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VARIOUS ENDOGENOUS OPIOIDS AND AUTISTIC BEHAVIOR: A RESPONSE TO GILLBERG

Endorphins, enkephalins (and dynorphins) refer to different opioid systems, derived from distinct precursor molecules and possessing unique receptor affinity profiles. The endorphins are derived from pro-opiomelanocortin (POMC) and consist of a family of fragments, beta, alpha, and gamma, characterized by different molecular weights. Of these, β-endorphin is the largest (31 amino acids) and in its unmodified (natural) form has the strongest affinity for the mu receptor and possesses analgesic properties. The enkephalins are derived from proenkephalin and are smaller molecules than the endorphins. Although derived from different precursors, there are structural similarities among the opioids. For instance, Leu-enkephalin also is contained in the structure of prodynorphin and Met-enkephalin is embedded in the β-endorphin fragment of POMC.

Gilberg, Terenius, and Lönnerhelm (1985), in their important study, stated in their discussion that there was a significant association between decreased sensitivity to pain and elevated endorphin in psychotic children (p. 783). However, they indicated earlier in the paper that the opioid they were measuring, termed Fraction II, was derived from proenkephalin A and that Met-enkephalin-Lys was the *major opioid* in this fraction. Thus, although they referred to Fragment II as "endorphin," it is a member of the enkephalin family. We agree with Gillberg that there are many endogenous opiates whose label and function are not equivalent. We apologize for our contribution to the confusion.

It is interesting that Gillberg reports lower BE in CSF of autistic patients in his recent preliminary findings. Ross, Klykylo, and Hitzemann (1987) reported higher levels of CSF BE in baseline measures of autistic children compared with control samples, and Coid, Allolio, and Rees (1983) reported higher Met-enk-"like" activity in plasma of self-injuring adult borderline patients. The methodological disparities evident among studies in Table I suggest more empirical work is needed and that conclusions are premature about the significance of endogenous *opioid* levels in autistic and self-injurious behavior.

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Table I. Published Studies of Opiate Levels in SIB and Autism

Study	Fluid	Target group	Opiate	Results
Coid et al., 1983	Plasma	Borderline SIB (n = 10)	Met-enk end-like	Elevated in SIB
Weizman et al., 1984	Plasma	Autistic (n = 10)	Humoral endorphin	Not Different
Gillberg et al., 1985	CSF	Autistic (n = 20)	Fractions of Pro-enk	Elevated in SIB
Ross et al., 1987	CSF	Autistic (n = 9)	β-endorphin	Elevated
Sandman et al., 1990	Plasma	Autistic SIB (n = 40)	β-endorphin	Elevated compared to patients
Sandman et al., 1991	Plasma	Autistic SIB (n = 8)	β-endorphin	Elevated compared to patients; decreased compared to controls

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