the proton, causing the proton to fall apart. By recording the outcomes of thousands of collisions, HERA researchers can piece together a picture of a whole "sea" of virtual quarks and gluons and calculate its density. And in this picture, says Caldwell, "the proton's a very active beast."

So far, says DESY physicist Joel Feltesse, who leads the group running H1, the detectors see evidence for about 30 gluons and three or four virtual quarks at any given time. Because these numbers couldn't have been predicted from theory, says Columbia's Sciulli, they "will have a profound effect in the long run on our understanding of protons and neutrons." And Caldwell notes that HERA may also answer another open question: whether exotic quarks can make a fleeting appearance in everyday protons and neutrons. Only two of the six quark types, "up" and "down," ever show up as valence quarks, but QCD leaves open the possibility that others—"charm" and "strange" quarks, for instance—can pop up in the virtual sea for long enough to get knocked out by an electron. Because these quarks are more massive than up and down quarks, they would leave different signatures in HERA's detectors.

The unexpectedly dense virtual sea is just one of the surprises emerging at HERA. There's also an unidentified object whose existence is inferred from a strange set of collision tracks. Generally, collisions transform the proton into spectacular showers of other particles—a display predicted by QCD, which holds that the proton's three valence quarks have different "colors" (arbitrarily called red, green, and blue), analogous to the positive and negative charges of electromagnetism. The proton's mix of colors renders it neutral and therefore stable. When an electron ejects a quark from a proton, both the quark and the proton lose their color neutrality. And objects with net color, such as naked quarks, immediately "clothe" themselves by pulling other quarks from the vacuum. That conjuring process stirs up additional particles in a kind of chain reaction. As a result, electron-proton collisions usually result in two particle jets, one generated by the ejected quark, the other—aimed in the proton's direction of travel—by the proton fragment.

Mystery particle. In the collisions that are raising eyebrows, however, the electron seems to ricochet off something within the proton. Meanwhile a jet—sparser than the ones generated by the ejected quarks—suggests that something has been knocked out of the proton. Yet the proton seems to continue unscathed, without dissolving into a jet itself. Apparently the loss of the particle, whatever it is, doesn't upset the proton's color neutrality. "This was totally unexpected," says Caldwell.

Theorists' best guess so far about what the electron is hitting is a theoretical throwback called a pomeron. Invented in the early 1960s by the Russian theorist Isaac Pomeranchuk to explain early indications that the proton has internal anatomy, the pomeron was set aside later when quarks were identified as the proton's internal components. Says Argonne National Laboratory physicist Malcolm Derrick: "The nature of the pomeron has been a mystery for 30 years." Perhaps this new result will help unlock the mystery, he says.

Derrick and his colleagues think that, unlike the top quark, which recently catapulted Illinois' Fermilab into the headlines, the pomeron is not a new particle. It may be something more like a temporary lump in the soup—perhaps a temporary clump of gluons. But even a cluster of known particles would be a surprise, because nothing in established theory predicts such behavior.

And that's the excitement of HERA's exploration of inner space, say physicists; no one knows what else they might find in coming years, when they plan to amass 100 times more data than they have collected so far. For a field in which many recent experiments have simply explored territory that theorists have mapped, having experiment take the lead into the inner-space frontier is a welcome change. Says Derrick, "We're in uncharted territory."

–Faye Flam

GENETICS

Lucky Break for Kidney Disease Gene

By spotting a clue in the chromosomes of a Portuguese family, an international team of researchers has homed in on the cause of the most common inherited kidney disease. The researchers, led by geneticist Peter Harris of the Medical Research Council's Molecular Haematology Unit in Oxford, England, announce in the 17 June issue of *Cell* that they've identified the gene at fault in most cases of autosomal dominant polycystic kidney disease (ADPKD), in which fluid-filled cysts form in the kidneys, damaging or destroying them.

So far, the group knows part of the gene's DNA sequence but not how it works. As a result, they're a long way from being able to prevent or treat the disease, which afflicts about 1 person in every 1000 and, in half of the cases, ultimately requires dialysis or a kidney transplant. But other workers in the field are hailing the Oxford group's achievement, which entailed singling out the errant gene from a region on chromosome 16 containing over 20 suspect sequences. Says geneticist Steve Reeders of Harvard Medical School, "This is very exciting—we've been on [the trail of] this gene for 3 years but found it very difficult to find any mutations." Reeders adds, "All the data together make me convinced [this is the gene responsible]."

Reeders and his group had identified the approximate location of the ADPKD gene in 1985 by tracing the inheritance of genetic markers in afflicted families. But when various laboratories analyzed parts of the suspect chromosome region, they found a number of unknown genes, any one of which might have been at fault. In hopes of pinpointing the culprit, Harris's Oxford team, collaborating with scientists in Wales, Portugal, and the Netherlands, went hunting for visible abnormalities on chromosome 16 in ADPKD sufferers. A Portuguese family supplied the lucky break: In afflicted members of the family, part of chromosome 16 was swapped with a piece of chromosome 22.

By examining the DNA of ADPKD sufferers, the team was able to identify a large gene that was disrupted at the breakpoint on chromosome 16, making it the obvious candidate for being the gene at fault. With the gene sequence in hand, Harris's group examined the corresponding DNA in 300 more ADPKD families and identified three other mutations that can impair the gene, confirming its role in the disease.

The Harris group calls their prize *PKD1* (the 1 is an acknowledgement that another, as-yet-unidentified gene on chromosome 4 is responsible for perhaps 15% of ADPKD cases). Screening for *PKD1* mutations should ultimately make it possible to identify most people who will develop polycystic kidney disease, which usually becomes apparent in middle age. Doctors could then be vigilant for damaging symptoms, such as hypertension, that can be treated.

To understand the disease, however, the researchers will now have to learn what kind of protein *PKD1* encodes and what function it plays in the body, as well as how the mutations disrupt it. "We're not sure whether the mutation results in the absence of the protein or a defective product," says Harris, who adds that answering these questions is his next goal.

Reeders cautions that determining how *PKD1* works is likely to be "tough sledding," because the gene's unusual length will make it difficult to characterize. As other successful gene hunts have shown, finding a disease gene is only the first step toward a cure.

–Claire O'Brien

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