The traditional view has it that conditions in which viscosity is increased may be associated with cardiovascular disease, and that vice-versa cardiovascular disease leads to hyperviscosity. Whether a cause or a consequence, the role and size of the variability in viscosity has not yet been put in relationship with changes in endothelial function at a population level. Further complicating this issue, direct assessment of viscosity has been performed in a very little number of sufficiently large studies.

More recently, the understanding of the role of the vascular endothelium in the control of vasomotion, and of the importance of shear stress and viscosity in modulating endothelial activity has cast some doubt on the relationship between viscosity and endothelial function. Vascular homeostasis is a dynamic system in continuous equilibrium between vasodilation and vasoconstriction, constantly kept in balance by the release of endothelial autacoids which in turn have an effect on the regulation of the deformability and aggregability of circulating cells, erythrocytes, leucocytes and platelets, thus controlling both arterial tone and blood viscosity. Increases in viscosity might be seen as stimuli for endothelial activation but also as a consequence of impaired endothelial function. In our hands, we found a strong association between risk factors, blood pressure and viscosity, but very limited associations with parameters of endothelial function.