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Loss of p16Ink4a

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The CDKN2A (INK4a/ARF)locus encodes two distinct cell cycle inhibitors, p16Ink4a and p19ARF, both of which have been implicated in tumorigenesis. In the September 6 Nature, two independent groups report the generation of knockout mice specifically lacking p16Ink4a. Surprisingly, mouse embryonic fibroblasts (MEFs) lacking p16Ink4a were normal in terms of growth characteristics, senescence phenotypes and resistance to oncogenic Ras-induced transformation. These observations are in contrast to results obtained with p19ARF-null fibroblasts. But both groups provide evidence for a role in tumour suppression: Krimpenfort *et al.* report that *p16Ink4a* mutation results in a susceptibility to spontaneous and carcinogen-induced melanoma formation (*Nature* 2001, **413:**83-86). Sharpless *et al.* also found sarcomas, adenomas and lymphomas (*Nature* 2001, **413:**86-91). These results establish that the *CDKN2A* locus does indeed contain two distinct tumour suppressor genes.

References

1. The *INK4A/ARF* locus and its two gene products.

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

3. Tumor suppression at the mouse *INK4a* locus mediated by the alternative reading frame product p19ARF.