Name: Gbadebo Ogungbade
PI Name: Larry Holmes Jr

Email: debogungbade@gmail.com
PI Email: drlholmesjr@gmail.com

Pediatric Chronic Oxycodone and CYP2D6 Aberrant Epigenomic Modulations in Autistic Spectrum Disorders (ASD): Quantitative Evidence Synthesis (QES)

Laurens Holmes, Jr
Gbadebo O. Ogungbade

Global Health Services Initiatives Inc

Background: Autistic Spectrum Disorder (ASD) remains unimaginable with respect to causation, while brain/CNS biodegradation is indicative of ASD pathogenesis. Regarding hepatic function that facilitates the application of Cytochrome P450, the associated gene CYP2D6 that facilitates metabolism remains causal in ASD.

Rationale/Significance: ASD among children is treatable but incurable. While ASD remains genetic, environmental involvement in risk tends to increase ASD indicative of aberrant epigenomic modulation (AEM).

Hypothesis: The Cytochromes P450 and CPY2D6 affiliated with this process down-regulates the metabolic process and increased ASD causation. This assessment requires the identification of the Cyt P450 and the gene involved in metabolic process in ASD. Systematic review and QES as well as Dersirmonian – Laird as common effect size (CES) and precision measure, 95% CI were utilized.

Results: Seven studies were utilized in this QES. The sample size was 198, White, 102, and B/AA 68, and Other race, 28. The CPY2D6 gene in ASD association, CES = 3.10, 95% CI =2.68-4.36. This CPY2D6 among B/AA with ASD was higher, CES, 4.16, 95% CI = 2.78-9.66, relative to Whites, CES, 2.01, 95% CI = 1.96-4.95.

Discussion: The ASD remains unimaginable due to several risk determinants. The understanding of how environmental differential, as substance use (Oxycodone) affect the Cyt P450 and AEM of CPY2D6 in ASD remains a significant pathway in ASD.

Conclusion: The AEM of the CPY2D6 gene and Cyt P450 remain the risk determinant in ASD, indicative of unhealthy environment evaluation, understanding/application in ASD incidence reduction among B/AA and White children in the US.