Preventing cancer without killing cells

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Inducing senescence in aged cells may be sufficient to guard against spontaneous cancer development, according to a paper published online this week in EMBO reports. It was previously unknown whether cellular senescence or programmed cell death – apoptosis – was the more important safeguard mechanism for suppressing tumours arising from dysfunctional telomeres.

Aged cells have abnormal chromosomes with dysfunctional telomeres – shorter ends – that can promote tumorigenesis in the absence of the tumour suppressor p53, and may be related to the higher incidence of cancer in older individuals.

However, in the presence of p53, dysfunctional telomeres can induce a permanent arrest of cell growth, known as senescence. Sandy Chang and colleagues studied mutant mice with dysfunctional telomeres and copies of the p53 gene that cannot initiate p53-dependent apoptosis but can execute p53-mediated senescence.

The authors found that activating the senescence pathway was sufficient to suppress spontaneous tumorigenesis. Their findings suggest that, by halting cellular proliferation, p53-mediated senescence may act as an important tumour suppressor mechanism in aged cells.

Source: European Molecular Biology Organization