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RN3C1 Glucocorticoid Receptor Gene and Serotonin Gene (5-HTT) Aberrant Epigenomic Modulation in Maternal Depressive Disorders (MDD) among Racial/Ethnic Minorities in the US: Quantitative Evidence synthesis (QES)

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Background: Substantial MDD as Major Depressive Episode (MDE) reflects several causal environments such as isolation, discrimination, racism, and low socioeconomic status (SES) indicative of pathophysiologic alteration within the brain and central nervous system (CNS), post-synaptic neuronal depolarization and hyperpolarization.

Rationale/Significance: MDE among women within any geographic locale is driven by social, economic, and environmental alterations pertaining to human health. MDE narrowing requires the application of NR3C1 and 5-HTT gene receptors in understanding and marginalization of MDE.

Hypothesis/Method: The NR3C1 and 5-HTT receptor genes are implicated in MDE, which was conducted using QES. The common effect size (CES) was estimated using Desermonian – Laid as point estimates and precision measure.

Results: The QES assessment included Black/AA, 98 (18.2%), American Indian/Alaska Natives (AI/AN), 32 (5.9%), Hispanic, 122, (22.7%), and underserved White, 286, (53.2%). The NR3C1 gene associated with MDE among B/AA was, CES = 8.63, 95% CI, 6.65- 9.67; AI/AN CES = 8.54, 95% CI, 0.83-12.67; Hispanics CES = 1.89, 95% CI = 1.23-4.89; and White CES = 2.36, 95% CI, 1.96-3.39.

Discussion: MDE involves gene/environment interactions that involved isolation, discrimination, and social gradient. This study observed aberrant epigenomic modulation in NR3C1 and 5-HTT receptors in racial/ethnic minorities populations, namely B/AA, and AI/AN. Specifically, MDE reflects maternal mortality, which is three times as likely in B/AA and AI/AN in the US indicative of environmental alteration in decreasing maternal mortality.

Conclusion: MDE is driven by aberrant epigenomic modulations involving NR3C1 and 5-HTT gene expression, hence cellular dysfunctionality reflecting disproportionate burden of MDE among B/AA, AI/AN, and Whites.