

Summary: Perinatal Pitocin as an Early ADHD Biomarker: Neurodevelopmental Risk?

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For over 30 years I have been working as a psychotherapist serving children and families. During the course of the past ten years, I began tracking a clinical pattern that seemed to be more than coincidence. This pattern surfaced during my routine interviews of mothers while reviewing their children's developmental histories. In particular, children who sought treatment for ADHD bore an all too common perinatal history; that of having been exposed to a well-respected and universally utilized obstetric agent, Pitocin. In addition, mothers of many of these children reported similar histories of a longer than average maternal gestation, an extended maternal labor, and a heavy birth weight in these offspring. As this early developmental pattern among ADHD children grew more typical, the need for scientific exploration of this trend could no longer be ignored, and this pilot study was launched. To our knowledge, this study marks the first known attempt to identify a signature etiological mechanism associated with subsequent ADHD onset in children.

To date, the precise etiology of Attention Deficit Hyperactivity Disorder (ADHD) remains a mystery. The steadily rising incidence of childhood ADHD and the coincidental increase of Pitocin (exogenous Oxytocin) usage throughout the U.S. to expedite labor/delivery underscored the rationale to investigate the relationship between these two factors. Specifically, it was proposed that perinatal exposure to obstetric Pitocin risks ADHD onset in children. The specific mechanism of this impact was unclear, but proposed as being neurobiological in nature.

To test this theory, twenty-one variables were analyzed as potential ADHD predictors: select obstetric complications, a confirmed ADHD diagnosis and history of a first degree ADHD relative. The medical labor and delivery records of a heterogeneous sample of 172 ADHD and non-affected children, including siblings of children diagnosed with ADHD (ages 3-25), from throughout the U.S. and Canada were gathered from various agencies and independently reviewed by trained raters for select obstetric markers and presence/non-presence of ADHD.

A PLUM ordinal regression, chi-square, independent samples t-test and step-wise multiple regression comparatively analyzed and determined the predictive relationship(s) of obstetric factors to subsequent ADHD onset. In support of this hypothesis, results revealed fetal exposure to Pitocin (via maternal induction/augmentation) was *the chief ADHD predictor* ($p < .001$) (**67.1%**). Pitocin exposure time, labor length and maternal gestation length also emerged as ADHD predictors. Noted trends throughout the archival files included maternal epidurals, AROM (assisted rupture of membranes), post-birth oxygen supplementation, nuchal cord events and heavier newborn weight (>7lbs). Familial history (i.e. first degree relative with a diagnosis of ADHD) played no predictive role in this analysis, which came as a surprise.

These findings suggest a Pitocin-linked, interactive constellation of factors initiates a neuro-developmental cascade that disrupts cognitive executive functioning, promoting ADHD.

We speculate a host of overlapping mechanisms that may underlie this outcome. These include: Restricted neural oxygen flow and/or restricted blood flow to the fetus; insult to soft neural tissue via prolonged, Pitocin-induced hypertonic uterine contractions (whereby this pressured uterine force may impose neural convolutions or architectural imprints on the immature fetal brain, altering cortical topography, and adversely affecting long-term neural development); and initial whetting of the neural appetite to a chemical stimulant, which may foster a “neurodevelopmental storm.” Other contributing dynamics may include the down-regulation of fetal (hormonal) oxytocin via obstetric Pitocin infusion, which could trigger a switch in inhibitory neurotransmitter (GABA) signaling in the fetal brain, risking hypoxia and neuronal cell death. Blood-brain barrier crossover issues may also impact this outcome. Finally, given the broad body of literature that exists on family genetic predisposition and ADHD, we believe an *epigenetic* dynamic may play a significant role, whereby some mechanism inherent in this activity could effectively trigger the genetic underpinnings of ADHD for certain individuals, subsequently altering their developmental trajectory. Furthermore, given the well-established male proclivity to develop this disorder (three times greater than females), we believe gender may also have a bearing, such that females may possess some inherent toxins insulation that protects them from any adverse perinatal dynamics males may be more vulnerable to.

As concerns of genesis peak around other neurodevelopmental disorders (i.e. Autism and other ASD [autism spectrum disorders]) these results may provide for a fresh etiological conceptualization of these important issues. Earlier detection of ADHD via perinatal markers and intervention in high-risk perinatal circumstances is implied within the clinical arena. In viewing ADHD as a disorder potentially likened to neural insult, clinical protocols may effectively employ multi-modal treatment approaches of cognitive behavioral skills, appropriate pharmacotherapy and specific strategies aimed at strengthening and maximizing cognitive executive functioning in ADHD.

It is concluded that Pitocin induced labor may carries iatrogenic fetal risks. Although Pitocin use during labor may not be the sole litmus test for ADHD onset, our results suggest it may play a key role in its outcome. These findings respectfully suggest reconsideration of current obstetric practices toward more careful assessment of Pitocin’s impact and enhanced protocols to manage its use among autonomous birthing facilities. In summary, the potential risks of prolonged fetal exposure to obstetric Pitocin should be carefully explored, as should the precise neurobiological mechanism(s) and pathophysiology possibly involved. Replication of this research via an expanded sample is urgently warranted to retest this hypothesis.

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Author Disclaimer: The views in this article are of scientific nature only, with the sole intention to advance the progress of research in ADHD. No liability can be taken for any consequences potentially arising from claims made in or in relation to this article. No grants have yet been accepted supporting this work.

Research Presentations:

This research was recently presented as a Scientific Poster at APA Convention (Toronto, Canada, August, 2009); International Hershey Conference on Developmental Brain Injury (Snowbird, Utah, June, 2010); International ChADD Conference (Atlanta, Georgia, November, 2010) and as a Symposium at International OTIS Conference (San Diego, California, June, 2011).

Drs. Kurth and Haussmann are available to speak about this topic upon request.

Partial List of References

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