PRINCIPE FELIPE
CENTRO DE INVESTIGACION

Seminario CIPF

Mitochondria: More is different

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Abstract: Few years ago we discovered that RNA polymerase II elongation activity was dependent on cellular ATP content, and also that individual cells have a characteristic mitochondria content that varies across a clonal cell population. These two facts resulted in individual cells transcribing at different speeds as a function of mitochondria content.
Since then we have been studying the consequences of this variability. One consequence is that cells with different mitochondrial content accumulate transcripts at different speeds. Moreover, these transcripts are different. This has important functional implications. Alternative splicing is altered by mitochondria content as well as cell differentiation and response to apoptotic signals.

Fractional killing is the main cause of tumor resistance to chemotherapy. This phenomenon is observed even in genetically identical cancer cells in homogeneous microenvironments. To understand this variable resistance, we investigated the individual responses to TRAIL in a clonal population of HeLa cells using live-cell microscopy and computational modeling. We found that the cellular mitochondrial content determines the apoptotic fate and modulates the time to death, cells with higher mitochondria content are more prone to die. This circumstance is mediated by the mitochondrial control of apoptotic protein abundance, especially pro-apoptotic proteins Bax and Bid. Modeling the apoptotic network, we demonstrate that this correlation confers on mitochondria a powerful discriminatory capacity of apoptotic fate.

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