HYPOTHESES

*Severe iron deficiency in early life during the period of rapid dopamine system differentiation, (PND4-21) impairs monoamine system functioning in the forebrain leading to attentional deficits similar to those observed in ADHD

*Previous work from our laboratory clearly illustrates that gestational and lactational (i.e. preweaning) iron deficiency in rodents alter dopamine transporter and receptor levels in regions that are involved in attentional functioning and that deficits in the dopamine system and in behavior persist despite iron repletion (Beard et al., 2003).

RESULTS

Conclusions

*Our results showed that ID rats performed more poorly than controls in all within-dimension aspects of attentional set-shift testing. The differences were not as pronounced, however, when the dimension was shifted.

*MPH at 1mg/kg and 5mg/kg significantly improved ID and control rats' performance. There was a tendency in this group, however, for 1mg/kg to be more effective in improving performance in ID rats, compared to 5 and 10 mg/kg doses. In both groups, 10 mg/kg appeared to be suboptimal in improving performance, indicating possible interfering behaviors at this dose.

*These data support our hypothesis that early iron deficiency produces persistent effects on attention and perhaps underlying dopamine neurobiology. Moreover, we are the first group to show that methylphenidate can improve attentional processes in early iron deficient animals.

REFERENCES:


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