Biology

Brazilian cave yields skeletons

Some 10,000 years ago, waters from the rain forest flowed through Toca da Boa Vista Cave, not far from the Bahia coast in Brazil. The stream swept dead Pleistocene mammals into the soggy hole, where they remained undisturbed until the arrival of a Brazilian caving club in 1992. The group's prize find: two nearly complete skeletons from the primate family *Atelinae*, cousins of today's spider monkeys.

One specimen, *Protopithecus brasilienus*, is now considered the largest nonhuman primate ever to call the Americas home, report Walter C. Hartwig of the University of California, Berkeley's anthropology department and Castor Cartelle of Brazil's Instituto de Geociencias in the May 23 NATURE. At about 25 kilograms, the monkey weighed roughly the same as a large dog—and more than twice as much as the largest South American primate today.

P. brasilienus had a stocky build and a set of menacing canine teeth, which it bared as it fought for food and females, says Hartwig. The rest of the primate's teeth were adapted to a vegetarian diet. Its skull shows that the big primate had an enlarged vocal sac, which it used to communicate over long distances, as howler monkeys do today.

Although *P. brasilienus* was identified and named in 1838 by a Danish naturalist who found leg bones in another Brazilian cave, Hartwig says the new skeleton gives scientists their first detailed look at the creature.

The second skeleton from Toca da Boa Vista represents an extinct member of *Atelinae* that Hartwig says is wholly new to scientists. The discoverers named the monkey *Caipora bambuiorum* for a mythical forest creature of the local Indians and the caving club, respectively, report Hartwig and Cartelle in the June 25th PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Weighing in at about 20 kg, *C. bambuiorum* appears to have been slightly smaller than its cousin. Although *C. bambuiorum* had canine teeth for display purposes, this primate, like its kin, spent most of its life in the upper branches of trees, seeking fruit.

There are two competing explanations for why New World primates had reached large sizes by the late Pleistocene, Hartwig notes. First, the forest grew over a wider area during that era than it does today, supplying ample food for big animals. Alternatively, the retreat of the forest by the late Pleistocene could have created a fiercely competitive climate in which bigger animals bullied their way to the dinner table. In either case, dwindling resources eventually pulled the rug out from under the large species.

New Brazilian monkey takes to trees

Since 1990, when researchers discovered the black-faced lion tamarin (SN: 6/30/90, p. 406), a surprising six additional living primates have come to light in the rain forests of Amazonia. Brazilian researchers have named the latest addition to the primate order *Callithrix saterei*, after the Satere Indians of the region, according to a report in the June GOELDIANA.

Commonly called the Satere marmoset, the squirrel-sized creature sports an elegant coat of reddish fur. The lively little animal spends its days scampering over tree bark, which it assaults with its razor-sharp teeth. The marmoset then laps up sweet, energy-rich sap as it oozes out.

Almeida Noronha/Amazon For. Found.

Callithrix saterei, *the Satere marmoset, clings to the trees on which it feeds.* Callithrix *means "beautiful hair" in Greek.*

Biomedicine

Cancer gene scores a mouse knockout

Small, infertile, beset with neurological problems, and prone to deadly cancer within their first few months of life, the mice in Anthony Wynshaw-Boris' laboratory at the National Center for Human Genome Research in Bethesda, Md., are an unenviable lot. When subjected to ionizing radiation, the mice die from cancer much more readily than normal mice. The sad state of these rodents stems from their inability to synthesize the protein encoded by a gene called *ATM*.

Children who have mutations in both their copies of the human version of this gene suffer from a fatal condition called ataxia-telangiectasia, marked in its early stages by slurred speech and involuntary movements. Those afflicted also run a higher than normal risk of cancer.

The knockout mice, so called because their *ATM* genes have been knocked out by scientists, suffer problems remarkably similar to those of humans with ataxia-telangiectasia, note Wynshaw-Boris and his colleagues in the July 12 CELL. As a result, the researchers expect that studying the mutant mice will help them understand the function of the protein encoded by the *ATM* gene. Previous studies suggest it plays a role in repairing damaged DNA.

"The fact that we have a good model will also allow us to test a lot of things that might [alleviate] ataxia-telangiectasia. We can try more speculative therapies on a mouse than we would dare try on humans," says Wynshaw-Boris.

Some research has suggested that carriers of ataxia-telangiectasia, who have one mutant copy and one normal copy of the *ATM* gene, may be more vulnerable to cancer, particularly breast cancer. The investigators are therefore looking also at mice with only one mutant copy of the *ATM* gene. "If there's an increased risk of cancer, we would hope to see it in our mice," says Wynshaw-Boris. Though none of these mice have developed tumors so far, investigators caution that they have followed the animals for only a few months.

Mutant gene is not always a killer

The gene behind the neurological disorder Huntington's disease continues to surprise investigators. After finding the gene in 1993, researchers discovered that the deadly disease results only when the gene contains an abnormally large number of occurrences of a small sequence of DNA. If a person has too many of these so-called CAG repeats, Huntington's usually strikes. The more repeats the person's gene has, the earlier the disease tends to start. While Huntington's usually strikes adults, the disease has shown up in teenagers.

Recently, investigators have tried to pin down exactly how many CAG repeats it takes to turn the Huntington gene into a killer. Though most patients have 40 to 90 repeats, physicians have identified Huntington's patients with as few as 36.

Yet a set of 36 repeats, even 37, 38, or 39, in the Huntington gene is not always a death sentence. Among 178 people with 30 to 40 CAG repeats in the gene, 10 individuals having 36 to 39 repeats had no signs of the disease, even though they were all more than 67 years old, report David C. Rubinsztein of the East Anglian Medical Genetics Service in Cambridge, England, and his coworkers in the July AMERICAN JOURNAL OF HUMAN GENETICS.

One man with 39 CAG repeats lived until the age of 95. Two physical exams during his latter years revealed no symptoms of Huntington's, and an autopsy did not find the brain cell loss typical of the disease. Such cases suggest that factors besides the number of CAG repeats can, in rare instances, determine whether a person will suffer from Huntington's, says Christopher A. Ross of Johns Hopkins University in Baltimore, a survey coauthor. Unearthing those cofactors, suggests Ross, may help explain how CAG repeats produce disease and point the way to treatments for Huntington's.