

Abstract

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Telomere dysfunction triggers developmentally regulated germ cell apoptosis.

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BACKGROUND: Telomere dysfunction results in fertility defects in a number of organisms.

Although data from fission yeast and *Caenorhabditis elegans* suggests that telomere dysfunction manifests itself primarily as defects in proper meiotic chromosome segregation, it is unclear how mammalian telomere dysfunction results in germ cell death.

OBJECTIVE AND METHODS: To investigate the specific effects of telomere dysfunction on mammalian germ cell development, we examined the meiotic progression and germ cell apoptosis in late generation telomerase null mice.

RESULTS: Our results indicate that chromosome asynapsis and missegregation are not the cause of infertility in mice with shortened telomeres. Rather, telomere dysfunction is recognized at the onset of meiosis, and cells with telomeric defects are removed from the germ cell precursor pool.

CONCLUSION: This germ cell telomere surveillance may be an important mechanism to protect against the transmission of dysfunctional telomeres and chromosomal abnormalities.

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