Significance of Prenatal Nicotine Exposure on Electrophysiological Markers of Sensory Processing and Attention in Infants

Nicotine is a teratogen known to cause many developmental deficits including low birth weight and premature birth. Prenatal nicotine exposure (PNE) is associated with aberrant brain function such as hyperactivity, decreased attention, impaired auditory processing, and decreased cognitive ability. However, there is a lack of evidence that PNE is the main cause of these deficits. Further, the influence of nicotine exposure on the specific neural mechanisms that alter brain function remains mostly unknown. The current study investigated infants of mothers who smoked tobacco during pregnancy. Prefrontal cortical activity was measured, using electroencephalograms (EEGs), during the presentation of auditory stimuli, with a particular focus on the infants’ cortical response to novel stimuli. PNE infants showed increased Event-Related Potential (ERP) responses to all stimuli compared to controls who demonstrated habituation to standard tones. Cognitive and communication development scores (measured with Bayley Scales of Infant Development) did not correlate with Mismatch Negativity (MMN), N200, or P300 ERP complexes. The results are discussed in the context of animal models and implications for advancing research on attention deficit and hyperactivity disorder.

Keywords: prenatal nicotine exposure; auditory processing; EEG; MMN; P300; N200; ADHD