provements over the control group in every test of motor coordination. James A. Joseph of HNRCA and his colleagues report their findings in the Sept. 15 JOURNAL OF NEUROSCIENCE.

After eating blueberry-laced chow for 2 months, 21-month-old animals outperformed unsupplemented, younger rats, Joseph says. "So, we got reversals in age-related declines." The blueberries that each animal downed were equivalent, when adjusted for body weight, to 1 cup daily in a person's diet, he notes.

The scientists measured a variety of chemical-signaling characteristics in each rat's striatum, a brain region pivotal to coordination. Each supplement showed a different benefit pattern, Joseph says, suggesting that blueberries' protectiveness may trace to more than oxidant quenching.

"A next important step in the research will be to see if the improvements are long lasting," says Molly Wagster of the National Institute on Aging in Bethesda, Md., which funded the study in part.

The differential benefits seen with the three diets reinforce what many other recent studies have suggested: "All antioxidants aren't alike," observes William A. Pryor of Louisiana State University in Baton Rouge. Some reach different places in the body; others do more than halt oxidation, he says.

It's therefore important, he argues, not to rely on supplements containing a single antioxidant, such as vitamin E. "You've still got to eat plenty of different fruits and vegetables," Pryor says. Since pigments can be very potent antioxidants, he prizes deeply colored foods—especially "anything blue." —*J. Raloff*

Insulin attracts immune wrath in diabetes

In juvenile diabetes, immune cells attack a person's own pancreas. They single out pancreatic cell clusters called the islets of Langerhans and destroy the tiny insulin factories within, called beta cells. How beta cells invite such immune damage is a long-running mystery.

Recently, several discoveries have shed light on the biological mechanism behind this carnage. A study in mice now points to a fragment of the insulin protein itself as the target that draws friendly fire from immune-system warriors called CD8 T cells, researchers report in the September NATURE MEDICINE.

In mice and people prone to juvenile diabetes, these cells become rogues, killing off beta cells as if they were invaders. In neither species do beta cells grow back once destroyed.

Microbes or compounds that initiate immune responses are called antigens. Because part of the insulin protein draws an assault to the body's own tissues, researchers consider it an autoantigen. The immune system programs T cells to destroy anything carrying a specific antigen or autoantigen. Once created, that's all CD8 T cells do.

The new study is the first to finger an autoantigen for CD8 T cells, says study coauthor Charles A. Janeway Jr., an immunobiologist at the Howard Hughes Medical Institute at Yale University. Earlier research had identified the same area of the insulin molecule as an autoantigen for CD4 T cells, which also play a role in the attack on beta cells.

"To find what these [T cells] are actually targeting is very important," says endocrinologist George S. Eisenbarth, director of the Barbara Davis Center for Childhood Diabetes in Denver. "It's coming out that insulin . . . might be a primary or dominant autoantigen" for juvenile, or type 1, diabetes, he says.

Insulin is necessary for the proper metabolism of carbohydrates. In less than 1 percent of the population, beta cells are absent or fail to make enough insulin, resulting in juvenile diabetes. Genetic flaws—many details of which remain hidden—predispose the T cells to incite an immune attack on beta cells, Janeway says.

Mice make a useful, though not perfect, model of human diabetes. The mice used in this experiment came from a strain that frequently gets diabetes after 12 weeks of age. Janeway's group modified CD8 T cells so that they would change color when they come into contact with their activating antigen. The researchers were then able to identify the antigen as a stretch of nine amino acids on the insulin molecule's B chain.

Janeway's team now is looking for means to stimulate immune responses that turn off the rogue T cells. Scientists have been able to flood the bodies of mice with insulin fragments, preventing diabetes, Eisenbarth says. They suspect that T cells lock onto the insulin fragments instead of attacking the pancreas.

Indeed, Eisenbarth and other researchers are giving insulin or its derivatives to children at genetic risk of type 1 diabetes in hopes of inoculating them against beta-cell destruction. —*N. Seppa*

DNA strands connect the quantum dots

Borrowing from biology, chemists have devised a new way to assemble semiconductor bits into potentially useful materials. The specks are quantum dots, sometimes thought of as artificial atoms.

DNA can connect tiny pieces of cadmium selenide into three-dimensional arrays, report Gregory P. Mitchell, Chad A. Mirkin, and Robert L. Letsinger of Northwestern University in Evanston, Ill. The resulting composite of inorganic and organic materials could have applications in biological sensing and electronics.

The researchers create the composite by attaching lengths of single-stranded DNA to cadmium selenide particles suspended in water. Then, they add to the solution double-length DNA sequences that complement the strands bound to the quantum dots. Two DNA strands attached to dots hook to a free strand like "chemical Velcro," bringing the particles together, says Mirkin.

The Northwestern researchers and a separate group at the University of California, Berkeley had connected gold beads in a similar fashion (SN: 8/17/96, p. 100). The current study, however, is the first to extend the technique to quantum dots, which have useful electronic and optical properties, says Mirkin.

Each cadmium selenide dot, 3.2 nanometers in diameter, bears 5 to 10 DNA strands, in contrast to the 220 strands on the 13-nanometer gold beads.

The biggest challenge in attaching the DNA strands to cadmium selenide was getting the semiconductor bits to dissolve in water, says Mirkin. The researchers synthesize the quantum dots in organic solvents, which makes them "very greasy particles," he explains. Mirkin's group bound acid molecules to their surface and then removed protons to give the dots an electric charge.

Mirkin and his colleagues describe their findings in the Sept. 8 JOURNAL OF THE AMERICAN CHEMICAL SOCIETY.

Because of its adaptability, DNA offers researchers more control over the architecture of a three-dimensional array, says A. Paul Alivisatos of Berkeley. "It would be more difficult to do that with some kind of non–information-bearing self-assembly. DNA contains more information to use to create spatial organization," he says. For example, Mirkin and his colleagues have also created a hybrid material consisting of both gold and semiconductor dots linked by DNA.

Quantum dots have sparked the interest of scientists because the particles fluoresce in a wide range of colors depending on their size. They glow much brighter and for a longer time than conventional organic dyes, making them especially good for marking cells to view with microscopy (SN: 10/24/98, p. 271).

If electrons can travel through the DNA strands (SN: 8/14/99, p. 104), the quantum-dot aggregates might have interesting electronic properties, Mirkin suggests. He and his group are now exploring ways to use DNA-linked quantum dots as photonic materials, catalysts, and biological sensors. —*C. Wu*