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Opinion Article

Comprehensive Study on Isoprostane Biology and Platelet Functionality

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DESCRIPTION

Platelets' major function is hemostasis, the act of stopping bleeding from an injured vessel, hence maintaining good platelet function is essential. The hemostatic plug that forms to stop blood loss must have the ability to dissolve quickly once the wound has healed. However, to enable efficient nutrient and waste exchange in all other circumstances, blood flow must remain unrestricted. Thus, platelets are unavoidably tightly regulated blood components that must react fast and strongly to activating stimuli while remaining "fully" dormant otherwise. A variety of disorders result from problems with either of these activities. Additionally, a variety of activation defects lead to bleeding disorders that are linked to morbidity and mortality and may require lifelong treatment.

In contrast, myocardial infarction, ischemic stroke, peripheral artery disease, and other thrombotic diseases, which collectively represent a significant source of mortality, are characterized by inappropriate activation or recruitment of platelets to areas where hemostasis is not required. Understanding these signalling pathways will enable the creation of targeted and well-thought-out therapeutic intervention strategies, making the regulation of platelets and, more specifically, their activation, of major interest.

With hundreds of millions of them per millilitre of whole blood, platelets are the second most plentiful blood cell after red blood cells. Since they are all so little, this still only makes up a very small portion of the blood volume. This results from the fact that platelets are essentially cellular fragments and not "real" cells in and of themselves. As a result, some changes to their signalling or effector molecules are irreversible because they lack nuclei (e.g. nonspecific cyclooxygenase inhibition when platelets are exposed to aspirin). Only when the platelets are replaced with newly created cells does their function return. To this goal, megakaryocytes, very big cells, are used to create platelets, which are then created in the bone marrow.

Megakaryocytes undergo a budding process as they grow which releases thousands of platelets per megakaryocyte and enables rapid replenishment in the absence of platelet control problems. Additionally, platelets have adhesive proteins on their surface that enable them to stick to neighboring platelets' surface proteins as well as the exposed sub endothelium in a damaged blood vessel. As a result, the subsequent stage of activation is characterized by platelet adhesion and aggregation as they bind to the injured tissue and to one another, stopping additional blood loss from a lesion. Additionally, different intercellular granule types can be found in platelets (i.e., alpha and dense granules).

Due to the produced fibrinogen and thrombospondin, which further bind the platelets together, as well as the agonists released from dense granules, which can signal more secretion, aggregation is strengthened (thus providing a strong positive feedback loop). The effects of these drugs are supposed to enhance one another. As active platelets condense the loose clot that had previously been created to seal a vascular wound into a hard, compact mass capable of resisting dispersion until wound healing is complete, actin and myosin finally mediate platelet retraction.

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