Biomedicine

Diabetes results from suicidal cells

Insulin-dependent diabetes mellitus, or type I diabetes, occurs when an autoimmune reaction leads to the death of insulin-secreting islet cells in the pancreas. But is that cell death a case of murder or suicide?

Diabetes researchers have long known that the disease appears after certain immune cells invade the pancreas. The mystery is whether the immune cells destroy the islet cells directly or induce apoptosis, a form of cellular suicide. The destruction of islet cells normally takes place over several months or years, so it is difficult to catch one in the act of dying, notes Jonathan D. Katz of Washington University School of Medicine in St. Louis.

Now, by breeding genetically engineered, diabetes-prone mice with mice lacking a normal immune system, Katz and his coworkers have created a mouse strain that develops diabetes quickly, compressing the period when the islet cells are dying into a few days. In the Jan. 7 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, the researchers report evidence—DNA fragments unique to cells undergoing apoptosis—that the islet cells of these mice commit suicide.

Researchers may one day be able to genetically engineer islet cells to resist apoptosis and transplant those islet cells into people with diabetes, says Katz. The first step toward that goal, he adds, is to determine how the immune cells induce the apoptosis. —*J.T.*

The latest salvo in the prion debate

"Researchers Rule Out Proteins As Cause of 'Mad Cow' Disease" blazed the headline in the Jan. 17 Washington Post. Don't believe everything you read, however. When it comes to mad cow disease, all results remain open to interpretation.

The Post story, and similar articles elsewhere, centered on an unexpected experimental result described in the Jan. 17 SCIENCE. A research group headed by Dominique Dormont of the Atomic Energy Commission in Fontenay-aux-Roses, France, ground up brains of cows with bovine spongiform encephalopathy (BSE), the fatal neurodegenerative disorder also known as mad cow disease. The scientists then injected samples of the bovine brains into the brains of 30 mice.

After periods ranging from 368 to 719 days, all of the mice began experiencing symptoms of a BSE-like neurological disorder. Yet when the scientists searched the brains of those mice for prions, they found none in 55 percent of them. That was surprising, since prions are now thought by many scientists to be the infectious agent for BSE. According to this hypothesis, prions are malformed versions of a cellular protein called PrP. Prions cause BSE by converting normal PrP proteins into their own, misshapen form.

Once ridiculed, the prion hypothesis has slowly gained a following among scientists (SN: 9/24/94, p. 202). Because they did not detect these abnormal proteins in the brains of all the diseased mice, however, Dormont and his group speculate that prions are not the agent for BSE. Still, the prions probably play a crucial role in how the disease develops, they note.

While the few researchers who argue that an undetected virus or bacterium causes BSE have taken heart from the results of the French group, other scientists are skeptical. "Our experience is quite different than the French group's," observes Moira E. Bruce of the Institute for Animal Health in Edinburgh, whose research group has also injected BSE brain tissue into mice. The most important distinction, Bruce notes, is that in every mouse tested so far, her group has found accumulations of prions.

Bruce argues that the work of Dormont's group does not shatter the prion hypothesis, but she also remains undecided as to whether prions or some infectious microbe causes BSE. Like many other scientists, "I'm still on the fence," she says. —*J.T.*

Earth Science

Is bigger better? The fossils speak up

All living organisms, from fungi to fig trees to flounders, tend to develop larger bodies over geologic time, according to a venerable theory of evolutionary biology. This rule, derived by Edward Drinker Cope in 1871, appears to make biological sense because beefy bodies can better defend themselves against predators and have an advantage in the struggle for food and territory. A new study of mollusk evolution, however, threatens to erase Cope's rule from the textbooks.

Paleontologists can point to many examples of animals that seem to follow the rule, but nobody had ever tried to assess the idea using statistically rigorous methods until David Jablonski of the University of Chicago took it upon himself to size up the issue, a task that took 10 years. As a test, he analyzed the fossils of clams and snails that lived along the Atlantic and Gulf Coasts of North America from 81 to 65 million years ago. Jablonski measured the sizes of the largest known fossil specimens belonging to 1,086 species. Then he grouped the data into 191 separate mollusk lineages and tracked how the sizes changed over time.

According to Cope's rule, both large and small species in each lineage should have gotten bigger with time. Jablonski found no such pattern in his data. While 27 to 30 percent of the mollusk lineages consistently developed bigger bodies, 26 to 27 percent evolved smaller body sizes. Another substantial portion of lineages, 25 to 28 percent, moved toward wider variation, with smaller species getting smaller and larger species getting larger. Jablonski reports his findings in the Jan. 16 NATURE. The moral, says Jablonski, is that "large size is not universally advantageous."

In the same issue, Stephen Jay Gould of Harvard University comments that Jablonski constructed "the most comprehensive set of data ever assembled to test Cope's rule—and the rule fails in this case." —*R.M.*

Gearing up for more hurricanes

A spate of hurricanes and tropical storms has hounded the Atlantic in the last 2 years, and 1997 will bring no relief, forecasts a team of hurricane researchers. William M. Gray of Colorado State University in Fort Collins and his colleagues projected last month that the Atlantic would spawn seven hurricanes, three of them major storms. This level of hurricane activity ranks slightly above average.

Gray's group bases its 1997 forecasts on a statistical assessment of recent weather patterns in the Atlantic Ocean, Africa, and the Pacific Ocean. Gray notes, for instance, that sea surface temperatures in the equatorial Pacific have ranged between cool and normal, a pattern that historically enhances hurricane formation in the Atlantic. The scientists also analyzed winds in the equatorial stratosphere; air temperature over Singapore; rainfall, temperature, and air pressure in West Africa; air pressure at sea level around the Azores; and Atlantic water temperatures and winds.

Hurricane activity in the Atlantic remained depressed from 1991 through 1994. With the surge in storms during the last 2 years, Gray suggests that the Atlantic may be shifting into a very active period for hurricanes, similar to the mid-1940s and the 1960s.

Robert W. Burpee, director of the National Hurricane Center in Miami, warns against drawing conclusions from a few years. "One has to be cautious in thinking that this represents a long-term trend," he says.

Burpee adds that the number of hurricanes in a given year has little bearing on how many will make landfall and how destructive they will be. For example, 1992 was a relatively quiet year that produced only four hurricanes—but one of them was Andrew, the costliest hurricane in U.S. history. —*R.M.*