Development of a model to test pre-eclampsia therapeutics

Experts in placental biology and disease, our researchers have developed an in vivo model appropriate for the testing of potential therapeutics for preeclampsia.

Despite years of study, we still do not have a clear understanding of the underlying causes of preeclampsia. Our experts have developed a world-first in vivo model that can be used to develop an understanding of this disease and test potential therapeutics.

Summary

Preeclampsia is the most common medical complication of pregnancy, affecting 5-8% of pregnancies. It is a major cause of fetal and maternal morbidity and mortality.

Preeclampsia can have severe outcomes for mother and baby, and failure to manage or diagnose the disease can be life-threatening. Preeclampsia and associated complications are responsible for approximately 15% of maternal deaths, and up to 20% of the 13 million preterm births worldwide each year.

The causes of preeclampsia are still not well understood, despite many years of research. Preeclampsia is unique to humans, and the lack of an appropriate animal model that fully recapitulates features of the disease has been a major limitation preventing the development of new diagnostics and treatments for preeclampsia.

The Hudson team, led by female reproductive health expert Professor Eva Dimitriadis, have developed a unique mouse model, characterized by elevated levels of the cytokine Interleukin-11 (IL11), that develops impaired trophoblast invasion and placental development as well as key symptoms such as hypertension and kidney pathology, including proteinuria. These characteristics mimic the main features of human preeclampsia. Our researchers have successfully used this model to develop an understanding of the role of IL11 in the development of preeclampsia, and its position as a potential therapeutic and screening biomarker.

Publication

Winship et al. (2015) Interleukin 11 alters placentation and causes preeclampsia features in mice. Proc Natl Acad Sci USA. 112(52):15928-33.

IP position

PCT/AU2016/050883 filed September 2016 – 'A method of treatment and prophylaxis'

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