ICGEB International Centre for Genetic Engineering and Biotechnology | Developing Knowledge

ICGEB International SEMINAR PROGRAMME 2017

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Senescent cells secrete a combination of factors collectively referred to as the senescence-associated secretory phenotype (SASP). Although the SASP comprises many pro-inflammatory cytokines, a plethora of other factors that also include proteases, chemokines and growth factors mediate its functions. The SASP reinforces senescence and activates an immune surveillance response but also display pro-tumorigenic properties and contribute to age-related pathologies. In order to find novel SASP regulators, we have conducted drug and siRNA screens. In the drug screen we uncovered the mTOR inhibitor rapamycin as a potent SASP suppressor. Here we report a mechanism by which mTOR controls the SASP by differentially regulating the translation of the MK2/MAPKAPK2 kinase through 4EBP1. In turn, MAPKAPK2 phosphorylates the RNA binding protein ZFP36L1 during senescence, inhibiting its ability to degrade SASP mRNAs. Consequently, mTOR inhibition or constitutive activation of ZFP36L1 impairs the non-cell-autonomous effects that senescent cells display during tumorigenesis. Altogether, our results place regulation of the SASP as a key mechanism by which mTOR could influence cancer, age-related diseases and immune responses.

"Linking senescence and inflammation: the senescence-associated secretory phenotype (SASP)"

Host: M. Giacca

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