Stellingen

1. Pharmaceutical interventions should be an addition to, rather than a substitute for, dietary and lifestyle interventions.

2. Lifelong exposure to elevated LDL-cholesterol levels increases atherosclerosis in mice and cardiovascular event rates in humans, and lowering LDL-cholesterol early in life may result in long term gains (this thesis & Paul M Ridker Lancet 2014; 384: 607-17).

3. LDL-cholesterol remains the leading target for the treatment of cardiovascular disease and the efficacy of many pharmaceutical compounds, including HDL-cholesterol-raising drugs depends on their ability to lower LDL-cholesterol (this thesis).

4. HDL-cholesterol is a marker and not a causal risk factor for cardiovascular disease (this thesis).

5. The extent of lipid-lowering by compounds observed in the APOE*3Leiden.CETP mice, initially developed as an animal model for familial dysbetalipoproteinemia (FD) or type III hyperlipoproteinemia, is comparable to that of FD patients (this thesis).

6. Proprotein convertase subtilisin/kexin type 9 (PCSK9) and cholesteryl ester transfer protein (CETP) are important proteins in lipid metabolism and these proteins are affected by lipid-modifying compounds that do not specifically target PCSK9 and CETP (this thesis).

7. Statins are hindering the development of other treatment options for patients that do not benefit from statins.

8. The patient population and study design are just as important as the type of compound in determining the success rate of a clinical trial.

9. A passport is not a requirement to consider a country home.

10. It is not where you go, it is who you know.