PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Ink and Arf

ArticleInfo		
ArticleID	:	4168
ArticleDOI	:	10.1186/gb-spotlight-20010807-02
ArticleCitationID	:	spotlight-20010807-02
ArticleSequenceNumber	:	239
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2001–08–07 OnlineDate : 2001–08–07
ArticleCopyright	:	BioMed Central Ltd2001
ArticleGrants	:	
ArticleContext	:	130592211

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When primary murine fibroblasts are placed in culture they exhibit replicative senescence, associated with the induction of cell-cycle inhibitors. The Ink4a-Arf locus encodes two proteins, p16Ink4a and p19Arf, which regulate the cell cycle by modulating the activities of pRb and p53, respectively. In the Early Edition of the Proceedings of the National Academy of Sciences, Randle *et al.* describe the role of p19Arf in preventing immortalization of bone-marrow-derived preB cells and macrophages (*Proc Natl Acad Sci USA*, 10.1073/pnas.171217498). PreB cells from *Arf*-null mice evaded the senescence observed in wild-type cells and continued to express high levels of p16Ink4a. But *Arf*-null macrophages lost p16Ink4a during the immortalization process, by methylation-suppression of the *Ink4a* promoter. Wild-type macrophages also lost p16Ink4a expression in established lines. The authors conclude that loss of the two *Ink4a-Arf*transcripts has different effects on senescence of preB cells or macrophages.

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