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ATM, telomeres and aging

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ATM, the product of the gene mutated in ataxia-telangiectasia (A-T), is involved in the cellular response to DNA damage and has been implicated in telomere maintenance. *Atm* mutant mice display modest aging and growth retardation defects compared to A-T patients. In an Advanced Online Publication in Nature Wong *et al.* describe studies in mice to assess the interaction between ATM and telomeres (*Nature*, 22 January 2003, DOI:10.1038/nature01385). They bred *Atm*-deficient mice with animals lacking the telomerase RNA component (*Terc*). Double knockout mice showed an increase in genomic instability and chromosomal fusions, and premature lethality. Cell proliferation defects were also observed in primary cells in culture and in several stem-cell populations *in vivo*. The interaction between telomeres and ATM, and the impaired stem-cell reserve, explains the multi-organ affects in A-T patients.

References

1. ATM: genome stability, neuronal development, and cancer cross paths.

2. *Nature*, [http://www.nature.com]