Cologne Evolution Colloquium

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The chronology and tissue specificity of FH-deficient cancer

The role of mitochondrial dysfunction in cancer has been debated for over a century. The discovery that core metabolic the of enzymes mutations in mitochondria, such as Fumarate (FH). Hydratase renal strongly indicates that cancer cause mitochondrial dysfunction can drive cancer. Today, I will provide an overview of our recent findings about molecular mechanisms through the which mitochondrial dysfunction can drive transformation. In particular, using a novel genetically modified mouse model, I will show that FH loss has different outcomes in different tissues, and whilst the kidneys are very robust to FH loss, other tissues don't tolerate FH loss, and here, FH-deficient cells are negatively selected. Our work provides some insights into potential mechanisms of tissue-specific tumorigenesis.

Thursday, 21 November 2024, 17:00 Institute for Biological Physics, Zülpicher Str. 77a Seminar Room 0.02, Ground Floor Hosted by Michael Lässig