Exploring the dynamics of cell fate in cancer evolution
Welcome to Cell Fate Dynamics Group

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Group Research Focus

Cells constantly face challenges—from stress signals to metabolic fluctuations—that shape their destiny. In our lab, we study how **replication stress (RS) and metabolism interact to drive senescence**, a state where cells stop dividing but remain active. While senescence can act as a barrier against cancer, some cells find ways to **escape this arrest**, leading to aggressive, therapy-resistant tumors.

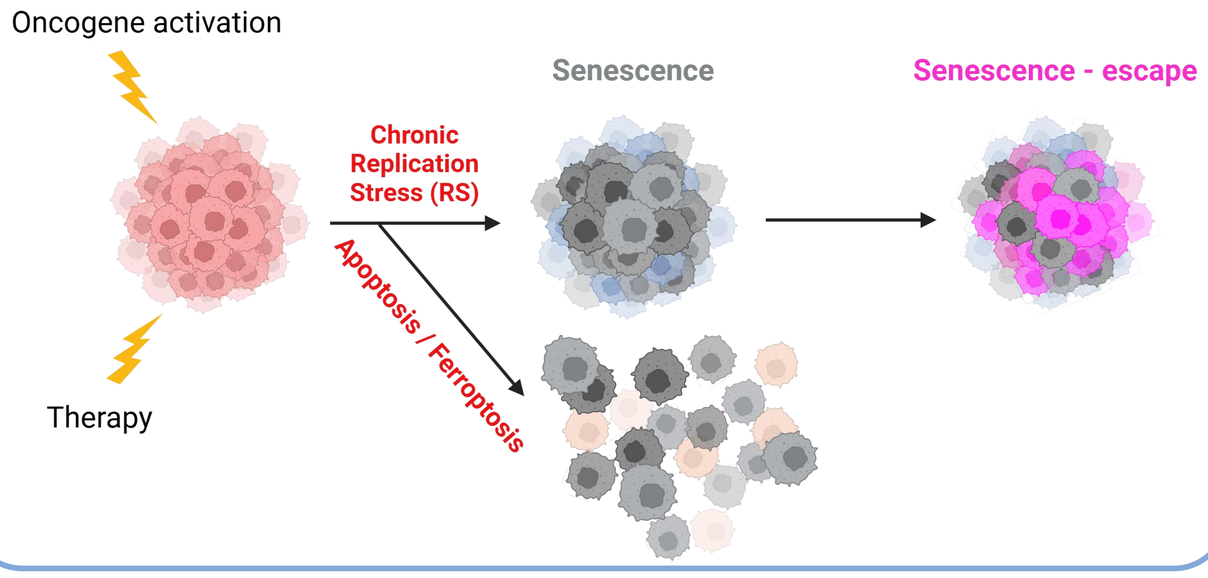
Our research focuses on:

🔬 **How chronic replication stress rewires metabolism** to maintain the senescent state.
🧬 **The molecular switches that allow cells to escape senescence** and fuel disease progression.
💊 **Targeting these vulnerabilities** to prevent cancer cells from becoming more aggressive.

