

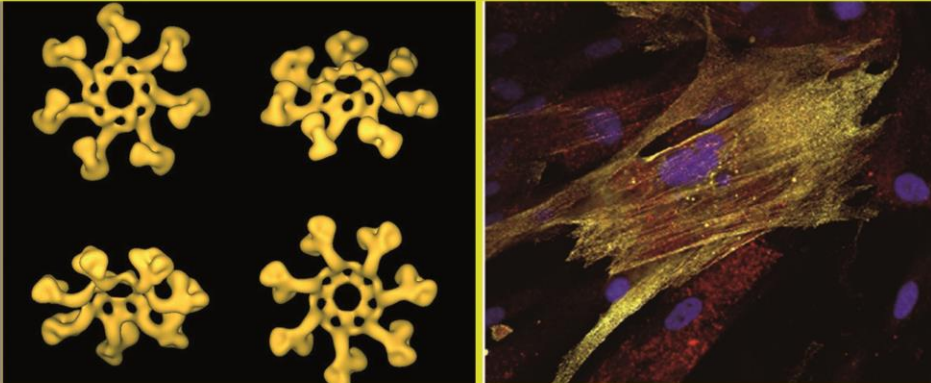


UNIVERSITÀ
DI TRENTO

Dipartimento di
Biologia Cellulare,
Computazionale e Integrata - CIBIO

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SEMINAR

JUNE, 1st

4.00 p.m. Room B109

Povo 2

via Sommarive, 9

Povo, Trento

How does the tumour suppressor p53 protect us from cancer?

The p53 gene, encoding the tumour suppressor p53 (also called TP53 or TRP53) is the most frequently mutated gene in human cancer (~50% of human cancers overall). P53 is a transcription factor that can directly activate ~500 target genes and indirectly many additional genes. P53 can activate diverse cellular responses, including cell cycle arrest/cell senescence, cell death by apoptosis, DNA damage repair, coordination of cellular metabolism and several others. Some of the direct p53 target genes encode regulators that play essential roles in the initiation of some of these cellular responses. For example, the cyclin dependent kinase inhibitor (CDKI) p21 is critical for p53 induced cell cycle arrest and cellular senescence, whereas Puma and Noxa are essential for p53 induced apoptotic cell death. Despite 40 years of research on p53, we still do not know which of the p53 activated cellular responses are critical for its ability to suppress tumour development and what factors determine whether after p53 activation a cell will survive and undergo cell cycle arrest or die by apoptosis. To address these questions we have generated an exciting panel of new gene-targeted mice, including GFP reporter mice for p53 induced cell cycle arrest/cell senescence, Tomato reporter mice for p53 induced apoptotic cell death and two complimentary strains of knock-in mice in which we can convert at will cells from wt p53 into mutant p53 and then back to either wt p53 (strain 1) or into a p53 deficient state (strain 2). Our initial analysis of these new strains of mice have already yielded new unexpected insights into the factors that determine cell outcome after p53 activation and on the mechanisms by which point mutations in p53 promote the development and sustained expansion of cancers.

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