

Mitochondrial DNA Enhances Adrenaline-Induced Platelet Activation in Type-II Diabetes Mellitus

Abstract category: Hemostasis, transfusion medicine, vascular, laboratory medicine,

Durre Shehwar¹, Alessandro Aliotta¹ Madeeha Rasool², Samra Fatima², Saima Barki², Debora Bertaggia Calderara¹, Lucas Veuthey¹
Cindy Pereira Portela¹, Muhammad Rizwan Alam², Lorenzo Alberio¹

1. Division of Hematology and Central Hematology Laboratory, Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), Lausanne, Switzerland

2. Department of Biochemistry, Quaid-i-Azam University, Islamabad, Pakistan

INTRODUCTION

Diabetes, a chronic metabolic disorder associated with number of complications, is one of the major cause of morbidity and mortality worldwide. Diabetic patients have hyperactive platelets, which are more prone to agonist-induced aggregation. It is known that oxidative stress in diabetic patients affects mitochondrial function, which releases mitochondrial DNA (mtDNA) and contributes to platelet activation. However, whether or not cell-free mtDNA, can prime the adrenaline-induced platelet aggregation remains incompletely explored.

AIM

The aim of the study is to explore the priming effect of mtDNA on adrenaline-induced activation of platelets.

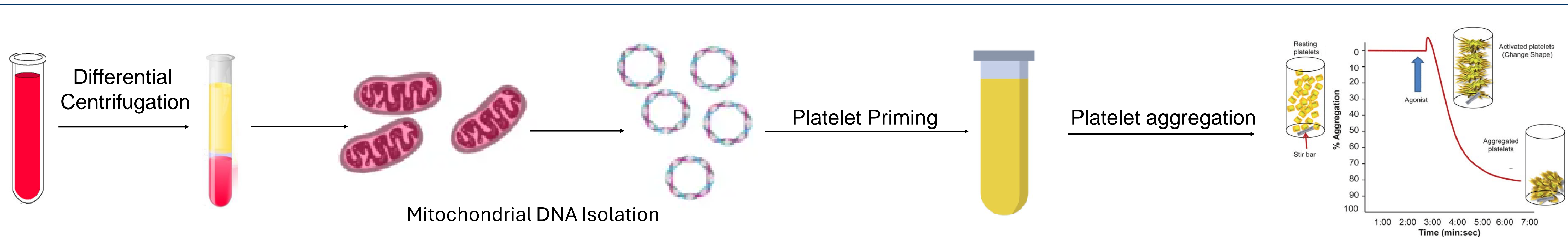
CONCLUSIONS

mtDNA Priming

Accelerates adrenaline-induced aggregation, which could reflect the pathophysiological condition in diabetic patients.

Enhances platelet reactivity and that this may be hampered by metformin

METHOD



RESULTS

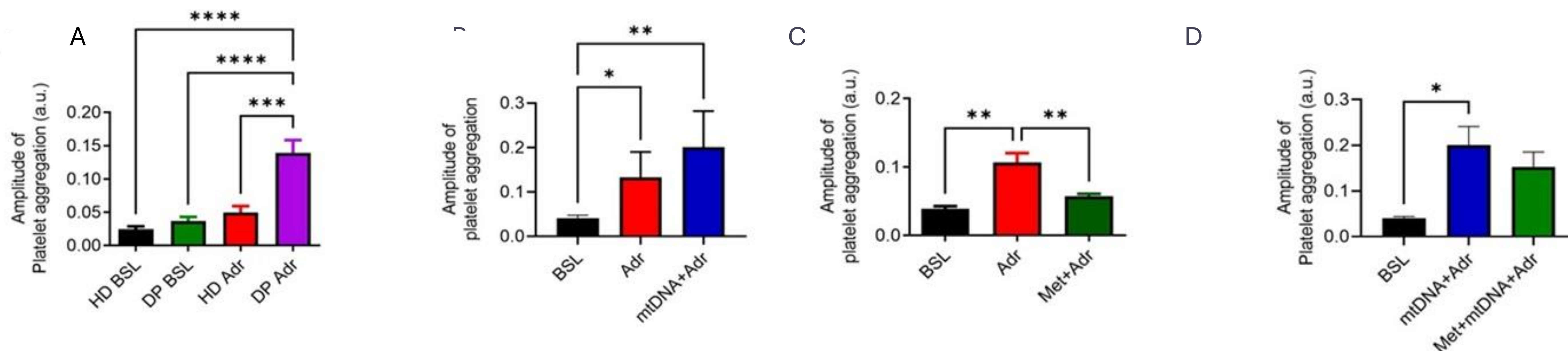


Figure 1. Mitochondrial DNA (mtDNA) enhances adrenalin-induced platelet aggregation. (A) Increased aggregation of platelets stimulated by adrenaline (Adr) in diabetic patients (DP) vs healthy donors (HD) and compared to unstimulated baseline (BSL). **(B)** mtDNA priming enhances adrenaline-induced aggregation in platelets from HD. **(C)** Metformin (Met) reduces adrenaline-induced aggregation in platelets from HD. **(D)** Met reduces the trend in mtDNA primed adrenaline-induced platelet aggregation in HD platelets. Data are mean \pm SEM from n = 4.

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CONTACT INFORMATION

Durre Shehwar: durre.shehwar@chuv.ch
Alessandro Aliotta: alessandro.aliotta@chuv.ch
Lorenzo Alberio: lorenzo.alberio@chuv.ch
Muhammad Rizwan Alam: mr Alam@gau.edu.pk