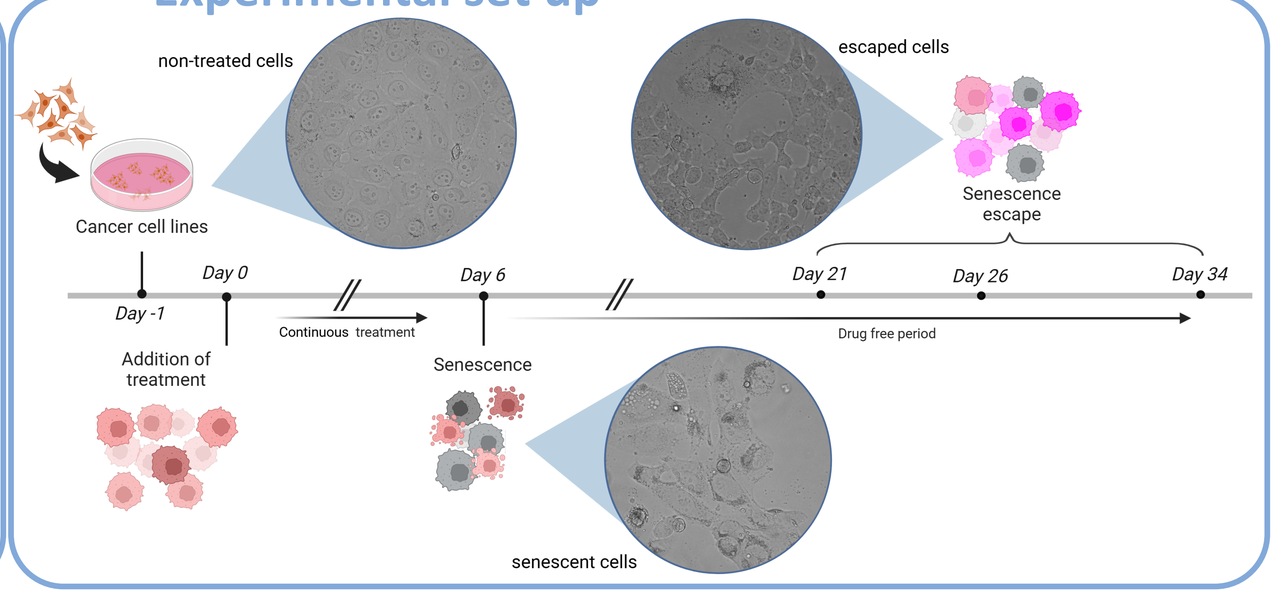
By combining high-content imaging, metabolic profiling, and functional genomics, we **decode the mechanisms that lock cells in senescence—or set them free.** Understanding these processes could lead to **new therapeutic strategies** that stop cancer cells before they evolve into resistant, more dangerous forms.
Join us in exploring how cell fate decisions shape disease—and how we can control them! 🚀

If you are interested in joining the Galanos Group, please contact Panos: panos@bmb.sdu.dk 📩

Model of cancer evolution



Experimental set up



Projects

1 Metabolism & Senescence
How do changes in **metabolism** help cells stay in senescence?

2 Escape from Senescence
What makes cells **escape senescence** and become aggressive?

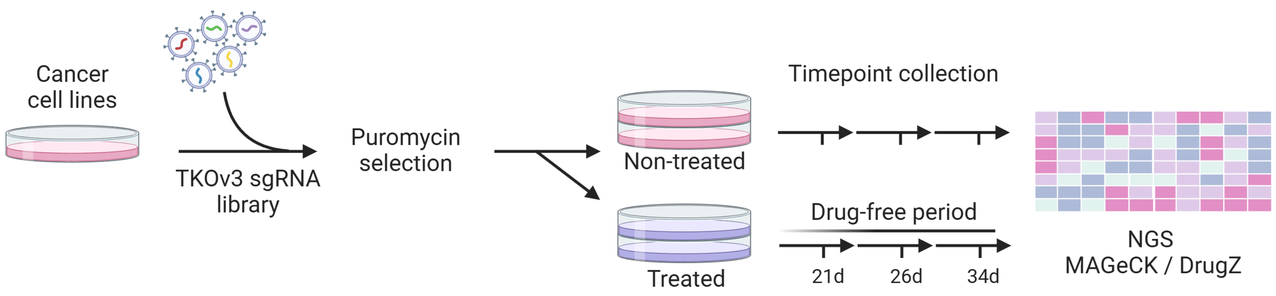
3 Targeting Senescent Cells
Can we **block senescence escape** to stop cancer from becoming resistant to therapy?

Why It Matters?

By understanding how **senescence works** and how cells escape it, we can find **better ways to treat cancer** and prevent relapse.

