

Crashing debut of an exotic heavy particle?

A high-speed positron, the antimatter counterpart of an electron, smashes head-on into an onrushing proton. It penetrates the particle, striking one of the proton's constituent quarks; then, picking up an enormous amount of momentum from the struck quark, it rebounds nearly straight back.

Such spectacular events are rare among the thousands of positron-proton collisions monitored at the HERA particle accelerator in Hamburg, Germany. Most of the time, the particles either miss or merely glance off each other.

Nonetheless, collisions in which positrons are sharply deflected seem to occur more frequently than expected, based on the standard model of particle physics. The standard model represents the current theoretical understanding of the fundamental particles and the forces of nature (SN: 7/1/95, p. 10).

Members of the large international team operating the H1 detector at HERA observed 12 high-momentum events in 3 years' worth of data, whereas theory would predict only 4.7 such events. Independently, the physicists operating the ZEUS detector at HERA obtained comparable results. Each group describes its findings in separate papers submitted for publication to *ZEITSCHRIFT FÜR PHYSIK*.

"It might be that what we are seeing is some statistical fluctuation," says ZEUS team member Malcolm Derrick of Argonne (Ill.) National Laboratory. "On the other hand, both experiments independently see an excess, so we have some confidence that maybe it's a real

effect."

The findings, if confirmed, suggest the existence of new particles or forces that are not part of the standard model. One possibility is that a positron may merge briefly with a quark, creating a particle that theorists call a leptoquark. This particle would then decay and spit out a positron.

Alternatively, "the underlying effect might not be the formation of a new particle but the action of a new force that causes a stronger interaction between the positron and the quark than anything in the standard model," says physicist David J. Miller of University College London.

A third possibility is that quarks are themselves made up of still smaller building blocks. Last year, physicists at the Fermi National Accelerator Laboratory in Batavia, Ill., detected a subtle effect hinting at such a quark substructure in debris scattered from high-energy collisions between protons and antiprotons (SN: 2/17/96, p. 102). However, subsequent analysis showed it was possible to explain the results in terms of conventional theory.

Which interpretation, if any, proves correct depends on future research. "As we collect more data, we'll find out whether our initial results hold up," Derrick says. The researchers expect to double the number of observations during 1997.

"The [high-momentum] events are so spectacular that we can see them immediately when they occur," Derrick adds. "We don't have to collect all the data before analyzing them." Stay tuned! —*J. Peterson*

Two monkeys 'cloned' from embryo cells

The firestorm of discourse over cloning, ignited by the recent news that Scottish researchers had created a lamb from the DNA of an adult sheep, has been fanned by scientists who announced this week that they have created rhesus monkeys with DNA from the cells of developing monkey embryos.

While this feat does not meet the classic definition of cloning—using a cell from an adult animal to create a genetic copy—the researchers say it is the first time live primates have been generated by this method.

The purpose of the work is not to clone adult monkeys but to develop a way of producing genetically indistinguishable primates, according to a statement released by the scientists at the Oregon Regional Primate Research Center in Beaverton who conducted the as-yet-unpublished research.

Such animals would be useful in drug studies and other research projects because they would enable investiga-

tors to dismiss genetic variability as a confounding factor in interpreting their experiments. Researchers have long experimented on mice made genetically similar by generations of inbreeding.

Like the sheep cloners, the Oregon scientists created their monkeys with a technique called nuclear transfer. The researchers stripped the genes from unfertilized monkey eggs and then added new genetic material by fusing each egg to a cell taken from an eight-cell monkey embryo.

This fusion sometimes tricks the egg into developing as if it had been fertilized by a sperm cell. When this occurs, researchers implant the resulting embryo into a surrogate mother monkey.

The Oregon group produced two monkeys by this method. The animals are not genetically identical to each other because two different embryos were used as sources of genetic material. —*J. Travis*

Gene heats up obesity research

Researchers have identified a gene apparently used by cells to convert excess calories to heat. The discovery may offer insight into why some people stay trim on the same diet that induces weight gain in others.

The gene, described in the *MARCH NATURE GENETICS*, encodes a molecule that researchers have dubbed uncoupling protein 2, or UCP2.

"UCP2 may be a major player in the body's energy metabolism," says obesity researcher Craig H. Warden of the University of California, Davis.

The protein, explains Warden, directs a cellular biochemical reaction that converts calories to simple heat rather than to ATP, the prime energy-storage molecule that powers cells' activities. As a result, cells with a more active UCP2 gene may have to burn additional calories to meet their energy needs, which would leave fewer excess calories to be stored as fat.

The tantalizing hypothesis raised by the discovery of this gene is that individual differences in UCP2 and its regulation may determine a person's overall metabolic rate and consequent propensity toward obesity.

The UCP2 gene was discovered independently by Warden's research group and a team led by Daniel Ricquier of the National Center for Scientific Research in Meudon, France. The groups joined a third team, led by Richard S. Surwit of Duke University Medical Center in Durham, N.C., to investigate the new gene further.

The scientists found that the gene is active in many types of cells, including fat and immune cells. Surwit speculates that infections trigger increased UCP2 activity in immune cells and that the heat generated produces fevers.

The researchers also discovered that dietary fat regulates the activity of the UCP2 gene. When they fed mice a high-fat diet, the gene's activity increased.

Moreover, the investigators found that the diet-induced UCP2 activity varies among strains of mice with different propensities toward obesity. Mice that become obese when placed on a high-fat diet seem to make less UCP2 than do the strains that resist obesity, the researchers report.

While there is so far no direct evidence linking the UCP2 gene to human obesity, researchers have started to contemplate obesity therapies involving the gene. Surwit contends that most human obesity is the result of a low metabolic rate, and he suggests that this defect might one day be corrected by administration of a drug that increases the activity of the UCP2 gene. —*J. Travis*