

Regulation of mitochondrial function as a promising target in platelet activation-related diseases

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© 2019 Elsevier Inc. Platelets are anucleated cell elements produced by fragmentation of the cytoplasm of megakaryocytes and have a unique metabolic phenotype compared with circulating leukocytes, exhibiting a high coupling efficiency to mitochondrial adenosine triphosphate production with reduced respiratory reserve capacity. Platelet mitochondria are well suited for ex vivo analysis of different diseases. Even some diseases induce mitochondrial changes in platelets without reflecting them in other organs. During platelet activation, an integrated participation of glycolysis and oxidative phosphorylation is mediated by oxidative stress production-dependent signaling. The platelet activation-dependent procoagulant activity mediated by collagen, thrombin and hyperglycemia induce mitochondrial dysfunction to promote thrombosis in oxidative stress-associated pathological conditions. Interestingly, some compounds exhibit a protective action on platelet mitochondrial dysfunction through cont