Disulfide bridges play a significant role in protein-folding as well as enzyme activity and thus regulate many intra- and extracellular processes.

Sulfhydryl oxidase QSOX1 forms S-S bridges *de novo*, modulating the activity of its substrates and thus directly or indirectly influences vital cellular processes. The first part of this thesis focuses on characterization of the role of QSOX1 in cancerogenesis, using breast cancer cell lines (MCF7, MDA-MB-231) and pancreatic cancer cell line (Panc-1), while the second part emphasizes the regulation of *QSOX1* expression by different oxygen concentrations.

To study the effect of QSOX1 on proliferation of triple-negative cancer cells MDA-MB-231, two genetically modified cell lines – *QSOX1*-overexpressing and *QSOX1* knockout cell lines – were constructed. While increased QSOX1 protein levels do not have a significant effect, the absence of QSOX1 leads to a decreased cellular growth. Lack of *QSOX1* also results in visible change in cellular morphology. *QSOX1* knockout cells can be mostly characterized as more round-shaped with less noticeable or completely missing lamellipodia. This finding is with agreement with to-date literature suggesting that QSOX1 is important not only for cellular proliferation but also for migration and invasiveness.

While authenticating the theory of QSOX1 being regulated by atmospheric oxygen concentration via hypoxia-responsive elements included in *QSOX1* promoter, we found that tested cells responded to hypoxia by an increase at the level of *QSOX1* mRNA, there was almost no change on the protein level within the cells, yet, the hypoxic conditions led to a significant secretion of QSOX1 into the media.

These findings support the QSOX1 as a putative target for development of anti-neoplastic drugs and confirms its important role in cancerogenesis.

Keywords: QSOX1, cancer, sulfhydryl oxidase, proliferation, hypoxia