REFERENCES


NALTREXONE IN INFANTILE AUTISM

A long history of unsubstantiated claims in infantile autism makes it essential that new therapies be evaluated rigorously. This is particularly important when the therapy may ultimately require long-term pharmacological treatment, as in the proposal that the opiate antagonist naltrexone be used to treat autism. The report of Leboyer et al. (1992) of a “beneficial clinical action” of naltrexone in a double-blind study is therefore of great interest, not least because they reported positive statistical evidence despite exceedingly low power due to a small group. Unfortunately, their conclusions are rendered doubtful as a result of a questionable method of analysis.
The sample of four autistic children was selected on behavioral criteria with particular emphasis on symptoms theoretically relevant to the use of naltrexone. However, the data of Child 4 were subsequently excluded from the statistical analysis on the basis of an inspection of the results (this child "exhibited neither biochemical abnormalities nor a clear clinical response"). Such post hoc data selection creates a difference between the groups and makes the statistical tests meaningless. To illustrate this point, one may ask whether this child's data would have been excluded had his clinical response been more positive.

An additional problem arises with regard to the conclusions of this experiment relating to plasma β-endorphin levels. Even after the elimination of the "normal plasma neurohormone values" of Child 4, it was still not possible to demonstrate a statistical difference among the conditions for β-endorphin plasma levels at the conventional 5% level of statistical significance for the remaining three children. Nevertheless, their plasma levels were described as "substantially elevated" during baseline and placebo periods, and "normalized" by naltrexone treatment, claims repeated as conclusions of the experiment, together with the statement that "our data affirm the existence of an endogenous opioid disorder." However, the analysis provided is inadequate to support such a conclusion. It is also unfortunate that Leboyer et al. did not provide either individual values for the three cases, or a measure of variability about the mean.

Accordingly, the report of Leboyer et al. does not constitute the "compelling evidence for the therapeutic effect of naltrexone" sought by these authors. While their data are suggestive, the small group makes statistical analysis unpromising, a situation that cannot be improved by post hoc data selection. It is also worth noting the authors' statement that "on the basis of other ongoing work, we believe naltrexone benefits most autistic children and therapeutic effects appear to last indefinitely." It would be unfortunate if this assertion, as yet undocumented, were to prematurely encourage the use of naltrexone as a therapy for infantile autism.

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