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Id1 is one of a family of helix-loop-helix (HLH) proteins that inhibits transcription by sequestering other HLH factors. It has been implicated in the regulation of cell growth and cellular aging. In the July 3 Proceedings of the National Academy of Sciences, Alani *et al.* provide genetic evidence supporting a role for Id1 in preventing cell aging (*Proc Natl Acad Sci USA* 2001, **98:**7812-7816). They found that fibroblasts from Id1-null mice display premature senescence with increased expression of the cell-cycle regulators p16/INK4a, cyclin D1 and cyclin E. They show that Id1 specifically inhibits transcription of the *p16/INK4a* promoter, but does not affect p19/Arf regulation (although p19 is transcribed from the same locus as p16). Two E-box motifs in the *p16/INK4a* promoter are essential for Id1 repression. Finally, they report increased expression of *p16/INK4a in vivo*, in the ventral telencephalon of *Id1*-null embryos. The authors speculate that Id1 repression may be responsible for deregulation of p16 expression in the early stages of tumorigenesis.

References

1. ID helix-loop-helix proteins in cell growth, differentiation and tumorigenesis

2. *Proceedings of the National Academy of Sciences* , [http://www.pnas.org]

3. High incidence of T-cell tumors in E2A-null mice and E2A/Id1 double-knockout mice.