Diabetes mellitus type 1 (T1D) is characterized by hyperglycaemia resulting directly from inadequate insulin secretion because of pancreas destruction. Approximately 78 000 children develop T1D each year, but T1D may develop at any age. The epidemiological studies have shown that the risk increases from birth with peaks at 10–14 years. The long term hyperglycaemia in poor controlled T1D patients leads to kidney injury resulting in decrease of renal ability to blood filtration and increased albumin excretion with urine. Kidneys play a key role in human homoeostasis and about 20% of renal blood flow is filtrated in glomeruli through multilayers filter maintained by endothelial cells of glomerular capillary, basement membrane and podocytes. These cellular and acellular structures forms glomerular filtration barrier (GFB) which is fully permeable for water and electrolytes (e.g. ions of sodium, potassium, bicarbonate) and small molecular weight molecules (e.g. urea, creatinine) but is not permeable for proteins (e.g. albumin) with molecular weight greater than 70 kDa. The properties of GFB are modified by activation of receptors, including heterogeneous group of purinoceptors P2 (P2Rs) activated by extracellular nucleotides (e.g. ATP, ADP, UTP, UDP and carbohydrate nucleotides), located on cells forming GFB and other glomerular cells e.g. mesangial cells. Nucleotides are released from cells into extracellular fluid in constitutive manner and after biochemical and/or pharmacological stimulations. In extracellular fluid, nucleotides are sequentially metabolised which finally leads to generation of metabolites which do not activate P2Rs and finally to attenuation P2Rs activity.

Hyperglycaemia leads to enhancement of GFB permeability for albumin and, in turn, to increased albumin excretion in urine – albuminuria. Albuminuria is a symptom of diabetic nephropathy but also is a key factor influencing the progression of renal dysfunction which may be, to some extent, pharmacologically modified. Hyperglycaemia is, modifiable factor involved in the development and progression of renal failure. Due to the fact that diabetic nephropathy may finally lead to renal insufficiency and then these patients receiving high-cost renal replacement therapy, thus it is worth unveiling dysfunctional regulatory mechanisms underlying disturbances in glomerular permeability for albumin and especially the role of purinome activity in this process under conditions of hyperglycaemia in diabetes type 1 patients.