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MYC requirement

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There is increasing evidence that sustained oncogene expression may be required to maintain the cancer state, implying that therapeutic strategies that use transient pharmacological inactivation may prove effective. In the July 5 Science, Jain *et al.* describe an elegant experimental model to explore the effect of brief oncogene inactivation (*Science* 2002, **297:**102-104). They studied a transgenic mouse strain in which the conditional expression of the MYConcogene can be regulated by tetracycline. Some of these mice develop osteogenic sarcomas. Inactivation of the *MYC* transgene caused regression and differentiation of the tumor cells. Upon reactivation of *MYC* expression the cells did not revert to neoplastic tumors but underwent apoptosis. The authors propose that brief inactivation of oncogene expression changes the epigenetic context, revoking the ability to maintain tumorigenesis. These are promising results for future cancer therapies directed against oncogenes, suggesting that it might be possible to mitigate toxicities without compromising efficacy.

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