Journal of Autism and Developmental Disorders, Vol. 22, No. 3, 1992

Letters to the Editor

DOES OXYTOCIN DEFICIENCY MEDIATE SOCIAL DEFICITS IN AUTISM?

We would like to suggest a possible new mechanism to account for social deficits in at least some cases of infantile autism. Kanner first suggested that autistic individuals suffer an inborn disturbance of affective contact, and some recent theories have returned to this view. Identification of imbalanced neuroendocrine functions that contribute to this disturbance of affective contact might lead to treatments to ameliorate not only social relating but also cognitive functions dependent on social awareness. Modahl (1992) suggested that a deficit in the social stimulating hormone oxytocin or its evolutionary precursor vasotocin may underlie some disorders of childhood attachment.

Oxytocin is a behaviorally active, physically and emotionally arousing peptide hormone produced primarily in the supraoptic nerves of the hypothalamus. It is released into the brain and blood during social, reproductive, and other activities (Insel, in press). There is a growing body of evidence that oxytocin promotes emotional attachment behavior across the wide spectrum of activities in which it is involved (Insel, in press). Newton (1973) first hypothesized that oxytocin is a primary general hormone which mediates interpersonal attachment to facilitate success of family life. Higher oxytocin blood levels have been correlated with stronger human mother–infant bonding and with more social and outgoing personalities in men and nonpregnant women, and supportive psychotherapy has also been found to raise human oxytocin (K. Uvnas-Moberg, personal communication, 1989).

Although no studies exist as yet on oxytocin levels in autism, there is indirect evidence to support the hypothesis that such a causal link exists. First, the opiate hormones beta-endorphin and dynorphin have been found to inhibit oxytocin action (Bicknell, Leng, Lincoln, & Russell, 1988). Beta-endorphin has been found elevated in studies of autistic children who exhibit self-injurious behavior (Sandman et al., 1990), and high opiate levels have been suggested on cogent theoretical grounds to relate to autistic social behavior, self-stimulation, and variability high pain thresholds (Sahley & Panksepp, 1987). In part, the hypothesized elevated beta-endorphin level may be suppressing the action of oxytocin.

0162-3257/92/0900-0449$06.50/0

Furthermore, the opiate blockers naltrexone and nalaxone, which raise oxytocin in humans, have been found by some, although not all, investigators to have beneficial effects on self-injury and social withdrawal in autistic children (Sandman et al., 1990). Perhaps these opiate blockers have some of their behavioral effects through the facilitation of oxytocin action.

Another treatment sometimes found helpful for autistic children is a B6-magnesium combination (Rimland, 1988). Kawarabayashi et al. (1990) found that magnesium potentiates the excitatory effect of oxytocin.

Anatomically, the limbic system has been a focus of theory and investigation in autism because of its role in social behavior and motivation (Bauman & Kemper, 1985). The greatest concentration of oxytocin receptors are found in limbic nuclei in the medial temporal lobe (Insel, in press).

Chamberlain and Herman (1990) proposed a comprehensive biochemical theory of autism that would be consistent with an oxytocin deficit. They suggested that hypersecretion of melatonin might reduce hypothalamic corticotrophin-releasing hormone causing reduction in pituitary beta-endorphin. Low pituitary endorphins might then feed back to cause elevation of hypothalamic beta-endorphin. This theory is consistent with an oxytocin deficit because elevated hypothalamic beta-endorphin would repress oxytocin function. Moreover, low oxytocin might feed back to promote further melatonin hypersecretion.

An oxytocin deficit theory of autistic social impairment is appealing because of its consistency with existing theories and neurobiological findings, with the actions of the most accepted current medications, and with behavioral findings in animals. It is clear, however, that oxytocin interactions are quite complex. Abnormalities could exist in oxytocin levels, in receptor number, distribution, or function, or in an area downstream of the direct oxytocin effects. It also seems likely that autistic symptomatology represents a final pathway for multiple etiologies, of which oxytocin deficit may be one, and which may result in somewhat different clinical presentations. These different presentations may ultimately be understood well enough to mark likely etiologies and potential treatments. We suggest, therefore, that investigations into this and related hypotheses focus not only on biochemical measurements but also on behavioral and cognitive correlates of biochemical abnormalities.

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CORRECTION TO PREVIOUS EVALUATION OF FACILITATED COMMUNICATION

Since publication of my personal evaluation of Facilitated Communication (Bettison, 1991), I have had some communication both with Rosemary Crossley of the DEAL Centre in Melbourne, Victoria, and with Annie McDonald. While their reaction to my comments was generally positive, McDonald was most unhappy with my references to her. She has asked me to bring the following information, not originally available to me, to the attention of JADD readers.