



Samenvatting van het proefschrift

Yi Li

"The role of SOCS3 signaling in ulcerative colitis and ulcerative colitis related carcinogenesis"

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Inflammatory bowel disease (IBD) represents a group of idiopathic chronic relapsing in ulcerative colitis (UC) and Crohn's disease (CD). Patients with IBD are at increasing risk for developing colorectal cancer, which can progress through stages of colitis with no dysplasia, low-grade dysplasia and high-grade dysplasia, to ultimately invasive carcinoma. The major molecular carcinogenic pathways, which lead to sporadic colorectal cancer, namely chromosomal instability, microsatellite instability, and hypermethylation, also occur in colitis-associated colorectal carcinoma. It is known that inflammatory and carcinogenic systems are controlled by multiple cytokines. The suppressor of cytokine signaling (SOCS) is a family of intracellular proteins, some of which work as key regulators of cytokine-mediated homeostasis. They make their biological functions through JAK and STAT transcription factors, and would express abnormally when activated in specific microenvironment. The SOCS proteins have been found to be involved in some disorders, such as growth hormone resistance, chronic renal failure and rheumatoid arthritis. But very few details are known about the role of SOCS family in the process of IBD and IBD related carcinoma. The aim of this project is to investigate how SOCS express and to evaluate their effects towards other cytokines in inflammatory and carcinogenic systems of IBD. In my PhD research, we explored the function of SOCS3 in colon biopsies, particular in colon epithelial cells from patients of ulcerative colitis. We have focused our research on understanding the gene/protein expression, immune-regulatory effects of the hot pathway: SOCS3-STAT3-IL-6, approaching the new mechanism and therapy for ulcerative colitis and ulcerative colitis-related colorectal cancer. ◀