INHIBITORY CONTROL AT 34 MONTHS OF COCAINE-EXPOSED NEONATES

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Cerebral damage sustained by children or adults that involves the limbic-striatal-prefrontal axis has been associated with inhibitory control (IC) deficits on neurobehavioral testing. Such as association has not been reported after neonatal damage. We hypothesize that neurotoxic effects of prenatal exposure to cocaine can result in atypical development of this pathway and hence could play a role in the evolution of IC deficits. Thus, cocaine-exposed children should have difficulty in their ability to control, plan, and execute intentional motor acts.

To test this hypothesis, we evaluated cognitive development and IC in two groups of healthy term infants when they were 34 months old. One group (n = 21) was exposed to cocaine prenatally as determined by maternal report, maternal or infant urine toxicology, or infant meconium toxicology. The other group (n = 52) was not exposed to cocaine or any other controlled substance. Griffiths Mental Development Scales were used to evaluate overall cognitive competence, as well as individual profiles of ability. A Rapid Sequential Alteration Naming Task (RSANT) and a Graphomotor task (GMT) were specifically designed to evaluate IC at this age. The RSANT consisted of 2 conditions. The first (Board A) presented a randomly alternating series of pictures of a cup and a fish, while the second (Board B) presented the same random sequence with additional extraneous stimuli (bubbles) overlayed on the figure. Both completion time and number of errors were scored. The GMT consisted of 4 condiitons of increasing difficulty of line and dot drawing and tracing that evaluated perseverative behavior.

We found that cocaine-exposed children, although within the normal range, had lowered scores on all Griffiths Scales except the Personal-Social Scale. Performance on RSANT (Board A) was marked by errors and slowed rate, even when scores on the Speech and Language Scale were used as covariates. Moreover, unlike RSANT findings with children with hypoxic-based neonatal brain injury, cocaine-exposed children performed better when an increased amount of information was present (Board B). This enhanced performance with increased stimulation is particularly interesting because it is consistent with findings of stimulus-seeking behavior on attention tasks during infancy. On the GMT, the cocaine-exposed children were similar to the noncocaine-exposed children in their capability to perform but they showed an increase in the number of perseverative errors as early as the second level of difficulty, which remained even when scores on the Eye-Hand Coordination and Performance Scales of the Griffiths Scales were used as covariates.

These findings support the hypothesis that prenatal cocaine exposure alters IC, a finding that might implicate some form of deviation in development of the limbic-striatal-cortical axis. Thus, neonatal cocaine exposure may play a significant role in neuropsychological development related to language, information processing speed, and motor integration including verbal and manual IC deficits.

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