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SELF-RELATED HEALTH AND BLOOD HYPERCOAGULABILITY: A POSSIBLE MECHANISM FOR CARDIOVASCULAR RISK ?

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Low self-related health (SRH) is one, single-item, measure of low chronic general health. SRH has been shown to be a predictor of total mortality and of cardiovascular mortality in prospective studies of generally healthy populations (1-3). Several factors influence SRH scores, including medically diagnosed conditions; adverse lifestyle and other risk factors for total and cardiovascular mortality (including age); and individual resilience and resources to cope with declining health (4,5). Adjustment for major established risk factors does not, however, account for the association between SRH and total and cardiovascular mortality in population studies (6,7).

Furthermore, SRH scores were also predictive of recurrent coronary heart disease (CHD) events and death in patients with CHD at baseline (8,9).

Components of the haemostatic system play several roles in atherosclerosis, arterial thrombosis and cardiovascular disease (10-12). Several blood hypercoagulability measures, including plasma levels of fibrinogen, von Willebrand factor (VWF) antigen, fibrin D-dimer and tissue plasminogen activator (t-PA) antigen are now established as risk factors for coronary heart disease, stroke and cardiovascular mortality in prospective studies (13-15). While plasma levels of these haemostatic factors are associated with several established cardiovascular risk factors (age, smoking, body mass index, blood pressure, cholesterol, triglycerides, and inflammatory markers), they are independently associated with cardiovascular risk after adjustment for these risk factors (13-15).

The association between SRH and components of the haemostatic system has not been previously studied. In this issue, von Kanel and colleagues report the association of SRH with plasma levels of fibrinogen, VWF antigen and fibrin D-dimer with SRH scores, in 190 patients within 48 hours of an acute coronary intervention for acute myocardial infarction (16). SRH, before their event, was rated on a visual analogue scale. In fully adjusted linear regression models, SRH was inversely associated with plasma fibrinogen (r=-0.33), VWF (r=-0.19) and D-dimer (r=-0.25). The Global Registry of Acute Coronary Events (GRACE) risk score was similarly associated with these three haemostatic factors.

The authors suggest that haemostatic activation may potentially link SRH with cardiovascular outcome in patients with acute coronary syndromes; because previous studies of such patients have reported that plasma fibrinogen, VWF and D-dimer levels are associated with risk of major CVD events and death up to 2 years after discharge (17-20). They also note that these and other haemostatic factors may be expressed during inflammation in the central nervous system (21), raising the possibility that they may have effects on the neural contribution to SRH scores.

The authors acknowledge some limitations of their study. First, their subjects were a selected group of patients, who had satisfied the criteria for inclusion in a randomized controlled trial of the effects

of early psychological counselling on the development of post-traumatic stress after acute myocardial infarction; most were men with higher education levels. They were asked to retrospectively score their SRH before their acute event, after their circulatory condition had stabilised. It is therefore possible that the acute effect influenced their recall of SRH.

Second, blood samples were also taken during the acute event, therefore measuring acute phase increments from basal levels in plasma levels of haemostatic factors (which were positively associated with GRACE risk scores), as well as the effects of antithrombotic treatments (although exploratory analyses adjusting for antithrombotic drug use did not significantly attenuate the associations of SRH with haemostatic factors).

Third, this cross-sectional study did not investigate the associations between SRH, plasma levels of haemostatic factors and long-term outcomes after acute coronary syndromes.

The study of von Kanel and colleagues (16) is of interest because it raises the hypothesis that activated haemostasis may be one potential causal mechanism for the established associations between low SRH and adverse cardiovascular outcomes. Further studies of this hypothesis should be prospective, and conducted in representative samples of both general populations and persons with baseline cardiovascular disease. As fibrinogen, VWF, D-dimer and some other haemostatic factors are inflammatory reactants, inflammation markers such as C-reactive protein and interleukin-6 should also be studied. While haemostasis and antithrombotic drugs play important roles in cardiovascular disease and its prevention and treatment, there is increasing interest in inflammation and anti-inflammatory drugs in cardiovascular disease prevention (22-25).

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