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Glucocorticoid receptor (NR3C1), Serotonin (5-HTT), Glutamate Decarboxylase (GAD1/GAD2) Genes in Childhood Aberrant Epigenomic Modulation in Bipolar Affective Disorder (BPAD) and Generalized Anxiety Disorders (GAD) in the USA: QES

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Background: The BPAD and GAD have increased in the USA after two decades, which reflects racial/ethnic disproportionate psychosocial and physical environmental burdens. The disproportionate risk factor in early age is not very fully understood. This study aimed to examine substance use associated with BPAD/GAD genes.

Rationale/Significance: The BPAD is indicative of chromosome 6,8,18, 22 and GAD genes as Glutamate Decarboxylase (GAD1 and GAD2) in GABA neurotransmitter involved in GAD. 5-HTT, NR3C1 and GAD genes are indicative of anxiety and depression as BPAD as well as GAD. The understanding of these genes signals anxiety and depression regulation, hence environmental stability in BPAD and GAD reduction.

Hypothesis: The NR3C1 and 5-HTT gene dysfunctionality as well as aberrant epigenomic modulation of these genes signal abnormal BPAD. Children ages 4 – 19 were included in some of the studies performed in this direction. The analysis utilized the common effect size (CES) as Desermonian/Laird in non-fixed effect method.

Results: The findings included 6 studies with BPAD (n=205) and 4 with GAD (n=312). The BPAD, CES = 3.62, 95%CI, 1.43-6.95 and GAD, CES= 2.97, 95% CI, 1.98-9.56.

Discussion: Children exposed to environment where parents use opioid, opioid derivatives, marijuana, tobacco, alcohol and other substances experience anxiety, depression. The exposure of children to these SUDs by the parents and the community predispose to their behavioral disorders, namely anxiety, depression, and bipolar.

Conclusion: The exposure of children to SUDs environment alter the gene, namely NR3C1, 5-HTT, GAD1/2 gene predisposing them to anxiety, depression and bipolar as maniac depressive disorder.