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Power of vascular protective pathways in preeclampsia and atherosclerosis

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The incidence of preeclampsia is reduced by a third in smokers, but not in snuff users. Soluble Flt-1 (sFlt-1) and soluble endoglin (sEng) are increased prior to the clinical onset of preeclampsia. Animals exposed to high circulating levels of sFlt-1 and sEng elicit severe preeclampsia-like symptoms. Smokers have reduced circulating sFlt-1 and cigarette smoke extract decreases sFlt-1 release from placental villous explants. An anti-inflammatory enzyme, heme oxygenase-1 (HO-1) and its metabolite carbon monoxide (CO), inhibit sFlt-1 and sEng release. Women with preeclampsia exhale less CO than women with normal pregnancies and HO expression decreases as the severity of preeclampsia increases. In contrast, sFlt-1 levels increase with increasing severity. More importantly, chorionic villous sampling from women at eleven weeks gestation shows that HO-1 mRNA expression is decreased in women who go on to develop preeclampsia. Collectively, these facts provide compelling evidence to support the proposition that the pathogenesis of preeclampsia is largely due to loss of HO activity. This results in an increase in inflammation and excessive elevation of the two key anti-angiogenic factors responsible for the clinical signs of preeclampsia. These findings provide strong evidence for a protective role of HO-1 in pregnancy and identify HO as a target for the treatment of preeclampsia. The cardiovascular drugs, statins, stimulate HO-1 expression and inhibit sFlt-1 release *in vivo* and *in vitro*, thus, they have the potential to ameliorate early onset preeclampsia. The StAmP trial is underway to address this and if positive, its outcome will lead to the very first therapeutic intervention to prolong affected pregnancies.