Kidneys play a key role in human homoeostasis. Under physiological conditions 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 (ions of sodium, potassium, bicarbonate) and small molecular weight molecules (urea, creatinine) but is not permeable for proteins (albumin) with molecular weight greater than 70 kDa. Many biological factors affect the physicochemical properties of GFB by membrane receptors activation (angiotensin II) or direct action on intracellular effectors (reactive oxygen species, ROS).

Congenital or acquired structural distortions or dysfunctions of specific layers of GFB lead to enhancement of GFB permeability for albumin and, in turn, to increased albumin excretion in urine – albuminuria. The dysfunction of GFB is mainly dependent on disturbances of podocytes; podocytes layer is characterised by the lowest permeability for albumin and their very low ability to regeneration. Albuminuria is one of laboratory symptom of chronic kidney disease (CKD) but also is a key factor influencing CKD progression which may be, to some extent, pharmacologically modified. CKD affects about 6-15% of population, by extension over 4 mln people in Poland suffer from CKD. The hypercholesterolemia is an another, modifiable factor involved in the development and progression of CKD and development of cardiovascular diseases. Furthermore, it is estimated that hypercholesterolemia affects about 61% people in the age range 18-79 years in Poland. Hypercholesterolemia leads to imbalance between generation and utilization of ROS - oxidative stress, and this situation leads to covalent modification of proteins and lipids, among others, low density lipoprotein (LDL) are oxidized (oxLDL). These modified lipoproteins bind to a family of scavenger receptors localized also on podocytes and endothelial cells and affect cell function and initiate immunological response. Except that, oxLDL acts via no-receptor route and disrupt the structure of cell membranes. Thus, interaction between oxLDL and glomerular filter that leads to increased permeability to albumin is crucial for understanding the pathogenesis of chronic renal disease. Due to the fact that CKD may lead to renal insufficiency and then these patients receive a high-cost renal replacement therapy, it is worth unveiling dysfunctional regulatory mechanisms underlying disturbances in glomerular permeability for albumin and especially the role of oxLDL in this process under conditions of hypercholesterolemia.