Vitamin D binding protein and endothelial injury after hematopoietic stem cell transplantation: an actin scavenger with a lipid-bound character

In a recent issue of Haematologica, Luebbering *et al.* described an association between filamentous-actin (F-actin), a mediator of endothelial damage, and vitamin D binding protein (VDBP), an actin scavenger, as modifiers of risk of clinical consequences of endothelial injury.[1] As mentioned by the authors, the amount of actin-VDBP complexes present differed among the individuals and was influenced by several factors. Besides the variables described in the manuscript, one should take into account the partly lipid-bound character of VDBP. More specifically, the transport of the macromolecular bound VDBP-actin complexes is partly carried out by very low-density lipoprotein (VLDL) particles. VLDL carry 90% of the serum triglycerides in the fasting state. In our previous work[2] investigating the evolution of the serum VDBP concentration in cardiac surgery patients, the delta serum VDBP concentrations correlated significantly with the delta serum triglyceride concentrations. The relationship between the serum concentrations of this 52-59 kDa a$_1$-globulin and a number of lipid-related parameters (serum total cholesterol, LDL-cholesterol, triglycerides) was demonstrated.[3] In addition, a partial coprecipitation of actin, VDBP, very low-density lipoprotein (VLDL) plus low-density lipoprotein (LDL) particles was observed using precipitation experiments, high-pressure gel permeation chromatography and ultracentrifugation.[2] Being part of the VLDL, VDBP associates with globular actin (G-actin) in VLDL particles and acts as a transport protein.[2-4] So based on these findings, the difference in serum actin-VDBP complexes following hematopoietic stem cell transplantation might be partly explained by the interactions between VDBP, actin and lipids.

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