A Case Report of Naltrexone Treatment of Self-Injury and Social Withdrawal in Autism

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The endogenous opiate release theory of self-injurious behavior (SIB) was investigated through double-blind placebo-controlled administration of naltrexone hydrochloride (Trexan®) to a 14-year-old autistic and mentally retarded male for treatment of severe SIB. Results yielded a marked decrease in SIB during two phases of active drug treatment, though SIB did not revert to originally observed placebo levels during a second placebo phase. An increase in social relatedness also was observed during phases of active drug treatment. Opiate theories of self-injury and the possible interrelationship of self-injury with pituitary-adrenal arousal and with social relatedness are discussed.

Self-injurious behavior (SIB) is one of the most perplexing and serious forms of psychopathology exhibited by developmentally disabled patients. Estimates of its prevalence range from 22% of the institutionalized developmentally disabled population to 11% of developmentally disabled individuals in the community (Hill, Balow, & Bruininks, 1985) to 40% of autistic individuals (Bernstein, Hughes, Mitchell, & Thompson, 1987). The risk of permanently disabling and/or life-threatening injury is high.

The mechanisms by which self-injury is developed and maintained are not well understood, producing difficulty in defining efficacious treatment

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Operant behavioral explanations have been advanced by some authors (cf. Carr, 1977) and certainly behavioral treatment, including the use of both reinforcement and aversive techniques, has been effective with a proportion of the population (cf. Barrett, 1986). Psychotropic medications have been used with success with certain patients, but potential untoward effects as well as the durability of reduction in self-injury have been questioned (cf. Singh & Millichamp, 1985). An additional complicating factor is that although specific disorders involving self-injurious behavior (i.e., Lesch-Nyhan syndrome) have clearly identifiable characteristics as well as biochemical bases, for the most part, self-injurers appear to be a heterogeneous and ill-defined group. Efforts to more carefully delineate potential biological underpinnings have led to investigation of neurochemical processes involved in this behavior, with the hope that knowledge about these processes can guide treatment efforts.

Correspondingly, disorders in the endogenous opiate system have been postulated to play a role in the maintenance of SIB for certain autistic and mentally retarded patients (Deutsch, 1986). Theories about the mechanisms of these opiate effects cluster into three categories. The first, termed the down-regulation hypothesis, involves the notion of a disturbed perceptual system and refers to the possibility that an excess of endogenous opiates results in the absence or dampening of nociception, such that self-injury is not experienced as painful (Sandman et al., 1983). The second theory, termed the pituitary-adrenal hypothesis, maintains that SIB operates within a negative reinforcement paradigm and suggests that arousal of the pituitary-adrenal system due to the stress of novelty, uncertainty, or conflict leads to self-injury which then functions to reduce physiologic arousal through production of endogenous opiates (Cataldo & Harris, 1982). This notion is corollary to the third hypothesis, termed the opiate-addiction hypothesis, that self-injury leads to the release of endogenous opiates with positively reinforcing sensory consequences (i.e., a narcotic effect), resulting in the development of tolerance and dependence (Richardson & Zaleski, 1983; Sandman et al., 1983).

If such hypotheses are accurate, treatment with an opiate antagonist may act to ameliorate SIB through blockade of opiate receptors. Several treatment studies have been published, with results alternately supportive (e.g., Barrett, Feinstein, & Hole, 1989; Bernstein et al., 1987; Herman et al., 1987; Richardson & Zaleski, 1983; Sandyk, 1985; Sandman et al., 1983) and discouraging (e.g., Szymanski, Kedesky, Sulkes, Cutler, & Stevens-Ours, 1987; Beckwith, Couk, & Schumaker, 1986; Davidson, Kleene, Carroll, & Rockowitz, 1983) of this approach. These equivocal findings may be the result of differences stemming from the drug itself, such as dose effect or variations in duration (half-life) of drug effect depending on the type of