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This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Drug platforms			
Engineering botulinum toxin to target non-neuronal cells to broaden therapeutic applications	*In vitro* studies suggest that modifying botulinum toxin at its catalytic site could result in compounds to treat non-neuronal secretory diseases. The botulinum toxin light chain cleaves neuronal synaptosomal-associated proteins such as SNAP25 but does not affect non-neuronal isoforms like SNAP23. *In vitro*, a toxin with a point mutation at the catalytic site cleaved SNAP23 as efficiently as SNAP25. In HeLa cells, the mutated toxin cleaved endogenous SNAP23 and inhibited tumor necrosis factor-α (TNF-α)-mediated secretion of mucin and IL-8, which are associated with some secretory diseases. Next steps include engineering the toxin to bind a cell-specific receptor to improve efficacy and specificity. At least seven companies have botulinum toxin–based therapeutics in development stages ranging from preclinical to marketed.	Patent application filed for the mutated botulinum; available for licensing	Shen, C. & Barbieri, J. *Proc. Natl. Acad. Sci. USA*; published online June 1, 2009; doi:10.1073/pnas.0903111106 **Contact:** Joseph Barbieri, Medical College of Wisconsin, Milwaukee, Wis. e-mail: jtb01@mcw.edu

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