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Sommario/riassunto

A large number of publications have described impaired angiogenesis and vasculogenesis present in the feto-placental circulation after pregnancy diseases such as pre-eclamptic pregnancies, gestational diabetes, and intrauterine growth restriction, among others. Results suggest impaired secretion and activity of pro-angiogenic factors such as vascular endothelial growth factor (VEGF), interleukin 8 (IL-8), adenosine and nitric oxide, associates with compromised secretion and activity of anti-angiogenic factors such as soluble receptor of VEGF (sFlt-1), thrombospondin 2, endostatin among others. More recent evidences include the participation of endothelial progenitor cells (EPC), which circulating number is reduced infeto-placental circulation in pregnancies such as pre-eclampsia. Despite this knowledge, therapies for placental angiogenesis recovery during pathological pregnancies are far to be tested. However, from the cardiovascular field, it has been described the administration of EPC, alone or used as gene-transfer therapy; or it has been described the potential role of statins (HMGCoA inhibitors), or angiotensin-converter enzyme (ACE) inhibitors for enhancing angiogenesis. Finally, feto-placental tissue is an exceptional source of progenitor and stem cells, which could be used for treated other human diseases such as stroke, myocardial infarction, hypertension, or even cancer. In this research topic, authors highlight physiopatological and clinical importance of the impaired placental angiogenesis, and suggest potential targets for developing innovative

therapies.