

Plasma phospholipid transfer protein deficiency in mice is associated with a reduced thrombotic response to acute intravascular oxidative stress. C. Desrumaux, V. Deckert, S. Lemaire-Ewing, C. Mossiat, A. Athias, D. Vandroux, L. Dumont, S. Monier, J.-P. Pais de Barros, A. Klein, E. De Maistre, D. Blache, A. Beley, C. Marie, P. Garnier, L. Lagrost, *Arterioscler. Thromb. Vasc. Biol.*, 30, 2452-2457 (2010).

OBJECTIVE: Earlier in vitro studies suggested a putative role for the plasma phospholipid transfer protein (PLTP) in the modulation of blood coagulation. The effect of PLTP expression on blood coagulation under both basal and oxidative stress conditions was compared here in wild-type and PLTP-deficient (PLTP^{-/-}) mice.

METHODS AND RESULTS: Under basal conditions, PLTP deficiency was associated with an extended tail bleeding time despite a significant depletion of vascular α -tocopherol content and an impairment of endothelial function. When acute oxidative stress was generated in vivo in the brain vasculature, the steady state levels of oxidized lipid derivatives, the extent of blood vessel occlusion, and the volume of ischemic lesions were more severe in wild-type than in PLTP^{-/-} mice.

CONCLUSIONS: In addition to its recognized hyperlipidemic, proinflammatory, and proatherogenic properties, PLTP increases blood coagulation and worsens the extent of ischemic lesions in response to acute oxidative stress. Thus, PLTP arises here as a cardiovascular risk factor for the late thrombotic events occurring in the acute phase of atherosclerosis.