分子細胞生物学セミナー

Gene expression in cellular senescence

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Cellular senescence is a stress responsive phenotype that accompanies stable cell cycle arrest. Despite their static appearance, senescent cells are metabolically active. It has been shown that multiple effector mechanisms are involved in this process, depending on the cell type and the particular stress. Such cellular stresses include constitutively active oncogenic stimuli, which paradoxically trigger pro-senescence effectors as a fail-safe mechanism. During oncogene-induced senescence (OIS) in human diploid fibroblasts (HDFs), the gene expression profile dramatically changes. We have previously identified a global and progressive chromatin structure alteration during senescence – senescence associated heterochromatic foci (SAHFs) – which have since been widely used as a marker of senescence. Our current model is that SAHF formation somehow contributes to the altered gene expression profile in senescent cells. More recently, we have also identified that autophagy, a cytoplasmic protein degradation program, is a new effector mechanism of senescence. Autophagy is highly activated during OIS, and facilitates the production of some secretory proteins essential for senescence establishment, at the post-transcription level. Here we propose that global protein turnover might be involved in the regulation of gene expression in parallel with the epigenetic regulation of transcription during senescence.

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