Abstract of the Project of Dr. med. Samuel Huber

Epithelial cell proliferation is important for wound healing. But it can be pathogenic and increase the risk for cancer, if it is uncontrolled. Accordingly, chronic mucosal inflammation and epithelial damage, like in inflammatory bowel disease, increase the risk for colon cancer. IL-22 has pro-proliferative and anti-apoptotic effects on epithelial cells. This is beneficial during tissue repair, but can also cause epithelial hyperplasia and intestinal pathology. Therefore IL-22 needs to be controlled. Of note, there is a soluble IL-22 receptor (IL-22 binding protein, IL-22BP), which specifically binds and neutralizes IL-22. Interestingly, IL-22BP shows an inverse expression compared to IL-22 during wounding in the colon. Therefore IL-22BP might be the factor, which controls IL-22. However, the cellular source, the mechanism of regulation, and the role of endogenous IL-22BP during tissue regeneration and colon carcinogenesis are unknown. We hypothesize that the IL-22 - IL-22BP regulation represents an endogenous mechanism controlling intestinal epithelial cell homeostasis.