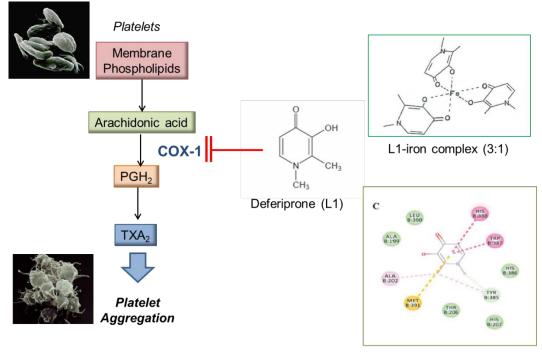
Iron overload and iron chelation therapy

Antiplatelet activity of deferiprone (L1)

Iron overload and thromboembolism are important clinical manifestation in thalassemia. In the past decade, orally active iron chelator, deferiprone (L1) is a drug of choice for iron chelation therapy because of its inexpensive and accessibility due to local production by Government Pharmaceutical Organization (GPO). Deferiprone removes myocardial iron and has been proven to prevent hart complications in thalassemia. Recently, *in vitro* antiplatelet activity of deferiprone has been reported. The results from molecular docking showed that deferiprone interacted closely with key residues in the peroxidase active site of cyclooxygenase-1 (cox-1), suggesting that deferiprone exhibited antiplatelet activity through inhibition of cox-1. Antiplatelet activity of deferiprone may prevent thromboembolism in thalassemia.



Interaction of L1 and COX-1

Reference:

Tran NT, Akkawat B, Morales NP, Pojunckarin P, Luechapudiporn R. Antiplatelet activity of deferiprone through cyclooxygenase-1 inhibition. Platelets, 2020;31:505-512.