

■ Figure 2-1 Diagram of platelet ultrastructure showing the major intracellular morphologic features. (Reprinted from Besa EC. Hematology. Baltimore: Williams & Wilkins: 1992:202 with permission.)

- von Willebrand's factor (factor VIII:R)
- d. β-Thromboglobulin (BTG)
- Platelet factor 4 (heparin-neutralizing)
- Fibronectin.
- 8. Dense granules contain nonprotein factors including:
 - Adenosine diphosphate (ADP)
 - b. Adenosine triphosphate (ATP)
 - e. 5-Hydroxytryptamine (5-HT; or serotonin)
 - d. Calcium

C. Platelet function

- Damaged subendothelium releases factors that activate the platelet, transform its shape, and evolve a "sticky" platelet aggregate to plug the leak. These factors include:
 - a. Collagen
 - b. Fibronectin
 - c. vWF (factor VIII:R)
 - Thrombin
 - e. ADP
- Tissue platelet activators cause the platelet to change shape from discoid to spherical.
 Dense and α-granules undergo internal contraction and centralization. The complete process is calcium dependent.
 - a. The exposure of surface membrane receptors to vWF and fibrinogen results in cytoplasmic calcium ionization, stimulation of ATP generation, and activation of the actin monomers in the micro filaments.
 - Contractions result in a centralization of the cytoplasmic granules and a release of their contents through the canalicular system.
- Plug formation, or secondary aggregation, is primarily stimulated by thrombin and thromboxane A₂ (TXA₂).
 - a. Membrane-binding of vWF and collagen to platelet receptors unmasks membrane phospholipid A₂, which is the precursor of arachidonic acid, and is important for the production of TXA₂ and other prostaglandins. Phospholipid A₂ is unmasked by the binding of vWF and collagen to platelet receptors on the membrane.
 - b. TXA₂ inhibits adenylate cyclase [thus, it inhibits the formation of cyclic adenosine monophosphate (cAMP)] and liberates sequestered calcium into cytoplasm. Calcium causes further cytoplasmic contraction, release of granule contents, and platelet aggregation.
 - Thrombin enzymatically cleaves fibrinogen to form fibrin, which is necessary to stabilize the platelet plug.

Study Guide For Clinical Laboratory Science

Mary Louise Turgeon

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