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Nanomechanics and Structure and Function of Platelets in Patients with Neurological Disorders

Adam Nicholas*

Department of Molecular Biology, University of Berlin, Germany

Introduction

Platelets, which are anucleated circulating blood cells, regulate hemostasis and thrombosis and are involved in inflammatory and pathological processes, all of which are risk factors for neurodegenerative diseases (NDDs). They also help with immunity and communicate with other cells and tissues. Platelets undergo dramatic changes in morphology and size in response to various biological stimuli and agonists, vascular damage, or shear stress, due to cytoskeleton reorganisation, a process known as platelet activation. The first event is a change in cell shape from discoid to spherical, followed by prominent cell-cell interactions and clustering via extending actin-rich philopodia; finally, flattening and spreading of cells on the damaged surface is observed, resulting in the sealing of the impaired vessels. Platelets also play a role in metastasis, inflammation, innate and adaptive immune defences, and embryonic development. As a result, studying platelet activation is critical for understanding the regulation of blood coagulation in both healthy and diseased individuals. This is especially important during pregnancy, which is a hypercoagulable state [1-3]. Platelets contain a large number of bioactive molecules that are stored in dense and alpha granules that are secreted upon platelet activation and mediate platelet function, as well as glycogen granules that provide energy for platelet interactions. Small molecules such as ADP, ATP, polyphosphate, serotonin, and calcium are found in the dense granules.

Description

Fibrinogen, coagulation and growth factors, adhesive molecules, cytokines and chemokines are all stored in the alpha granules. Platelets, in particular, have structural and biochemical properties that are similar to neurons. Platelet granules resemble neuron vesicles, and thus they have been considered a good peripheral model to study neurodegenerative pathologies. Platelets are activated and aggregation occurs in a variety of NDDs, including Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS), and Parkinson's disease (PD). Furthermore, oxidative and physiological stress cause structural and functional alterations as well as platelet activation in a variety of NDDs, including AD, ALS, and Parkinson's disease, in the same way that ageing does.

Platelets are the major source of amyloid trans membrane protein precursor (APP) and provide a large percentage of amyloid peptide (A) in blood plasma, and their contribution to NDDs has been most extensively studied for AD. The concentration of APP isoforms in platelets is comparable to that in the brain, but the expression pattern is different. APP is thought to function as a receptor on the platelet surface and is also important for regulating intracellular Ca^{2+} concentration [4,5]. A was discovered in dense platelet micro vesicle fractions and has the ability to be secreted upon platelet activation. The A peptide variants secreted are the same as those found in the senile plaques of Alzheimer's disease patients. Platelets, on the other hand, primarily release the A (1-40) peptide, whereas neurons produce large amounts of A(1-42) peptides.

Conclusion

APP affects the functions of platelets and the brain, regardless of where it comes from. Changes in the operation of APP, membrane fluidity, cholesterol, serotonin uptake, and intracellular Ca^{2+} levels have been observed in AD platelets. A link was discovered between changes in the ratio of different APP isoforms in platelets and a decline in cognitive skills in patients in the early stages of Alzheimer's disease, implying that the ratio of APP isoforms in platelets is a biomarker for the early stage of Alzheimer's disease. AD family mutations cause hyper activation of circulating platelets, which is visible as the disease progresses. The vascular damage seen in Alzheimer's disease is a natural cause of platelet activation and degranulation. Platelets from Parkinson's and ALS patients have abnormal characteristics as well. The disordered protein synuclein is associated with changes in platelet morphology in Parkinson's disease, and it is overexpressed and found in microvesicles. The pathology is accompanied by platelet hyperactivation and granulation, as well as a significant increase in reactive oxygen species production. In Parkinson's disease, there is also evidence of changes in the ultrastructure of these cells, mitochondrial dysfunction, and elevated glutamate levels.

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***Address for Correspondence:** Adam Nicholas, Department of Molecular Biology, University of Berlin, Germany, E-mail: AdamNicholas3@gmail.com.*

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