Academy of Sciences, Institute of Pharmacology, Kraków, Poland

Key words: Quinpirole; Imipramine; Mianserin; Citalopram; D-2 receptor; Hippocampal slices

A growing body of experimental evidence suggests that antidepressive treatments induce an increase in the sensitivity indicate that D-2 rather than D-1 dopamine receptor subtype may underlay the observed increase in the responsiveness to dopaminomimetics. Our earlier studies showed the imipramine-induced sensitization of hippocampal neurons to dopamine (Smialowski and Bijak, 1986, 1987). The present study was carried out to determine whether responsiveness of hippocampal CA1 neurons to the selective D-2 dopamine receptor agonist quinpirole is affected by chronic treatment with antidepressants which have different pharmacological profiles.

The effects of single or repeated treatment with imipramine, mianserin and citalopram (10 mg/kg, p.o.), on the responsiveness of the rat hippocampal neurons to the dopamine D-2 receptor agonist quinpirole (0.1 and 10 μM) were studied. The bath applied quinpirole dose-dependently increased the firing rate of CA1 pyramidal neurons in the slice preparation. That excitatory effect was attenuated by sulpiride (10 μM) which suggests that the quinpirole-induced excitation is due to stimulation of dopamine D-2 receptors in the hippocampus. In hippocampal slices prepared from rats treated with antidepressant drugs repeatedly (twice a day for 14 days), the quinpirole-evoked increase in the neuronal firing rate was significantly potentiated, whereas no change was observed in the reaction to quinpirole after a single dose of the drugs.

It is concluded that prolonged treatment with imipramine, mianserin and citalopram produces an increase in responsiveness of hippocampal CA1 neurons to stimulation of the dopamine D-2 receptor.

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