

Propositions belonging to the thesis:

Modulating NO-cGMP signalling in ageing models

1. DNA damage in vascular smooth muscle cells causes tissue-autonomic ageing (this thesis).
2. Vascular smooth muscle cell dysfunction begets endothelial cell dysfunction (this thesis).
3. Improvement of NO-cGMP signalling with a soluble guanylate cyclase activator slows down vascular ageing, fostering the repurposing of such medication for the prevention of vascular ageing (this thesis).
4. Among all the options to block phosphodiesterases in the vasculature, phosphodiesterase 1 inhibition is a promising newcomer that shows capability to improve vasorelaxation via both cAMP and cGMP and to attenuate vascular ageing (this thesis).
5. The clinical potential of sildenafil treatment to improve hemodynamic function has not been reached yet (this thesis).
6. Inflammaging serves as an early indicator of cardiovascular associated morbidity and mortality.
7. Dietary restriction-mimics are the way to go.
8. If one is to target an ageing-related disease, one is to target ageing itself.
9. sGC activators might make senolytics obsolete.
10. Scientists who conduct animal studies value the lives of animals
11. *“Research is what I’m doing when I don’t know what I’m doing.” – Wernher von Braun*