Synopsis of Original Research Paper

Regulation of replicative senescence by apollon

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Apollon is a large protein containing baculoviral-IAP-repeat and ubiquitin-conjugating enzyme domains at the amino- and carboxy termini, respectively. Apollon inhibits apoptosis by facilitating proteasomal degradation of caspase-9 and SMAC. Homozygous disruption of apollon gene in mice results in embryonic lethality, and the mutant embryos showed polymorphic phenotype including growth retardation and defects in vasculogenesis. Here we show that embryonic fibroblasts from apollon-deficient mice showed earlier replicative senescence and over-duplication of centrosome. The time required for completion of mitosis was extended in the apollon-deficient cells compared with wild-type cells. Thus, cell cycle regulation is affected in the apollon-deficient cells, which may be involved in the earlier replicative senescence.