

Extracorporeal apheresis to treat preeclampsia

A novel VEGF-multimer based approach

Invention



Elimination of sFLT-1 restores VEGF and PIGF pro-angiogenic function and ensures fetal blood supply through sufficient vascularisation. In addition, maternal multiorgan failure is prevented.

Preeclampsia is still a threat to pregnant women's health world-wide and in industrialized countries the most common cause of prematurity, including all potential complications of preterm delivery and its sequelae later in life. In the pathogenesis of preeclampsia, increased expression of soluble FMS-like tyosine kinase 1 (sFIt-1) acts as an antagonist by scavenging and neutralizing the proangiogenic vascular endothelial growth factor (VEGF) and placental growth factor (PIGF). The resulting angiogenic imbalance may progress to enothelial dysfunction and thrombotic microangiopathy, manifesting with seizures, stroke, or multiorgan failure. Effective therapeutic and prophylactic measures are lacking, limiting interventional options to premature termination of pregnancy. Thus, therapies for preeclampsia are still an unmet and urging medical need.

The underlying invention aims at using an apheresis based approach with recombinant monomeric (moVEGF¹⁶⁵) or two single-chain dimers of VEGF¹⁶⁵ (scVEGF¹⁶⁵). Both compounds enhance binding affinity to sFIt-1 and efficiently reduce or eliminate sFIt-1 levels from plasma of preclampsia affected women. Compared to approaches based on antibodies in the art, the use of both compounds as a ligand has the additional effect that bound VEGF and PIGF are displaced and released from sFIt-1. Overall, this minimizes the risk of life-threatening conditions and, in particular, restores uterine vascularization and establishment of uteroplacental circulation.

Commercial Opportunities

On behalf of the University Hospital of Cologne, PROvendis offers an access to rights for product development and commercial use of this invention with moVEGF¹⁶⁵ and scVEGF¹⁶⁵.

Current Status

Before starting clinical studies in pregnancies with preeclampsia, toxicity and compatibility, e.g. the safety of endogenous VEGF- and PIGF-release for mother and fetus, has to be tested in vivo.

Relevant Publications

Matin, M. et al. (2020) Hypertension. 2020 Oct;76(4):1176-1184. WO2021/185996 A1

An invention of University of Cologne.

Competitive Advantages

- Restoring the angiogenic balance by displacing endogenous VEGF and PIGF from its binding to sFlt-1
- moVEGF¹⁶⁵ and scVEGF¹⁶⁵ have a higher affinity to sFlt-1 as compared to antibodies
- Efficient elemination of sFlt-1 from plasma by moVEGF¹⁶⁵ and scVEGF¹⁶⁵

Technology Readiness Level

123456789 Experimental proof of concept

Industries

- Medical engineering
- Pharmaceutical industry gynecology

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