

Brain Opioids and Autism: An Updated Analysis of Possible Linkages¹

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Considerable clinical evidence suggests that autistic children lack the normal ability or desire to engage others socially, as indicated by their poor social skills and inappropriate use of language for communicative purposes. Specifically, these children seem to lack normal amounts of social-emotional interest in other people, leading perhaps to a decreased initiative to communicate. This paper summarizes experimental evidence supporting a neurological theory, which posits that autism, at least partially, represents a disruptive overactivation of hypersensitization of neurohormone systems in the brain, such as brain opioids. These substances modulate social-emotional processes, and the possibility that blockade of opioid activity in the brain may be therapeutic for early childhood autism is discussed.

INTRODUCTION

Accumulating evidence suggests that early childhood autism has a biological etiology. The disturbances appear to originate during gestation, are present at birth, and become manifest when they interfere with the normal course of development (Ciaranello, Vandenberg, & Anders, 1982; Pig-gott, 1979; Rutter & Schopler, 1978). No single anatomical or biochemical lesion has yet been demonstrated to account for autistic behavior, although

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modest success in symptom reduction has been achieved following the administration of antipsychotic drugs (e.g., Campbell, Cohen, & Anderson, 1981), the anorexigen fenfluramine (August, Raz, & Baird, 1985; Ritvo, Freeman, Geller, & Yuwiler, 1983), and pyridoxine (Lelord, Muh, Barthelemy, Martineau, Garreau, & Calloway, 1981; Rimland, Callaway, & Dreyfus, 1978). The general consensus is that future progress in the search for potential etiologies and treatment of autism will depend upon clarification of the neurochemical basis of this perplexing developmental disorder.

The aim here is to discuss the possible role of abnormal brain opioid activity in the genesis of autistic symptoms (Kalat, 1978; Panksepp, 1979; Panksepp, Herman, & Vilberg, 1978)—an idea supported (1) by a growing body of evidence indicating that autisticlike symptoms can be induced in animals with the administration of exogenous opioids (Panksepp, 1981), (2) by the apparent relationship between symptoms of opiate addiction and autistic symptoms (Kalat, 1978), and (3) by the fact that autisticlike symptoms in the severely mentally retarded can be attenuated by opioid blockade (Sandman et al., 1983). To clarify the conceptual basis of this neurochemical hypothesis, we would initially couch it within the context of knowledge concerning autism and delayed development of social signaling systems such as language.

Language Deficits in Autism

Even in the highest-functioning autistic individuals, problems in social interaction are the major obstacle to communicative competence (Akerly, 1974; Schopler & Mesibov, 1985). With respect to language development, autistic children exhibit a limited repertoire of communicative abilities (Wetherby & Prutting, 1984), although they may possess adequate linguistic ability (Wollner, 1983). The desire or intent to communicate in young children is displayed largely through preverbal gestures, and autistic children generally fail to use such gestures for communicative purposes (Kanner, 1943; Rutter, 1978). Such children seem to lack the initiative to communicate (Maurer & Damasio, 1982). For example, they fail to smile or point as if to show (Bartak, Rutter, & Cox, 1975; Curcio, 1978), they fail to maintain eye contact, and they fail to orient toward others. During infancy, they fail to show appropriate anticipatory behaviors when picked up (Rimland, 1964). The communication of the autistic child appears to serve primarily instrumental or regulatory functions, related to the child's immediate biological needs. Autistic children may engage other people as "object tools" in order to obtain environmental ends, and they seem to exhibit a pervading developmental delay in their communicative ability to achieve social ends and, hence, to share feelings and knowledge. Thus, they fail to learn much from others (Wether-