New study helps explain how botulism-causing toxin can enter circulation

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New research in the *Journal of Cell Biology* helps explain how the toxic protein responsible for botulism can enter circulation from the digestive system. The study appears online May 10.

Botulism, a rare but serious paralytic illness, is caused by botulinum neurotoxin (BoNT), an extremely toxic protein that is produced by the bacterium Clostridium botulinum. In food-borne botulism, the nontoxic components of BoNT—including hemagglutinin (HA)—protect the toxin from the low pH and enzymes encountered in the digestive tract. BoNT then passes through the intestinal epithelial barrier to enter circulation from the gut.

Although studies have examined how BoNT crosses the intestinal epithelial barrier, the mechanism by which it accomplishes this feat has remained a mystery. In this study, a team of Japanese researchers led by Yukako Fujinaga shows that HA plays a role. HA binds epithelial cadherin (E-cadherin), disrupting E-cadherin-mediated cell-to-cell adhesion and thereby disrupting the epithelial barrier.

Interestingly, the research demonstrates a species-specific interaction between HA and E-cadherin. Although HA binds human, bovine, and mouse E-cadherin, for instance, it does not bind rat or chicken.

More information: Sugawara, Y., et al. 2010. J. Cell Biol. doi:10.1083/jcb.200910119

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