

Abstract:

Uric acid is a main metabolite of purine degradation in humans and in higher primates. Its increased plasmatic level is called hyperuricemia and may be the cause of gout and many other similar diseases. Uricemia is controlled by many transporters, which are located in proximal tubule of human kidney. When some transporter have abnormal function, the physiological plasmatic level of uric acid may be impaired. In genome wide association study (GWAS) it was discovered that some hyperuricemia or gout patients have ABCG2 protein damaged. This protein carries out uric acid from epithelial cell to the urine. The goal of this diploma thesis is the determination of transport capacity of ABCG2 allelic variants found via GWAS (Institute of Rheumatology of 1st medical faculty UK in Prague) in vitro with *Xenopus laevis* oocyte expression system. Uric acid secretion was compared with wild type variant.

Keywords:

Uric acid, GWAS study, *Xenopus laevis*, membrane transport protein, ABCG2