

genotoxic stress, whereas overexpression of miR-34a mildly increases apoptosis. Hence, miR-34a is a direct proapoptotic transcriptional target of p53 that can mediate some of p53's biological effects. Perturbation of miR-34a expression, as occurs in some human cancers, may thus contribute to tumorigenesis by attenuating p53-dependent apoptosis.

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