

Christian Frezza

CECAD, University of Cologne

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 mutations of core metabolic enzymes in the mitochondria, such as Fumarate Hydratase (FH), cause renal cancer strongly indicates that mitochondrial dysfunction can drive cancer. Today, I will provide an overview of our recent findings about the molecular mechanisms through 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, Zùlpicher Str. 77

Old Physics Building, Seminar Room Theory

Hosted by Michael Lässig