Tooth analysis may decipher prehistoric diets

Scientists say they have identified the microscopic "fingerprints" of plant remains on the fossil teeth of *Gigantopithecus,* a huge Asian ape that lived from 6 million to 300,000 years ago. The evidence suggests that the extinct ape — which stood an estimated 10 feet tall and weighed more than 1,000 pounds — ate a varied diet, including both tropical fruits and fibrous grasses. If the dental decoding technique proves accurate, it may one day illuminate the feeding habits of other extinct animals, including human ancestors.

For now, analyses of plant residues on fossil teeth remain preliminary, says paleoanthropologist Russell Ciochon of the University of Iowa in Iowa City. Indeed, several paleontologists argue that no solid evidence links the tiny particles studied by Ciochon's group to the diet of living or extinct animals.

A scanning electron microscope revealed 30 floral fingerprints, known as phytoliths, on two out of four *Gigantopithecus* teeth under study, Ciochon and his co-workers report in the October PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (Vol. 87, No. 20). Monosilicic acid travels throughout a plant's vascular system and hardens inside and between the cells to create the phytoliths — remarkably durable silica impressions of the plant cells. Many plants and all grasses absorb the monosilicic acid as their roots take up groundwater.

"Phytoliths are widespread throughout the plant kingdom," says archaeologist and study participant Dolores R. Piperno of the Smithsonian Tropical Research Institute in Balboa, Panama.

Scientists first identified phytoliths nearly 150 years ago in Germany, and studies of phytoliths found in soil began in the 1950s. Since then, the hardy silica bits have turned up on stone tools unearthed at several archaeological sites. Piperno has identified phytoliths from more than 1,300 species of tropical plants, mainly in South America, as well as 19 plant species from three families native to a region of China where *Gigantopithecus* teeth and jaws have been found.

With Piperno's data in hand, the researchers identified silica bits on the *Gigantopithecus* teeth as belonging to a family of fibrous grasses that includes bamboo. Though *Gigantopithecus* may have eaten large amounts of bamboo, the phytolith evidence for bamboo consumption remains indirect, Ciochon says.

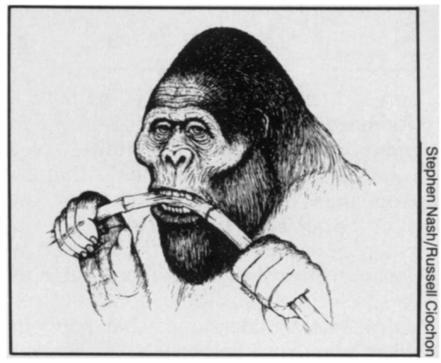
The researchers say other phytoliths found on these teeth belong to a family of tropical fruits common throughout Southeast Asia, suggesting that *Gigantopithecus* fed in forested areas. Regular consumption of the sugary fruits probably initiated the cavities observed in other *Gigantopithecus* teeth, they add.

Phytoliths apparently dig into tooth enamel during chewing and create surface scratches, possibly bonding to the tooth through the combined action of friction and moisture, Ciochon says.

But if friction welds phytoliths onto tooth enamel, the resulting heat may also deform their shape, argues Lawrence Martin of the State University of New York at Stony Brook. "Phytolith research is potentially a major advance in dietary analysis," he says, "but there are no studies of how phytoliths bond to the tooth surface."

Silica bits in the soil more likely attached to the *Gigantopithecus* teeth after death, adds Frederick E. Grine of Stony Brook, who has studied microscopic wear on the extinct ape's teeth.

No one knows whether phytoliths routinely attach to the teeth of living animals, says Mark Teaford of Johns Hopkins University in Baltimore. Ciochon says he plans to look for phytoliths on the teeth of lemurs living on Madagascar, and hopes to obtain teeth of human ancestors for phytolith analysis. *— B. Bower*



Stephen Nash/Russell Ciochon

Artist's rendition of Gigantopithecus *munching on bamboo.*

CF: Maxi-mutations make mini-diseases

Since last year's discovery of the gene causing cystic fibrosis (CF), researchers have learned that several different mutations in this "CFTR" gene can cause the inherited respiratory disease. Now, to their surprise, they find that some of the most severe mutations cause the mildest forms of the illness.

The odd revelation, which suggests that some patients with minor CFTR mutations might be better off with a completely botched gene, provides fresh clues about CFTR's normal function. It also hints at the possibility of treating some cystic fibrosis cases by knocking out the faulty gene's activity rather than by correcting it through gene therapy.

Normal CFTR genes direct the production of a protein that helps ions cross cell membranes. Most CF patients lack three DNA subunits in the gene and make a slightly gimpy CFTR protein lacking only one amino acid. The ensuing ion-transport deficiency leads to an accumulation of mucus in the respiratory tract and repeated lung infections that leave few CF patients surviving their 30s.

In the latest work, presented this week at an American Society of Human Genetics meeting in Cincinnati, several research teams catalogued some of the less common CFTR mutations and correlated them with clinical symptoms.

Garry R. Cutting of the Johns Hopkins School of Medicine in Baltimore and his colleagues identified two CF patients with "nonsense mutations," which appear to block the synthesis of the CFTR protein. Nonsense mutations in other genes often cause severe illnesses, but the two CF patients (aged 11 and 22) remain remarkably healthy with only very mild pulmonary disease.

In a separate study, Edward Highsmith Jr. and his co-workers at the University of North Carolina School of Medicine in Chapel Hill looked at three other CF patients with similarly severe mutations. These three cases "are extremely mild," says Highsmith, who notes that two of the patients weren't even diagnosed with CF until they were in their 50s and 60s. He adds that Belgian researchers recently reported comparable findings in patients with radical CFTR mutations.

The unexpected correlations may indicate that the CFTR protein normally functions by intertwining with a group of other proteins, say Cutting, Highsmith and others. They suggest that the complex may work reasonably well when the CFTR protein is either missing or so deformed that it fails to interact with the other proteins, whereas a mild mutation that only slightly distorts the protein's shape may completely disrupt the ensemble's activity.

If this theory holds true, says Cutting, CF symptoms might best be reduced by simply "knocking out" the faulty gene or its protein product — at least until gene therapy becomes practical. Highsmith adds that "it's easier to design a drug that inhibits a protein than it is to design a drug that tweaks a faulty protein back to normal." Both researchers say the novel approach of "finishing off" an already hobbled gene must await experimental proof of its value.

Michael Dean, a molecular biologist at the National Cancer Institute's research and development center in Frederick, Md., cautions that other genetic factors, as well as environmental variables, probably influence CF severity. At the Cincinnati meeting, he reported evidence that some severe CFTR mutations do cause severe disease. *— R. Weiss*