**NEUROBIOLOGY**

**Immune Molecules Prune Synapses In Developing Brain**

The complement cascade is part of the body’s innate immune defense: a protein work crew whose duties include tagging bacteria and other bad guys for elimination. A new study suggests that complement proteins may have a surprising yet analogous function in the developing brain, tagging unwanted synapses for removal. The work also hints that these proteins may promote synapse loss in early stages of neurodegenerative disease.

“It’s a pretty provocative finding,” says Greg Lemke, a neurobiologist at the Salk Institute for Biological Studies in San Diego, California. “This is part of a growing body of evidence that many molecules of the immune system have a second set of jobs in the brain,” says Lisa Boulanger, a neurobiologist at the University of California, San Diego.

**PHYSICS**

**Simple Scheme Stores Light by Converting It Into Vibration and Back**

A few years ago, physicists slowed light to a crawl and then stopped it entirely (Science, 26 January 2001, p. 566). To do that, they exploited strange quantum-mechanical interactions between light and atoms in a gas, converting a pulse of light into a subtle arrangement of spinning atoms. On page 1748, three physicists report a simpler way to hit the brakes: They convert light into an optical fiber. They fed a “data” pulse in one end and a short, intense “write” pulse in the other. When the two collided, the data pulse disappeared and was replaced by a vibration, which was fixed by the proper- sions between signals in optical networks. But that should make the effect more robust but rules out truly bizarre embellishments. For example, Hau and colleagues have encoded a light pulse in one cloud of atoms and revived it in another cloud by letting a few atoms drift between the two, as they reported 8 February in Nature. Such a feat would be impossible with the fiber technique.

The new study, which appears in the 14 December issue of Cell, began as an attempt to determine whether neural support cells called astrocytes have a role in refining synaptic connections between neurons during development, says senior author Ben Barres of Stanford University in Palo Alto, California. Postdoc Beth Stevens and colleagues used gene chips to look for changes in gene expression in neurons from the developing retinas of rats when the neurons were cultured with astrocytes.

To their surprise, astrocytes spurred the neurons to crank out a complement protein called C1q, which elsewhere in the body kicks off a cascade of chemical events that culminates in the destruction of an intruding cell. In experiments with mice, the researchers found that C1q concentrations in the retina and brain peaked a week or so after birth and dropped dramatically as mice matured. The peak coincided with the period when unwanted synapses are pruned. More intriguing, C1q seemed to concentrate...
at puny, immature-looking synapses in the developing nervous system.

When the researchers examined the brains of mice lacking a functional C1q gene, they found that development had gone awry in the lateral geniculate nucleus, a relay station in the brain that receives synaptic inputs directly from retinal neurons. In normal mice, geniculate neurons initially receive inputs from both eyes and then prune them so that they only receive input from one eye or the other. In the mutant mice, geniculate neurons maintained extraneous inputs from both eyes into adulthood.

That’s a striking finding, Boulanger says: “When you get rid of these proteins that we thought just functioned in the immune system, it disrupts a very specific event that we think is involved in making the precise, final connections in the developing visual system.” Many questions remain, however. Barres suspects that complement proteins mark unwanted synapses for removal by microglia, immune cells in the brain. More work is needed to demonstrate that, Boulanger says, and to figure out why only certain synapses are flagged for removal.

Finally, Barres and colleagues collaborated with Simon John’s group at the Jackson Laboratory in Bar Harbor, Maine, to investigate whether C1q might have a role in synapse loss in a mouse model of glaucoma.

Parasites From Fish Farms Driving Wild Salmon to Extinction

A new study suggests that fish farming could rapidly wipe out some populations of wild salmon in British Columbia. Although some researchers are calling for dramatic controls on the industry, others say the risk hasn’t been established firmly enough. At stake is the $450 million aquaculture business.

One of the top concerns about aquaculture is the spread of disease and parasites to wild species. On page 1772, the first population-level analysis suggests that sea lice from farmed salmon will cause several populations of one species of salmon in British Columbia to plummet by 99% within 8 years. “It’s a shocking number,” says salmon conservation expert John Reynolds of Simon Fraser University in Burnaby, Canada, who was not involved in the research. But environmental physiologist Scott McKinley of the University of British Columbia in Vancouver worries about rushing to judgment. “You cannot conclude anything from a correlation,” he says.

Sea lice are small crustaceans that latch onto salmon and other fish. They feed on tissue and create lesions that make it hard for fish to regulate their body fluids. The saltwater parasites naturally occur on adult salmon in the sea but not on juveniles, which hatch in fresh water and then swim to the sea. In 2001, however, researchers found significant numbers of sea lice on wild juveniles that had passed by fish farms in British Columbia. The situation was alarming because young pink salmon are more vulnerable to damage from lice than adult salmon are.

Graduate student Martin Krkošek of the University of Alberta, Edmonton, started studying the problem in 2003. In previous papers, he and colleagues calculated that juvenile pink salmon are 73 times more likely to be infected with sea lice after they passed by salmon farms than are fish that didn’t pass by and that lice can kill between 9% and 95% of juvenile pink salmon, depending on how many fish farms they must swim by. Some researchers are unconvinced, however, and point to other studies that suggest lower mortality from sea lice.

In the new work, Krkošek and colleagues investigated the extent to which sea lice are affecting pink salmon populations throughout the Broughton Archipelago near Vancouver Island. They analyzed 35 years of records from the Canadian fisheries agency on the number of salmon in seven rivers that flow into marine channels with fish farms. They also looked at 64 rivers from which migrating salmon do not pass by fish farms. Using a standard model, they calculated that pink salmon not exposed to fish farms showed the same range of population size for all 35 years, varying from year to year.

The pink salmon that swam past salmon farms showed the same pattern, until the lice infestations began in 2001. Then all seven populations shrank year after year. If these populations continue to decline at this rate, they will be 99% gone within four generations. “It’s very fast,” says Krkošek, who says immediate conservation steps are necessary. “We can’t sit around and do more research, because these fish will be gone.” Senior author Mark Lewis of the University of Alberta in Edmonton and another co-author were among 18 scientists who in September called for requiring salmon farms to be surrounded by barriers to prevent the spread of parasites or disease.

As with previous papers, the reaction to the new finding is polarized. McKinley and others say that there are too many unknowns to conclude that sea lice from farms harm wild salmon. Many factors influence their abundance, including fluctuations in ocean nutrients. But fisheries biologist Ray Hilborn of the University of Washington, Seattle, says it is too risky to farm fish in open pens near wild relatives: “The bigger concern is that [sea lice] are just one of many pathogens. There could be other things out there that we don’t know about.”