

OXYTOCIN, VASOPRESSIN AND ANXIETY **IN MAJOR DEPRESSION:** **AGO-ANTAGONIST NEUROHORMONES**

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BACKGROUND

Oxytocin (OT) and vasopressin (AVP) are very similar neurohypophyseal peptides which could be involved in mood disorders. They act as neuromodulators of the stress response. Besides its action at the periphery and at the hypothalamo-hypophyseal level, AVP exerts a positive effect on attention, immediate memory, learning and recognition. OT exerts a general amnesic influence on explicit memory. While AVP is known as an ACTH stimulating factor synergistic to CRF, it is likely that endogenous oxytoninergic system, which can be activated by physiological and/or pharmacological manipulation, can "buffer" the stress activated vasopressin-ACTH-cortisol action (Legros, 2001; Heinrichs et al., 2003). Intracerebral OT inhibits the stress-induced activity of the hypothalamic-pituitary adrenal (HPA) axis responsiveness suggesting an inhibitory influence of OT on stress-responsive neurohormonal system (Heinrichs et al, 2003). AVP seems to play an important role in the HPA dysregulation in depressive illness. The recruitment of AVP as the primary regulator of the HPA axis in chronic stress conditions may explain the hypercortisolaemia that is demonstrated in depressed subjects. The aim of this study is to assess a possible relationship between anxiety, OT, AVP and their neurophysins plasma levels among depressed patients.

METHOD

The study was conducted among 22 major depressive patients. The diagnosis of major depressive disorder was based on the DSM-IV criteria and the Mini International Diagnosis Interview. Patients had a score of at least 17 on the 17-items Hamilton Depression Rating Scale (HDRS). Blood samples for assessment of OT, AVP, OT- and AVP-neurophysins were collected and measured as previously described. Anxiety was evaluated by using the Spielberger State-Anxiety Inventory (STAI-A).

RESULTS

The mean score of STAI-A was $60,28 \pm 10,74$. Results showed a significant negative correlation between OT and anxiety ($r = -0,61$, $p = 0,005$) and a significant positive correlation between AVP-neurophysins and anxiety ($r = 0,41$, $P = 0,04$).

DISCUSSION

The main finding of the present study is a negative association between anxiety and OT and a positive association between AVP-neurophysins and anxiety in depressed patients. It supports the importance of the neurohypophysis in mechanism underlying anxiolysis. Since the first evidence that OT, the “sister” neurohormone of AVP, can share and have opposite effects to those of AVP on cognitive functions, it has been suggested “agonist-antagonist” actions of the two hormones. OT actions are all directed towards maintenance of the social group (fetal expulsion, milking let down, sexual behaviour), whereas AVP actions are all directed towards protecting homeostasis of the individual (water retention, blood pressure regulation, increased arousal and memory). An integrative ago-antagonist, or “ying-yang”, function was postulated by Legros (2001). Present knowledge about central AVP functions in animals is consistent with the assumption that impaired AVP transmission in the central nervous system could produce at least some symptoms of the depressive state. OT is known to have anti-nociceptive and analgesic effects, as well as anxiolytic and sedative properties. The precise involvement of OT and AVP in the pathophysiology of depression remains to be elucidated.

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