Endothelial dysfunction is a key pathophysiological state in diseases including pre-eclampsia. VEGF-A is detected at higher concentrations in plasma from patients with pre-eclampsia than normotensive pregnant patients when determined by radioimmunoassay, but not by ELISA, suggesting that binding of VEGF in pre-eclampsia is altered. However, the biologically activity of VEGF has never been measured \textit{in vivo}. We aimed to measure this and identify the isoforms responsible for this activity. Plasma from pre-eclamptic pregnant women significantly increases microvascular permeability of mesenteric microvessels. We have now recently shown that this increase is blocked by bevacizumab a VEGF antibody and by VEGF receptor inhibition, but it is VEGFR1 not VEGFR2 inhibition that prevents this response. VEGF$_{165}$ does not increase permeability through VEGFR1 (it acts through VEGFR2), but the anti-angiogenic splice variant, VEGF$_{165}^b$ does. Although VEGF$_{165}$ levels were not significantly altered in the PET samples the increase in permeability was inhibited by incubation of pre-eclamptic plasma with a monoclonal antibody specific for VEGF$_{165}^b$. Both VEGF$_{165}$ and another VEGF family member PIGF act on VEGFR1 and both are bound by circulating sVEGFR1, known to be enhanced in pre-eclampsia, resulting in undetectable PIGF1 levels. To determine whether it is loss of repression of VEGF$_{165}^b$-VEGFR1 signalling by an absence of PIGF-1 that causes the permeability response we added PIGF to pre-eclamptic plasma. This blocked the permeability response. Thus circulating VEGF levels in pre-eclampsia are biologically active, but it may be VEGF$_{165}^b$, binding VEGFR1 due to the absence of PIGF that regulates endothelial dysfunction in pre-eclampsia.