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This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Mouse model of schizophrenia	Mice with a mutation that impairs phosphorylation of the NMDA receptor NR1 subtype (GRIN1; NR1) could help guide the development of schizophrenia therapies. In schizophrenics, the serine residue 897 of GRIN1 is less phosphorylated than that in nonschizophrenics. In mice, a knock-in mutation of *Grin1* that blocked phosphorylation at the equivalent residue generated mice with impairments in social and sensorimotor behaviors similar to what is seen in humans with schizophrenia. Next steps include additional characterization of the knock-in model.	Patent and licensing status undisclosed	Li, B. *et al. J. Neurosci.*; published online Sept. 23, 2009; doi:10.1523/JNEUROSCI.2109-09.2009 **Contact:** Effat S. Emamian, Advanced Technologies for Novel Therapeutics LLC, Newark, N.J. e-mail: emame@atnt-usa.com

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