Platelets are the smallest blood cells.They play a crucial role in the maintaining of homeostasis after vascular injury by participating in the process of thrombus formation. Formation of platelet plug is followed by platelet activation in response to appropriate stimulation, like the contact with collagen of distrupted endothelial layer. However, excessive platelet activation and reactivity (ability to respond to stimulating factor) under normal conditions (without blood vessel injury) may contribute to atheroma formation and lead to micro- and macrovascular diseases. Abnormal platelet activation is often observed diabetes mellitus and may lead to the development of cardiovascular diseases, which cause the death of almost 80 percent of diabetic patients. However, some mechanisms underlying the abnormal platelet activation and their hypersensitivity in this condition remain obscure. The key role of energy in platelet activation makes mitochondria (site of energy production in the cell) a potential regulator of platelet function, and therefore also a potential target for antiplatelet therapy. It seems highly probable that the prolonged exposure of platelets to high concentrations of glucose may lead to changes in the functioning of mitochondria in these cells, especially considering the fact that glucose is the main energy source for blood platelets. The reason of such alternation is high glucose concentrations inside diabetic blood platelets which may lead to increased substrate delivery to mitochondria and accelerated work mitochondria, including elevated oxygen consumption by the mitochondria and increased mitochondrial membrane potential.
Therefore, the aim of this study is to assess the impact of long-term impaired carbohydrate metabolism in diabetes on platelet mitochondria respiration and mitochondrial membrane potential and to evaluate the associations between platelet mitochondrial respiration or mitochondrial membrane potential and platelet activation markers.