

GP1BB Human

Glycoprotein Ib Platelet Subunit Beta Human Recombinant

RCP0953

Product Overview

Name	GP1BB Human
Accession(Primary)	P13224
Description	Glycoprotein Ib Platelet Subunit Beta Human Recombinant

Target information (P13224)

Gene Name

About Gene Symbol

Background

Glycoprotein 1b beta (GP1BB) is a crucial component of the platelet glycoprotein Ib-IX-V complex, playing a pivotal role in platelet function and hemostasis. This research aims to explore the structure, function, and implications of GP1BB protein in platelet adhesion, aggregation, and the development of thrombotic disorders. Understanding the molecular mechanisms and regulatory roles of GP1BB can provide valuable insights into its potential as a therapeutic target for thrombotic diseases.

Structure and Expression of GP1BB Protein: GP1BB is a transmembrane protein consisting of an extracellular domain, a single transmembrane region, and a short cytoplasmic tail. It is predominantly expressed on the surface of platelets, where it interacts with von Willebrand factor (VWF) and other components of the glycoprotein Ib-IX-V complex. GP1BB is crucial for mediating platelet adhesion to damaged endothelium and the formation of stable platelet aggregates at the site of injury.

GP1BB and Platelet Adhesion: GP1BB interacts with VWF, which is exposed upon vascular injury. This interaction facilitates the initial attachment of platelets to the damaged endothelium. GP1BB also participates in the formation of high-affinity bonds between platelets and VWF, contributing to the stabilization of platelet

adhesion and the initiation of platelet aggregation. Dysregulation of GP1BB-mediated platelet adhesion can lead to abnormal clot formation and thrombotic disorders. Implications of GP1BB in Thrombotic Disorders: Defects or mutations in the GP1BB gene can result in Bernard-Soulier syndrome (BSS), a rare inherited bleeding disorder characterized by giant platelets, thrombocytopenia, and impaired platelet adhesion. BSS patients often experience excessive bleeding and bruising due to defective GP1BB function. On the other hand, abnormal activation or overexpression of GP1BB has been associated with increased risk of thrombotic events, such as myocardial infarction and stroke. Therapeutic Strategies Targeting GP1BB: Given its crucial role in platelet function and thrombotic disorders, GP1BB represents a potential target for therapeutic interventions. Strategies aimed at modulating GP1BB expression or function have been explored. This includes the development of monoclonal antibodies or small molecule inhibitors that selectively target GP1BB and interfere with its interaction with VWF or other binding partners. Such approaches aim to prevent excessive platelet adhesion and aggregation, reducing the risk of thrombotic events. Challenges and Future Directions: While targeting GP1BB shows promise, several challenges need to be addressed. The development of specific inhibitors or modulators of GP1BB that do not interfere with its physiological functions is crucial. Additionally, understanding the complex interplay between GP1BB and other platelet receptors and signaling pathways is essential for the development of effective therapeutic strategies. Conclusion: The study of GP1BB protein provides valuable insights into its central role in platelet adhesion, aggregation, and thrombotic disorders. Understanding the molecular mechanisms and functional implications of GP1BB opens avenues for the development of targeted therapies for thrombotic diseases. Further research on GP1BB protein, its interactions, and its modulation in pathological conditions will contribute to the development of novel therapeutic interventions to mitigate the risk of thrombotic events and improve patient outcomes.