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Cardiometabolic risk factors and venous thrombosis

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Stellingen behorend bij het proefschrift

Cardiometabolic risk factors and venous thrombosis

1. Levels of major lipids, i.e. total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and triglycerides, are not associated with risk of venous thrombosis (*this thesis*).
2. Low levels of apolipoproteins B and A1 are associated with an increased risk of a first venous thrombosis (*this thesis*).
3. Tests for lipid levels, glucose levels and hematologic variables do not identify patients at an increased risk of recurrent venous thrombosis, and these tests should not be done for this indication nor influence decisions on duration of anticoagulant treatment (*this thesis*).
4. Levels of vitamin K-dependent factors, including factor IX, are associated with serum and hepatic triglyceride levels (*this thesis*).
5. Hepatic triglyceride content and factor IX levels are associated in a dose-response fashion even after adjustment for several potential confounding factors, including total body and visceral fat (*this thesis*).
6. Venous thrombosis is a multicausal disease occurring as the result of interacting genetic, environmental and behavioral risk factors (*Frits R. Rosendaal. Hematol Am Soc Hematol Educ Progr, 2005*).
7. The pathogenic changes that occur in the blood vessel wall and in the blood itself resulting in thrombosis are not fully understood. Understanding these processes is crucial for developing safer and more effective antithrombotic drugs (*Nigel Mackman. Nature, 2008*).
8. Epidemiology seeks to be precise and quantitative, but we do not have a precise-let alone quantitative-definition of causation, notwithstanding thousands of years of trying (*Jan P Vandenbroucke. Int J Epidemiol, 2016*).
9. The research process of learning about and controlling for confounding can be thought of as a walk through a maze toward a central goal. As the layers of confounding are left behind, we gradually approach a deeper causal understanding of the underlying biology. Unlike a maze, however, this journey toward the goal of biologic understanding does not have a clear end point, because there is always room to understand the biology in a deeper way (*Kenneth J. Rothman. Epidemiology: an introduction, 2012*).
10. Nothing in life is to be feared, it is only to be understood. Now is the time to understand more, so that we may fear less (*Marie Skłodowska Curie, 1867-1934*).

Vânia Maris Morelli
Leiden, 28 November 2017