# Endothelial pathology in preeclampsia

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**Stellingen**

*behorende bij het proefschrift getiteld "Endothelial pathology in preeclampsia"*
*door R.J. Turner*

1. Mutations within genes encoding factors from the coagulation and fibrinolysis system are associated with preeclampsia. *(this thesis)*
2. Loss of placental thrombomodulin contributes to the development of preeclampsia and is associated with the production of the anti-angiogenic factor soluble Flt-1. *(this thesis)*
3. In preeclamptic nephropathy, endothelial thrombomodulin expression is increased, indicative of a protective process. *(this thesis)*
4. In renal damage in preeclampsia, the angiogenic imbalance leads to damage to both sides of the glomerular filtration barrier: to the endothelial cells, and to the podocytes. *(this thesis)*
5. The splicing pattern of vascular endothelial growth factor is stable in kidney disease. *(this thesis)*
6. Understanding the dysregulated antiangiogenic pathway in the syncytium and its role in mediating maternal vascular disease marks a significant advance in our efforts to explain the origins of preeclampsia. *(Karumanchi, Hypertension. 2016;67:1072-1079)*
7. Preeclampsia may serve as a marker for women at risk of developing cardiovascular disease. *(Adapted from Garovic and August, Curr Hypertens Rep. 2013 Apr; 15(2): 114-21)*
8. Podocytes "live and work" under precarious conditions. (*Kriz et al, Am J Physiol Renal Physiol. 2013 Feb 15; 304(4): F333-47)*
9. Future research should refocus from a podo-centric view back to one that examines the signals that pass between the three major different cell types in the glomerulus. *(Adapted from Quaggin, Kreidberg, Development 2008 135: 609-620)*
10. I have not failed. I've just found 10,000 ways that won't work. *(Thomas A. Edison, 1847 - 1931)*

***Studies where the null hypothesis could not be rejected (that yielded 'negative' results), should not disappear into oblivion; they can contain valuable information on the roads that have already been explored.***