In the news

BOTOXIC TRANSPORTATION

It is widely known that Botox exerts its wrinkle-decreasing effects through its toxicity for nerve cells: it was considered to be safe for clinical use because those effects were thought to be localized to the site of its injection. There is growing concern, however, that Botox — or botulinum neurotoxin A — might not be as static as was previously believed. A recent study published in *The Journal of Neuroscience* added a new dimension to those worries.

Botox blocks neurotransmitter release at synapses by cleaving SNAP25, a protein that is essential for synaptic vesicle fusion. The study's researchers identified cleaved SNAP25 in the rat facial nucleus following Botox injection into whisker-muscle tissue, and follow-up investigations indicated retrograde transport of the toxin through the nerves. According to one of the study's authors, Matteo Caleo of the Italian National Research Council, the discovery was surprising: "A significant portion of the toxin is active where it's not intended to be." (*ScienceNOW*, 2 April 2008.)

So how worried should we be? "The idea that there could be some transmission of this to the CNS needs to be followed up," says Matthew Avram of Massachusetts General Hospital. (*DailyMail*, 2 April 2008.) Christopher von Bartheld, of the University of Nevada, agrees because Botox is so potent: "...maybe even one toxin molecule per synapse can block neurotransmitter release at that synapse for many days." (*NewScientist.com*, 2 April 2008).

However, although the findings warrant further investigation, the news might not be all bad. Caleo thinks that, for certain patients undergoing Botox treatment for muscular disorders, migration of the toxin could even be beneficial: "Whether the distant actions result in 'negative' or 'positive' effects remains to be clarified." (*NewScientist.com*, 2 April 2008).

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