

Title: Environmental Effects on Human Placental DNA Methylation and its Role in Development of Preeclampsia

Abstract

Preeclampsia is a disorder in pregnant women characterized by hypertension following 20 weeks of pregnancy. It is known to be a major cause of maternal morbidity and can have detrimental outcomes on the fetus. While the exact cause of preeclampsia remains unknown, the general consensus suggests the disorder originates from poor early development of the placenta. Environmental exposures such as environmental pollutants, maternal obesity, nutritional imbalance, maternal stress, and lack of social support have been associated with the disorder, many of which are known to increase the risk of developing preeclampsia. These environmental factors can influence placental and fetal development through epigenetic mechanisms, including DNA methylation. This literature review synthesizes current research examining the effects of maternal exposure to environmental factors on placental DNA methylation. Review of recent studies revealed that maternal and prenatal exposure to common environmental influences were associated with a change in normal DNA methylation patterns, especially within genes and pathways important to placental angiogenesis, vascular development, and metabolic function. Several studies identified exposure-specific methylation changes, suggesting that the pathways involved in placental development are directly affected during gestation. These changes in methylation patterns are being recognized as factors contributing to the development of preeclampsia, and therefore require further in-depth research. Future research should continue to study the effects of different environmental influences on placental DNA methylation, in the context of how it directly affects the development of preeclampsia. As we deepen the understanding of these effects, research should then prioritize the development of non-invasive diagnostic and predictive tools, and hopefully develop drugs or prevention therapies that can target specific mechanisms or pathways.