



Universiteit
Leiden
The Netherlands

Endothelial pathology in preeclampsia

Turner, R.J.

Citation

Turner, R. J. (2018, September 5). *Endothelial pathology in preeclampsia*. Retrieved from <https://hdl.handle.net/1887/64970>

Version: Not Applicable (or Unknown)

License: [Licence agreement concerning inclusion of doctoral thesis in the Institutional Repository of the University of Leiden](#)

Downloaded from: <https://hdl.handle.net/1887/64970>

Note: To cite this publication please use the final published version (if applicable).

Cover Page



Universiteit Leiden



The handle <http://hdl.handle.net/1887/64970> holds various files of this Leiden University dissertation.

Author: Turner, R.J.

Title: Endothelial pathology in preeclampsia

Issue Date: 2018-09-05

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.