This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
K-Ras-mutant, *cyclin dependent kinase inhibitor 2A* (*Cdkn2a*; *Ink4a*; *Arf*; *p16ink4a*)-deficient mouse cell model of sarcoma	Mouse muscle-associated cells engineered to express a mutant *K-Ras* and to be deficient in *p16ink4a* could aid the development of new therapies for sarcomas. In normal mice, three different subsets of mouse muscle-associated cells with the *K-Ras* and *p16ink4a* modifications caused sarcomas, whereas the unmodified cell subset did not. Microarray analyses of the mouse sarcomas and primary sarcomas from patients identified 144 genes that were upregulated in the tumors, including 28 genes previously linked to sarcomas. Future studies could include testing sarcoma therapies in the engineered cells or in mice with tumors induced by the cells.	Patent and licensing status unavailable	Hettmer, S. *et al. Proc. Natl. Acad. Sci. USA*; published online Nov. 30, 2011; doi:10.1073/pnas.1111733108 **Contact:** Amy J. Wagers, Harvard University, Cambridge, Mass. e-mail: amy.wagers@joslin.harvard.edu

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