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Title of diploma thesis: Platelet aggregation in patients with type 2 diabetes mellitus

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by impaired glucose metabolism, which often leads to vascular complications and an increased risk of thrombotic events. One factor contributing to this risk is altered platelet reactivity. The aim of this study was to analyze and evaluate how platelets from patients with type 2 diabetes respond to various agonists compared to the platelets from healthy individuals. *Ex vivo*, inducers acting through distinct signaling pathways were tested to more comprehensively map the mechanisms of platelet activation: arachidonic acid (AA), collagen, ADP, thromboxane A_2 analogue (U-46619), ristocetin, and thrombin receptor-activating peptide (TRAP). Aggregation was measured using impedance aggregometry in fasting blood samples obtained from 45 patients with diabetes and 53 healthy volunteers. The results showed that platelet responses in diabetic patients are heterogeneous. Statistically significant differences were observed for only three inducers. Higher aggregation was observed in samples stimulated with AA and ristocetin, whereas collagen-stimulated samples exhibited lower aggregation in diabetic patients. These findings suggest that overall platelet reactivity in patients with diabetes is not systematically increased, but there is a targeted effect on specific platelet signaling pathways.