Neurotoxins

Much of what is known about synaptic transmission has been obtained from chemicals that other animals produce. These chemicals, called **neurotoxins,** block synaptic transmission. For example, symbiotic bacteria in a poison gland of the puffer fish, *Spheroides*, produce tetrodotoxin. Tetrodotoxin blocks sodium channels and prevents an action potential. Humans who eat puffer fish without removing the tissues/organs containing the neurotoxin often die by paralysis. In Japan, where this fish is a delicacy, specially trained chefs remove the organs that contain most of the neurotoxin. In spite of this precaution, about 100 diners die annually from improperly prepared puffer fish.

The dinoflagellates responsible for "red tides" produce a similar neurotoxin, called saxitoxin. Saxitoxin inhibits sodium transport in nerve cells, thus killing the fishes that eat the dinoflagellates. If filter-feeding molluscs consume the dinoflagellates, and humans eat the "shellfish," the humans may suffer from paralytic "shellfish" poisoning (PSP).

Another type of poisoning in humans is called "ciguatera." It results from eating marine fishes (red bass, moray eels, mackerel) that have consumed a dinoflagellate that produces the neurotoxin ciguatoxin. Ciguatoxin also blocks sodium transport.

Batrachotoxin, from the poison-dart frog (*Phyllobates*) of South America, keeps sodium channels open, leading to hyperpolarization and nerve dysfunction. Neurotoxins in the venom of the North African scorpion (*Leiurus*) and from the sea anemone (*Anemonia*) slow the closing of the sodium channels at the end of an action potential.

All of these neurotoxins normally help defend their animal producers against predation. They are also useful tools for experimentation, however, and have helped neurophysiologists to better understand the normal functioning of neurons.