

## M2 master internship project

### *Impact of complement-dependent inflammation in the development of colorectal cancer*

**Laboratory of Pathogens and Host Immunity (LPHI)**

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Chronic inflammation acts as a key driver of CRC development, as highlighted by the increased risk of developing colon cancer in people suffering from inflammatory bowel diseases. Indeed, prolonged activation of key immune effectors of the inflammatory response generates a microenvironment conducive to neoplastic transformation. Among these effectors, complement anaphylatoxin C5a and its receptor C5aR are already known to promote CRC progression, and their elevated levels in CRC patients correlate with shortened survival. C5a and C5aR have recently been described as major contributors to CRC initiation, but the precise mechanisms by which this induction occurs remain elusive.

The goal of this master project is to gain insights on how C5aR immune activation contributes to CRC spread, angiogenesis and metastasis, by using the well-established model of CRC xenografts in zebrafish larvae combined with loss-of-function or gain-of-function approaches to selectively block or enhance C5a/C5aR-dependent signaling. The underlying innate immune mechanisms will be explored in detail by intravital imaging with confocal microscopy and by targeted transcriptomics, drawing on the team's expertise in this field and on the different transgenic reporter lines available to study the dynamics of recruitment and activation of macrophages and neutrophils in the tumor microenvironment.

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