Procoagulant platelets are known by several synonyms, including collagen and thrombin–activated platelets, coated platelets, sustained calcium–induced platelet morphology, superactivated platelets, and superplatelets. This assortment of names is confusing to those outside the field and has remained a stubborn problem within the field. However, it is key to recognize that all of these variously named activated platelets share a common pentad of characteristics, mitochondrial depolarization, sustained cytoplasmic calcium elevation, surface expression of phosphatidylserine (PS), inactivation of glycoprotein Ib/IIa receptors, and enhanced retention of several procoagulant proteins, including fibrinogen, factor V, and von Willebrand factor, within a surface cap. This commentary is too brief to detail all the intracellular events associated with the production of procoagulant platelets but suffice it to say that simultaneous engagement of thrombin and collagen receptors is the most common mechanism for their production. Surprisingly, dual agonist activation results in only a fraction of platelets being converted into procoagulant platelets, on average 30% in humans. The remaining 70% of platelets demonstrate traditional activation endpoints: active fibrinogen receptors, granule release, and little to no exposed PS. The mechanism that bifurcates a platelet population into these 2 distinct products remains unknown.

See accompanying article from the August 2017 issue on page 1503

Procoagulant platelets are an efficient platform for assembly of the prothrombinase complex, an observation that has guided studies examining their physiological significance. Along these lines, Brooks et al identified German Shepherd dogs with an inherited bleeding diathesis resulting from an inability to produce procoagulant platelets. Studies in humans demonstrated that not all individuals make the same level of procoagulant platelets, as a mechanism whereby procoagulant platelets can be retained within a growing thrombus. Although some have suggested that procoagulant platelets may actually be negative regulators of thrombus growth because they do not propagate the aggregate, this premise contradicts the clinical and large animal data that support a prothrombotic role for procoagulant platelets (Figure).
The last 17 years have seen the introduction of procoagulant platelets, an examination of the synthetic steps involved in their formation, and some clarity concerning their physiological function. Future work will hopefully lead to a further understanding of procoagulant synthetic steps, as well as identification of specific antiplatelet medications aimed at the inhibition of either their synthesis or function.

Disclosures

None.

References


Procoagulant Platelets: Further Details but Many More Questions
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