Universität Zürich[UZH]

Institute of Anatomy Zurich

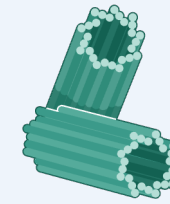
Melanoma initiation and metastasis formation - the secrets that the transcriptome cannot tell

The Sommer Lab is located on the Irchel Campus of the University of Zürich. The melanoma research branch of the lab focuses on the tumour translatome and proteome — layers of regulation that are frequently overlooked by transcriptomic studies but are increasingly recognized as critical drivers of cancer progression.

Project 1: NGFR and Centrosome–Cilium Coupling in Melanoma

NGFR (CD271, p75NTR) is a key marker of the dedifferentiated, neural crest-like state in melanoma. Melanoma cells can switch between proliferative and invasive states. The invasive state is associated with high NGFR expression and contributes to therapy resistance.
Primary cilia are progressively lost during melanoma progression. This has been linked to EZH2-mediated repression of ciliary genes. However, transcriptional regulation may not fully explain cilium loss. Centrosomes are essential for primary cilium formation and are frequently altered in cancer. Their role in melanoma cilium loss remains poorly understood.
This project investigates whether NGFR affects centrosome–cilium coupling in melanoma. We use doxycycline-inducible NGFR expression in patient-derived melanoma lines. Quantitative imaging and centriolar-bait proteomics will be used to identify changes in the centriolar protein network

What you will do
· Culture and characterize patient-derived melanoma lines with inducible NGFR expression
· Quantify ciliogenesis by immunofluorescence
· Perform loss-of-function and rescue experiments
· Test pharmacological NGFR inhibition
· Validate candidate centriolar regulators by western blot and
· Integrate experimental results with existing in-house IP/proteomics data



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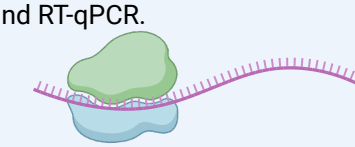
Start date: Flexible

Project 2: The role of translational control in melanoma tumorigenesis

Melanoma is the leading cause of death among skin-related diseases, and its incidence is particularly high in Western countries. As the largest organ in humans, the skin is highly susceptible to melanoma, and identifying the driver genes involved in its initiation and progression is crucial. Our laboratory previously developed hyperproliferative mouse melanocyte cell lines with $NRas^{Q61}$ and $Cdkn2a^{KO}$ mutations (RIH cell lines). These cell lines do not exhibit tumorigenic properties after transplantation into immunocompromised mice, yet overexpression of EZH2 induces tumorigenicity. However, the molecular mechanisms driving this transformation remain partially unclear. A major focus of our current research is understanding translational control in tumour cells compared to premalignant melanocytes. Recent studies suggest that many cancer processes are regulated at the translational level, with increased protein synthesis and ribosome content driving rapid cancer cell proliferation. While translational control in tumors has been investigated, the mechanisms underlying melanoma initiation remain poorly understood. This project aims to investigate the molecular mechanisms driving melanoma initiation on the translational control level.

Methods to be used: bioinformatics (mainly R), cell culturing, genetic modification of mammalian cells, cloning and virus production, mouse experiment, immunofluorescent staining and RT-qPCR.



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Start data: earliest from May 2027