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Surrogate Aberrant Epigenomic Modulations in Mental Health with Transporter Gene Receptors: Unequal Burden of Addiction and Mental/Behavioral Disorders among Racial/Ethnic Minorities and the Underserved Whites, Quantitative Evidence Synthesis

Background: The transporter genes receptors generally constituted by twelve transmembrane spanning regions, implies mediated Na^+ - or H^+ -dependent accumulation as neurotransmitters. The neurotransmitter uptake is the Na^+/Cl^- dependent neurotransmitter transporters, which contained monoamine-recognizing such as dopamine, noradrenaline, and Serotonin(5HT) and amino acid-like (GABA, glycine, betaine, taurine, proline) subfamilies. Additionally, there are other families such as (a) Na^+/K^+ /glutamate transporter, (b) Na^+ -dependent glucose transporter and (c) H^+ -dependent vesicular monoamine transporters. The Bipolar Affective Disorder(BpAD) and GAD disproportionately affects racial/ethnic minorities, which may be explained by substance use and physical environmental burdens in these communities. Disproportionate risk factor in early age in this racial/ethnic minorities is not very fully understood, indicative of aberrant epigenomic in BpAD and GAD gene expression downregulation.

Materials/Methods: To examine substance use, such as drugs and heavy alcohol associated with transporter gene receptor as BpAD/GAD genes. The Quantitative Evidence Synthesis (QES) is applicable in evidence discovery with Desermonian-Laid, and combined effect size (CES) and heterogeneity effect.

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 and 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 and aberrant epigenomic modulation of these genes signal abnormal BpAD.

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 parental opioid, opioid derivatives, marijuana, tobacco, alcohol and other substances experienced anxiety, depression and bipolar.

Conclusion: The children exposed to SUDs environment alter the gene, namely NR3C1, 5-HTT, GAD1/2 gene predisposing as anxiety, depression and bipolar.

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