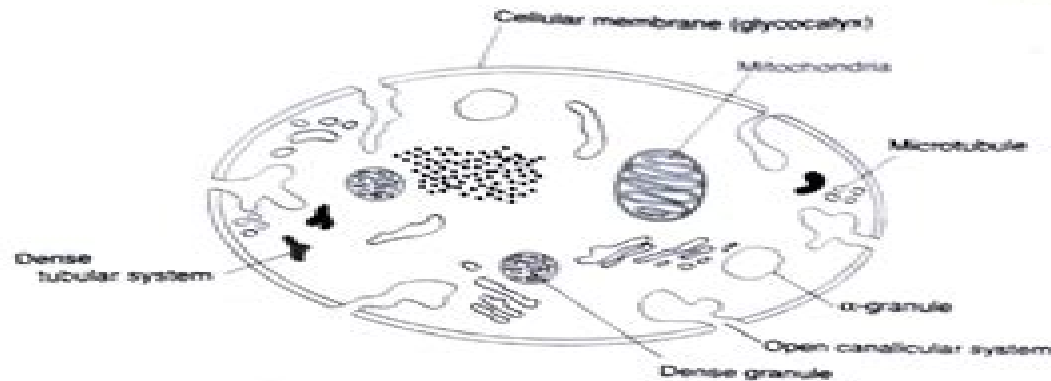




■ **Figure 2-1** Diagram of platelet ultrastructure showing the major intracellular morphologic features. (Reprinted from Bess EC. *Hematology*. Baltimore: Williams & Wilkins; 1992:202 with permission.)

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

C. Platelet function

1. **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)
 - d. Thrombin
 - e. ADP
2. **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.
 - b. Contractions result in a centralization of the cytoplasmic granules and a release of their contents through the canalicular system.
3. **Plug formation**, or secondary aggregation, is primarily stimulated by **thrombin and thromboxane A_2 (TXA_2)**.
 - a. Membrane-binding of vWF and collagen to platelet receptors unmasks membrane **phospholipid A_2** , which is the precursor of arachidonic acid, and is important for the production of TXA_2 and other prostaglandins. **Phospholipid A_2** is unmasked by the binding of vWF and collagen to platelet receptors on the membrane.
 - b. TXA_2 inhibits adenylyate 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.
 - c. **Thrombin** enzymatically cleaves fibrinogen to form **fibrin**, which is necessary to stabilize the platelet plug.

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Yicheng Fang



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