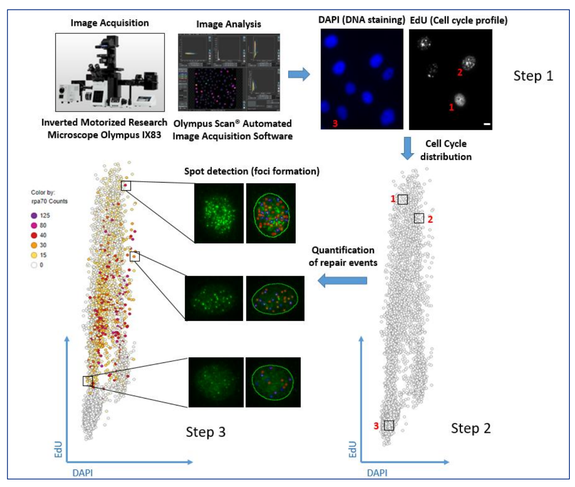
Technical approaches

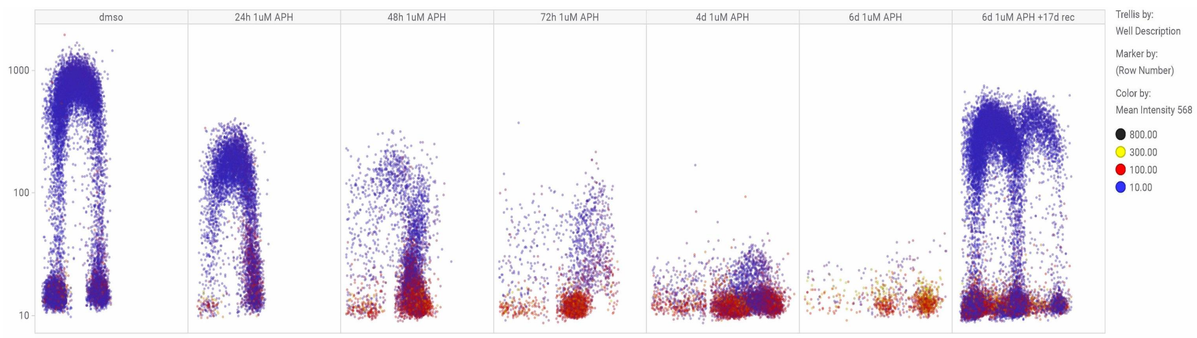
A



A genome-wide CRISPR/Cas9 knockout screen is used to identify key regulators of senescence maintenance and escape. Cells are induced into senescence, transduced with a pooled sgRNA library, and monitored for survival and escape. Enriched sgRNAs in escaping cells reveal potential drivers of senescence bypass, which are validated using functional assays.

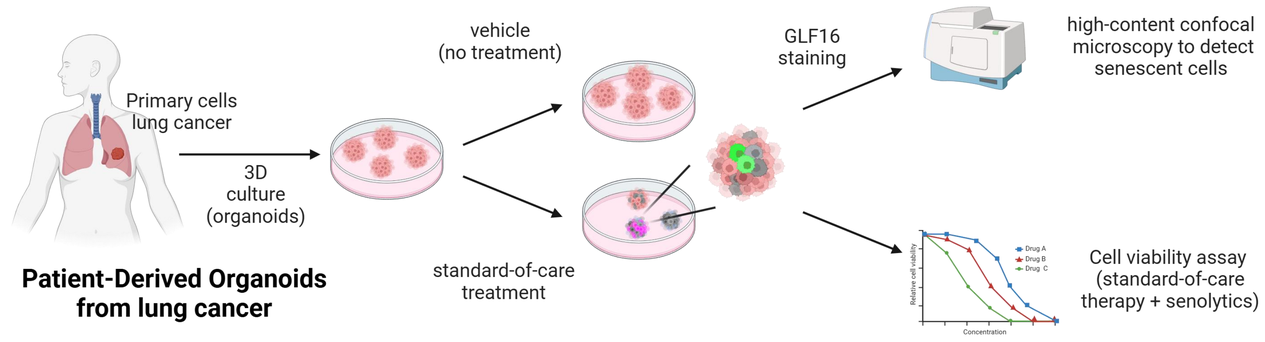
B





High-content automated microscopy is applied to detect RS and DNA damage events with the surrogate markers, coupled with the cell cycle distribution by measuring EdU incorporation.

C



Patient-Derived Organoids (PDOs) provide a clinically relevant model to study senescence and therapy resistance. Lung cancer PDOs are established from resected tumors, treated with standard-of-care therapies, and analyzed for senescence markers. This system allows us to test new strategies to block senescence escape, preventing tumor progression and therapy resistance.

Available Reading Material

1.Chronic p53-independent p21 expression causes genomic instability by deregulating replication licensing. **Nat Cell Biol (2016)**
2. Mutational signatures reveal the role of RAD52 in p53-independent p21-driven genomic instability. **Genome Biology (2018)**
3. A recurrent chromosomal inversion suffices for driving escape from oncogene-induced senescence via subTAD reorganization. **Molecular Cell (2021)**
4. Induction of APOBEC3 Exacerbates DNA Replication Stress and Chromosomal Instability in Early Breast and Lung Cancer Evolution. **Cancer Discovery (2021)**
5. A fluorophore-conjugated reagent enabling rapid detection, isolation and live tracking of senescent cells. **Molecular Cell (2023)**