CHAPTER V

CONCLUSION

Conclusion

Thalassemia red blood cells are subjected to oxidative stress due to the imbalance of hemoglobin chains to the cytoplasmic side of the plasma membrane leading to destruction of red blood cells and shortened life span, thus causing anemia [1, 2, 3]. The prior studies demonstrated a decrease of thalassemic eryptosis and the death ligand of activation that can induce apoptosis pathway. Thus this work aims to study the role of CD95 receptor on red blood cells sureface can induce apoptosis pathway. The percentage of PS positive cells in normal red blood cells did not increase PS positive cells. Thus CD95 receptor on red blood cells sureface is not functional. Thus these accelerated apoptotic red blood cells may occur through other pathways. Modulation of mechanisms regulating energy supply or oxidative stress impacts eryptotic behavior of red blood cells. In thalassemic blood cells enhanced eryptosis causes anemia. Insulin enhances the flux of glucose by stimulating glycolysis to produce ATP and NADH in human erythrocytes. Thalassemic red blood cells are prone to undergo eryptosis leading to anemia. This study gives evidence that the stimulatory effect of insulin leads to a protection of oxidative-stress-induced eryptosis, especially in red blood cells of thalassemic. Insulin decreased the percentage of phosphatidylserine exposing red blood cells in both, normal and thalassemic red blood cells. In conclusion, insulin plays an important role of reduce apoptosis of both normal and thalassemia red blood cells in oxidative stress

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