This thesis focuses on the importance of intermediate products of biosynthetic and catabolic pathway of cholesterol. The aim of the first part of the thesis is mainly to investigate, whether statins (HMG-CoA reductase inhibitors) possess antitumor properties and to compare the differences in antitumor potential of individual statins.

The other part of the thesis aims at the utilization of 7α-hydroxycholest-4-en-3-one (C4), a promising marker of cholesterol 7α-monooxygenase (CYP7A1) activity and bile acid malabsorption.

We demonstrated antitumor effect of statins on an experimental model of pancreatic cancer. Individual statins, however, differed significantly in their efficacy, depending on their physico-chemical properties. Our data suggests, that the most likely (but not the only) mechanism of antitumor effect of statins is decreased prenylation of signaling proteins, especially Ras protooncogene.

We set up a reliable method for measurement of C4, which facilitated our research in CYP7A1 regulation. We demonstrated, that promoter polymorphism -203A>C might affect CYP7A1 activity, that diurnal variability of CYP7A1 activity might be triggered by insulin, and that insulin resistance in patients with non-alcoholic fatty liver disease impedes the feedback regulation of CYP7A1, which may lead to disease progression. Finally, we demonstrated the importance of laboratory investigation of bile acid malabsorption in Crohn's disease patients.