# Transcriptional and epigenetic regulation of breast cancer

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## Gruppens kerneforskningsområder

Transcription is a key determinant of cell function, and deregulation of this process is a key driver of diseases such as cancer. Cancer cells often become dependent on these alterations in the transcriptional and epigenetic mechanisms controlling gene expression, and they are consequently termed transcriptional and epigenetic addictions.

We are enthusiastic about uncovering the transcriptional and epigenetic mechanisms controlling cancer cell biology, particularly in the context of breast cancer. Our research is centered around the transcription factors and coregulatory proteins that work together to regulate expression of our genes and thereby control breast cancer biology. Our overall goal is to provide fundamental insight into how breast cancer growth, metastasis and treatment resistance are controlled at the genomic level and use this information to identify new targeted therapeutic opportunities to inhibit progression of this disease.

**Methods**

1. Functional genomics and proteomics analyses, e.g., next-generation sequencing.
2. Genome editing approaches using CRISPR.
3. Single-cell analyses.
4. Cell lines, mouse models and patient samples.
5. Computational analyses of omics data.

Er du interesseret i at skrive projekt i gruppen, så kontakt :
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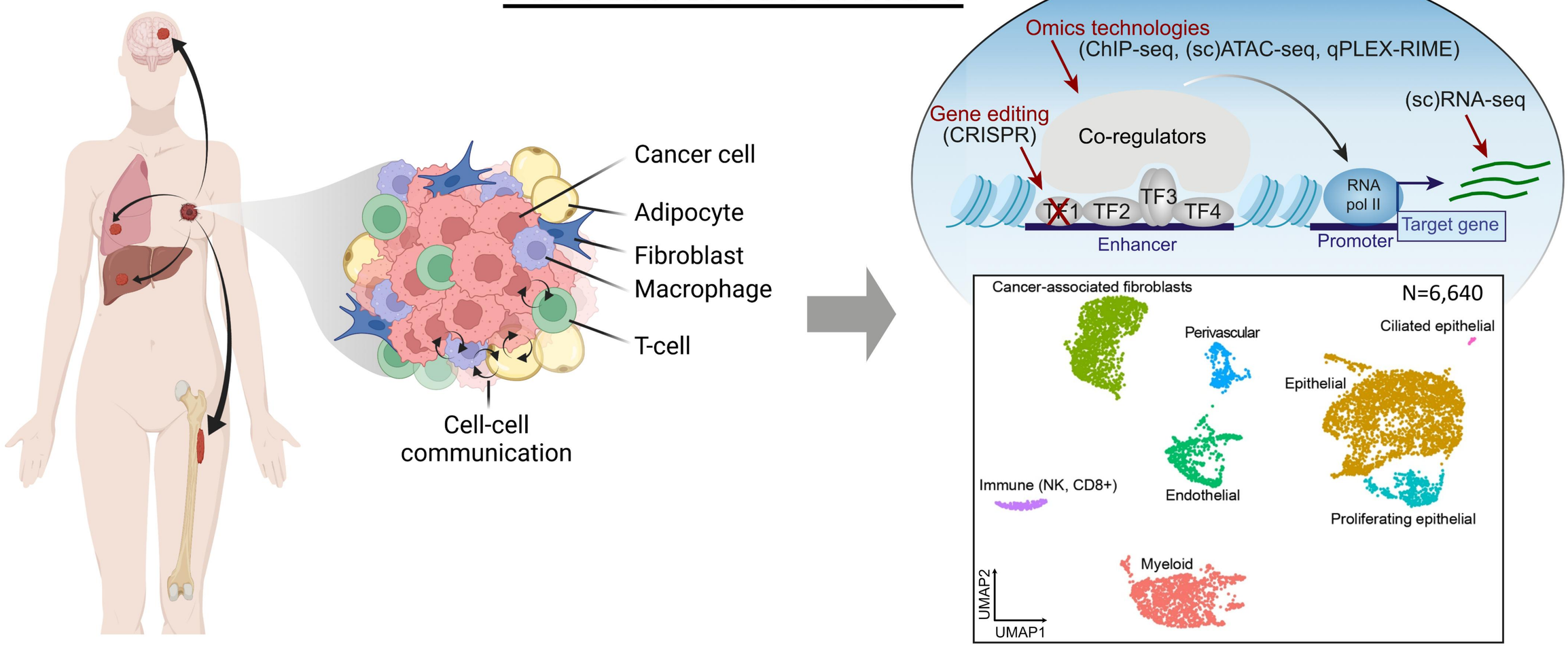
Functional Genomics & Metabolism

SDU

## Research overview




| Projekter | Beskrivelse |
|---|---|
| **The role of MYC in regulating cancer biology** | MYC is a key oncoprotein controlling many different aspects of cancer cell biology. It works primarily as a transcription factor but also has additional non-transcriptional functions in regulating the cellular response to stress. We use a range of omics approaches, including single-cell technologies, to understand how MYC operates within cancer cells and how alterations in MYC function affects cancer cell biology. |
| **Estrogen receptor function in breast cancer** | Estrogen receptor (ER) is a transcriptional driver in most breast cancers and a key therapeutic target. It cooperates with many different transcriptional and epigenetic regulators (including MYC) to promote growth in response to estrogenic hormones. We use functional genomics and single-cell approaches to understand this crosstalk between ER and other pathways and investigate how it impacts growth, metastasis and treatment responses. |
| **Obesity-associated breast cancer** | Obesity has been linked to increased incidence and aggressiveness of breast tumours. This is likely mediated by altered paracrine signalling between cancer cells and the normal cells in the tumour microenvironment (e.g., immune cells, fat cells and fibroblasts). To understand how obesity impacts the transcriptional and epigenetic mechanisms controlling breast cancer biology, we are analysing patient tumours using functional single-cell genomics approaches. |