Enzyme / factor /molecule	Function	Location	
(GPIb/IX & GPI/IA)	-bind and interact with collagen (in matrix) and the vWF (Von willbrand factor). (These interactions lead to the activation of the platelets.)	on the cell surface (platelet's surface)	glycoproteins
(GP IIB/ IIIa)	-for the purpose of platelet -platelet interaction, these aid with the aggregation process. (leads to the formation of the platelet plug.)	on the cell surface (platelet's surface)	glycoproteins
• Thrombin	Thrombin receptor activates a G protein that activates phospholipaseC-β (PLC-β). 2- activates Both factors V and VIII via a feedback mechanism. 3- cleaves fibrinogen releasing fibrinopeptides. 4-Platelet recruitment 5- Amplification of the coagulation complex 6- Formation of soft clot -Proteolytic cleavage of fibrinogen 7- Formation of hard clot - Activation of factor XIII 8- Attenuation of its own activity - Activation of protein C 9- Other actions 10- Binding to its receptor on the surface of platelets induces vascular remodeling (e.g. angiogenesis) and inflammation.	released from a platelet's granules, after release it binds to its receptor on the platelet surface.	Protease
thromboxane	1-induces the release of more granules, and thromboxane itself (and prostaglandins) also gets released. 2-is as vasoconstrictor and a further inducer of PLC-B activity (and platelet aggregation) 3-It acts in autocrine and paracrine manners		
Serotonin	vasoconstrictor		
PDGF (platelet derived growth factor)	stimulates proliferation of endothelial cells (thickening of vascular wall) to reduce blood flow		
NSAID / aspirin	1-inhibit the cyclooxygenase (COX) 2-inhibit prostacyclin (This enzyme is a vasodilator and inhibits platelet Aggregation)		

Ca2+	1-binds phospholipase a2 2-activates myosin light chain kinase (MLCK), which phosphorylates the light chain of myosin allowing it to interact with actin and resulting in altered platelet morphology induced motility, and release of granules.		
DAG , PKC	Phosphorylates and activates specific platelet proteins that induce the release of platelet granule contents including ADP.		
ADP	1-drives the formation of platelet plug 2-platelet activator that binds to its receptor and modifies the platelet membrane allowing fibrinogen to adhere to platelet surface glycoproteins resulting in fibrinogen-induced platelet aggregation, called platelet plug.		
An ER/Golgi carboxylase	-binds to prothrombin and factorsIX, VII, and X and converts 10≥ glutamate (Glu) residuesto □-carboxyglutamate (Gla), followed by a small (10 a.a.) hydrophobic region.		
vitamin K	Participates in the conversion of Glu to γ- carboxy-Gla. (Vitamin K becomes oxidized and must be regenerated by a reductase and NADH..)		
Tissue factor	1-It is the primary initiator of coagulation and is not exposed to blood until Disruption of the vessel wall. 2-It increases the proteolytic efficiency of VIIa *in Extrinsic pathway : TF with factor seven (vii) on the surface of platelets * TF with factor (vii) can activate factor nine (ix) in intrinsic pathway	integral membrane protein that is expressed on the surface of "activated" monocytes ,subendothelial cells, and other cells.	
Kallikrein from prekallikrein (PK) In intrinsic pathway	1-Increase activation of factor xii (positive feedback) 2-activate HMW kininogen into bradykinin which is vasodilator 3- Bradykinin is also generated by kallikrein.		
Va and VIIIa	-Increase the proteolytic efficiency of Xa and IXa, respectively.		cofactors(not enzymes)

von Willebrand factor	-increases VIII half-life,		
Factor XIII	-transglutaminase that is activated by thrombin. 1-catalyzes a transglutamination reaction that causes a covalent cross-linking reaction between a glutamine of one fibrin monomer to a lysine of an adjacent fibrin monomer. 2-Produces covalent cross-linking between fibrin molecules which convert platelet plug (soft clot) into hard clot 3-cross-link the fibrin clot to adhesive proteins on the endothelial tissue and to platelet surface strengthening the platelet plug		transglutaminase
Protein C and protein S	• Thrombin(when it binds to thrombomdulin) can then activate protein C, which forms a complex with protein S on the cell surface, both of which are vitamin K-dependent cofactors. • The complex degrades factors V and VIII so it decreases the activation of factor X)		
Antithrombin III	protease inhibitor of thrombin as well as an inhibitor of IXa, Xa, XIa, XIIa, and VIIa when complexed with TF (factor 2,9,10,11,12 and 7)		protease inhibitor
Heparin sulfate	-(important to the binding between thrombin and antithrombin iii) - anticoagulant , which activates the inhibition of clotting process by antithrombin iii	polysaccharide synthesized by mast cells and present on the surface of endothelial cells,	polysaccharide
Tissue factor pathway inhibitor (TFPI)	• It binds to and inhibits factor Xa. • The Xa-TFPI complex then interacts with the TF-VIIa complex and inhibits its activation of factorsX and IX. • TFPI also inhibits Xa-activated Va resulting in inhibition of the pro-thrombinase complex. • Protein S binds to TFPI localizing it to membrane surfaces (of the platelets) and enhancing the inhibition of Xa.	a protein found in plasma lipoproteins and bound to the vascular endothelium.	
Ca2+ chelators (EDTA) and vitamin K antagonists	1-Prevent blood clotting 2- vitamin K antagonists such as the drug warfarin, which inhibits the reduction of vitamin K (active vit k cannot be regenerated so, carboxylase enzymes cannot function) and thereby prevents the synthesis of active prothrombin and factors VII, IX, and X.		

Plasmin	1-protease formed from plasminogen and is responsible for fibrinolysis * won't be activated until it binds to fibrin clot) * Plasminogen activated by tissue plasminogen activator). Which activated by protein C 2- it binds to fibrin and catalyzes its hydrolysis(and degradation of the clot).		protease
• Thrombin activatable fibrinolysis inhibitor (TAFI)	-carboxypeptidase that removes the N-terminal lysine residues and prevents fibrinolysis.		carboxypeptidase
Streptokinase	-regulatory protein isolated from streptococci, allows autoactivation of plasminogen in blood, resulting in degradation of fibrinogen as well as fibrin. - Activates the conversation of plasminogen into plasmin and it helps in removing the clots		
α 2 antiplasmin	-Inhibit plasmin but not when plasminogen/plasmin are clot-bound		
Urokinase	(plasminogen activator) is a protease that is formed from the zymogen pro-urokinase. • It is a potent plasminogen activator and is used clinically.		protease
Role of endothelial cells in coagulation	-ECs release NO, prostacyclin (PGI2), and ADPase, which inhibit platelet adhesion and aggregation. • Membrane-bound heparin sulfate binds to antithrombin III (ATIII) inactivating several coagulation factors. • ECs express tissue factor pathway inhibitor (TFPI), which inhibits tissue factor (TF) and, consequently, factors VII, IX, and X. • Thrombomodulin (TM) binds thrombin activating protein C, which degrades factors Va and VIIIa. • ECs balance fibrin accumulation and lysis by releasing plasminogen activators, t-PA and u-PA, and their inhibitor (PAI).		

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