

Friday, November 3rd, 2023 at 14:00

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How does 'form-follows-function' in therapy-induced cancer cell senescence?

Quantifiable changes in cell morphology are one of the most commonly used observable biomarkers of (pre-)cancerous lesions in epithelial tissues and organs. One noticeable morphological alteration is the stereotypic increase of the whole-cell size in tumor cells undergoing nuclear expansion due to polyploidization. Such enlarged cells are thought to experience senescence/aging associated with permanent cell cycle arrest and functional decline. This is why, entire generations of medical students and biology majors for decades were taught to ignore these cells in functional studies as they are the cells in the process of degeneration. For this reason, inducing senescent polyploid cancer cells was also considered a desired outcome during chemotherapeutic interventions. However, recent studies show that these cells can seed tumor relapse upon chemotherapy withdrawal, suggesting that senescent polyploid cancer cells are capable of evading classic senescence programs. The molecular and cellular mechanisms of this evasion remain unclear partly due to lack of knowledge about the fundamental biology of cancer cells in a therapy-induced senescence (TIS) state. In my talk, I will describe our current research program addressing whether TIS cancer cells scale the rate of metabolic reactions according to their size, and whether it is beneficial or detrimental when it comes to survival under stress.

Venue: CCR Lecture Hall, Borschkegasse 8a

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Host: Iros Barozzi

