PROPOSITIONS

TO THE THESIS

“THE ROLE OF SOCS3 SIGNALING IN ULCERATIVE COLITIS AND ULCERATIVE COLITIS RELATED CARCINOGENESIS”

BY YI LI

1. The high expression of SOCS3 in inactive UC compared with non-disease controls suggests the involvement of this protein in UC development. (This thesis)

2. The loss of SOCS3 expression in UC-CRC may be a critical factor in the progression towards CRC development. (This thesis)

3. When IL-6 signaling results in DNMT1 expression, SOCS3 induction and consequently inhibition of IL-6 signaling through STAT3 are lost. Thus DNMT1 emerges as a rational target in preventive strategies aimed at counteracting UC-CRC. (This thesis)

4. STAT3-independent SOCS3 induction in inactive UC involves multiple pro-inflammatory signaling pathways, which is arguing the pathway-specific and broad-spectrum anti-inflammatory drugs for preventing UC relapse. (This thesis)

5. The subgroup of patients with high SOCS3 expression may have more vulnerable intestinal epithelial cells during remission thus is involved in the induction of relapse. (This thesis)

6. IL-6 signaling pathway would be an interesting therapeutic target in active UC and UC-CRC.

7. The restoration of SOCS3 expression suppresses IL-6/p-STAT3 signaling induced proliferation and survival of epithelial cells in CRC.

8. SOCS3 seems to impair IEC homeostasis in UC but behave friendly to UC-CRC.

9. Don't part with your illusions. When they are gone you may still exist, but you have ceased to live. (Mark Twain)

10. Happiness lies not in the mere possession of money; it lies in the joy of achievement, in the thrill of creative effort. (Franklin Roosevelt)

11. At twenty years of age, the will reigns; at thirty, the wit; and at forty, the judgment. (Benjamin Franklin)