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Psychoneuroendocrinology 28 (2003) 559–573

www.elsevier.com/locate/psyneuen

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Prolactin response to fenfluramine administration in patients with unipolar and bipolar depression and healthy controls

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Received 16 October 2001; received in revised form 21 March 2002; accepted 24 April 2002

Abstract

The hormonal response to the serotonin releasing agent/uptake inhibitor fenfluramine has been used as an indicator of central serotonin system function. The serotonergic system plays an important role in the etiology and pathogenesis of mood disorders. We compared the prolactin response to fenfluramine administration in unipolar depressed patients (major depressive disorder), depressed patients with bipolar disorder, and healthy controls. We found a trend towards a blunted prolactin response in depressed patients compared to healthy controls, after controlling for sex, family history, family history-by-gender interaction, and baseline levels. There was no significant difference between unipolar and bipolar patients in the baseline prolactin levels or the response to the fenfluramine administration. We also found a negative correlation between aggression and impulsivity scores and prolactin responses in subgroup with unipolar but not bipolar depression. Female patients with unipolar depression who had first-degree relatives with unipolar depression and normal controls had significantly higher prolactin responses than female patients with unipolar depression who did not have first-degree relatives with unipolar depression. The lack of difference in the response to fenfluramine administration between unipolar and bipolar depressed patients may indicate that overall serotonergic function in unipolar and bipolar depressed patients is similarly impaired.

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Keywords: Major depressive disorder; Bipolar disorder; Fenfluramine; Prolactin; Serotonin

1. Introduction

Multiple lines of evidence suggest that the serotonergic system plays an important role in the etiology and pathogenesis of mood disorders (Brown et al., 1994; Leonard, 1994; Mann, 1999; Dubovsky and Ruzan, 1999; Oquendo and Mann, 2000). Indirect evidence of a relationship between serotonergic dysfunction and depression comes from the antidepressant efficacy of selective reuptake inhibitors (SSRIs) (Åsberg et al., 1986; Kaplan and Sadock, 1998) and reversal of antidepressant response by tryptophan depletion (Neumeister et al., 1998; Delgado et al., 1990). More direct evidence comes from studies of the neurobiology of mood disorders. Some studies found lower cerebrospinal fluid (CSF) 5-hydroxyindolacetic (5-HIAA) levels in depressed patients (Åsberg et al., 1984), a blunted neuroendocrine response to serotonin agonists (Heninger et al., 1984; Siever et al., 1984; O’Keane and Dinan, 1991; Mann et al., 1995), lower platelet serotonin uptake (Tuomisto et al., 1979; Kaplan and Mann, 1982), and an association between depressive disorders and genes related to the serotonergic system (Mann et al., 2001; Sher, 2001; Steimer et al., 2001). CSF (Dencker et al., 1966; Coppen et al., 1972; Mendels et al., 1972; Banki, 1977; Roos and Sjöström, 1969; Vestergaard et al., 1978; Koslow et al., 1983), postmortem (Young et al., 1994), platelet (Meagher et al., 1990; Marazziti et al., 1991; Lewis and McChesney, 1985; Ellis et al., 1991), neuroendocrine challenge (Thakore et al., 1996; Yatham et al., 1999; Meltzer et al., 1984), and genetic (Potash and DePaulo, 2000; Kato, 2001; Craddock and Jones, 2001) studies provide some evidence that the serotonergic system is also involved in mania. Prange et al. (1974) formulated a permissive hypothesis of serotonin function in bipolar disorder. They suggested that a deficit in central serotonergic neurotransmission permits the expression of bipolar disorder, and that both the manic and depressive phases of bipolar disorder are characterized by low central serotonergic neurotransmission. Prange et al.’s (1974) hypothesis is supported by the fact that, with the exception of one study (Ashcroft et al., 1966), all studies found that CSF 5-HIAA levels in patients with mania were not different from depressed patients (Coppen et al., 1972; Mendels et al., 1972; Banki, 1977; Bowers et al., 1969; Goodwin et al., 1973; Sjöström and Roos, 1972; Gerner et al., 1984; Ashcroft and Glen, 1974; Roos and Sjöström, 1969; Koslow et al., 1983; Swann et al., 1994). Evidence has been mounting that there are abnormalities in serotonergic function in both unipolar and bipolar depression (Brown et al., 1994; Leonard, 1994; Mann, 1999; Dubovsky and Ruzan, 1999; Oquendo and Mann, 2000) and that these abnormalities are similar in the two disorders (Koslow et al., 1983; Mitchell et al., 1990).

The hormonal response to the serotonin (5-HT) releasing agent/uptake inhibitor fenfluramine has been widely used as an indicator of central 5-HT system function in humans (Siever et al., 1984; McBride et al., 1989; Coccaro and Kavoussi, 1994; Coccaro et al., 1994; Halperin et al., 1997; Mann, 1999). The release of 5-HT from

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