

# Platelet Hyperactivity and Dysfunction in Diabetes and Cancer

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## Editorial

However, the entire coagulation cascade is dysfunctional, in progressed chronic diabetes and cancer patients. Platelets (PLTs) in type 2 diabetic (DT2) involved in Thrombosis and Haemostasis (T&H) of individuals adhere to vascular endothelium and aggregate more voluntarily than those in healthy individuals, as are abnormalities in the microvascular and macrovascular circulations. However [1-4] it is already known that the circulating PLTs are essential for T&H, inflammation [3] growth factors delivery, regeneration; and knowledge of their function is fundamental to understanding the pathophysiology of vascular disease in diabetes and cancer-related diseases [2-5]. Though, PLTs significant role as participants in the resolution of thrombo-inflammation is underappreciated [3]. Recently our group has shown that human PLTs' aggregating function/-reactivity affected by advanced glycosylated hemoglobin [6]. Recent studies have also provided strong evidence for an association between diabetes complications and an increase in PLTs' reactivity [2,4,5] Though some metabolic abnormalities have been reported as the major causes of this reactivity and malfunction, which the defined mechanism has not been fully revealed. Intact healthy vascular endothelium play pivotal role in the normal functioning of smooth muscle contractility as well as its normal interaction with PLTs. What is not clear is the role of hyperglycemia in the functional and organic microvascular deficiencies and PLTs (hyper-) activity in individuals with diabetes and cancer-related diseases [4] Increased levels of fibrinogen and plasminogen activator inhibitor 1 favor both thrombosis and defective dissolution of clots once formed [1]. Insulin resistance

is a uniform finding in DT2, as are abnormalities in the microvascular and macrovascular circulations. These complications are associated with dysfunction of platelets and the neurovascular unit [1-4].

Recall, last 15 years studies have shown that Medicine failed to decrease mortality and morbidity rate of 5-years survival chance of Cancer patients. There are so many missing links that One might speculate that what are we doing really about preventing death machineries, which all correlated into PLTs' function and (dis-)functions. Why pharmaceuticals and developed therapeutics still fail to halt so high mortality and morbidity rates in-Hospitals and/or out of Hospitals. One might expect that in the 21st Century all people has right to know about so called "think mode" of policymakers, pharmaceuticals and Basic Research founders' principles, who support Research and Development of new drugs and therapeutics.

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