Propositions

belonging to the thesis

DNA Damage-related Vascular Dysfunction

Pathways and Interventions

1. Selective inhibition of phosphodiesterase 1 is an attractive option for the treatment of age-related cardiovascular disease. (this thesis)

2. DNA damage in endothelial cells and vascular smooth muscle cells results in changes that resemble aging-related vascular disease. (this thesis)

3. Dietary restriction, through genoprotection, allows the genome to be more resilient to a diseased state, as evidenced by the recruitment of prostaglandins to bypass the loss of NO-cGMP signaling after DNA damage. (this thesis)

4. Since mouse models of genomic instability represent the renin-angiotensin system blockade-resistant part of aging-related vascular disease, they allow to further explore such resistance. (this thesis)

5. The fact that oxidative activation of the protein kinase G 1α contributes to endothelium-derived hyperpolarization illustrates why the use of untargeted anti-oxidants cannot be an effective anti-ageing therapy. (this thesis)

6. Studies applying dietary restriction provide valuable biomedical information about health and longevity, but for humans this is as far as we can get.

7. To stay young senotherapy alone is insufficient: psychotherapy will be a mandatory complement.

8. The fact that there is no senescence-specific biomarker makes every scientific discussion or expectation for the clinic concerning "senolytic" drugs futile.

9. Eating less and exercising more abolishes the need for epidemiological and intervention studies addressing nutrition.

10. Science and everyday life cannot and should not be separated. (Rosalind Franklin)

11. If scientific progress for the major part depends on serendipity, making the decision to proceed is the most important step in a research program.

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