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Mouse model provides new insight in to preeclampsia

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Worldwide, preeclampsia is a leading cause of maternal deaths and preterm births. This serious pregnancy complication results in extremely high blood pressure and organ damage. The onset of preeclampsia is associated with elevated levels of a secreted protein, sFLT1, in maternal blood. However, the factors that regulate sFLT1 production are poorly understood.

A new study in the *Journal of Clinical Investigation* suggests that placental cells produce sFLT1 in response to maternal increases in VEGF, which binds sFLT1 and affects blood vessel formation and function.

Nihar Nayak and colleagues at Wayne State University evaluated tissue samples from women with preeclampsia and determined that VEGF is increased in maternal cells and sFLT1 is increased in placental cells. In a mouse model, enhanced VEGF expression in maternal cells induced expression of placental sFLT1.

Moreover, maternal VEGF expression induced preeclampsia-like symptoms and pregnancy loss in these mice.

The results from this study suggest that alterations in VEGF and sFLT1 levels during pregnancy could have serious consequences and that caution should be used for use of therapies targeting these proteins for treating preeclampsia.

More information: Endometrial VEGF induces placental sFLT1 and leads to pregnancy complications, *J Clin Invest*. DOI: 10.1172/JCI76864

sFLT1 in preeclampsia: trophoblast defense against a decidual VEGFA barrage? *J Clin Invest*. DOI: 10.1172/JCI78532