Maya beginnings extend back at Belize site

Evidence of the earliest known Maya, who cleared and farmed land bordering swamps as early as 4,500 years ago, has emerged from a site in northern Belize, researchers reported last week at the annual meeting of the Society for American Archaeology in Anaheim, Calif.

Until now, the oldest Maya settlements dated to about 3,000 years ago. These sites have yielded extensive pottery remains and led many investigators to assume that any prior farmers of the Yucatán Peninsula also fashioned ceramic vessels.

"Our evidence suggests that the first agriculturists in this region did not use pottery," asserts excavation director Thomas R. Hester of the University of Texas at Austin. "Beginning around 2500 B.C., they introduced crops from Mexico or perhaps beyond and left behind distinctive stone tools."

Later Maya occupations of the same site, called Colha, have undergone excavation since 1979. But in 1993, Hester's team made the first systematic effort to document a preceramic presence at the tropical, forested location.

Early Colha farmers inhabited the area in two phases, Hester notes. Stone tools in deeper soil layers date from 2500 B.C. to 1700 B.C., based on radiocarbon age estimates of accompanying charcoal bits. Comparable dates come from an adjacent swamp, where pollen analysis documents forest clearance by 2500 B.C., asserts John G. Jones of the Smithsonian Tropical Research Institute in Panama.

Jones finds pollen evidence for the appearance of several cultivated crops soon thereafter, mainly corn and manioc, a starchy plant.

From about 1400 B.C. to 1000 B.C., Colha residents made foot-shaped stone tools that were chipped and sharpened on one side. Preliminary scanning electron microscope analysis of polish on these tools, directed by Dale Hudler of the University of Texas, suggests that inhabitants used them to cut away vegetation after controlled burning of trees and perhaps also to dig.

An example of the same tool, known as a constricted uniface, also emerged last year at Pulltrouser Swamp, a Maya site 20 miles northwest of Colha. Mary Pohl of Florida State University in Tallahassee, who reported on the find at the Anaheim meeting, cites a preliminary radiocarbon date of 1300 B.C. to 1000 B.C. for the artifact. A sharpened stone point at Pulltrouser Swamp dates to between 2500 B.C. and 2000 B.C., she adds.

A graduate student chanced upon the first constricted uniface at Colha in 1987. Its unusual design led Hester's team to suspect that Colha might have harbored an extremely early Maya population.

Several other sites in Belize have yielded constricted unifaces, but archaeologists have been unsure of their ages and origins.

Techniques used to manufacture constricted unifaces show gradual refinement and modification in stone tools of Colha residents living after 1000 B.C., holds Harry J. Shafer of Texas A&M University in College Station, a member of Hester's scientific team.

Continuity in stone tool design and manufacture suggests that preceramic Maya inhabited Colha, according to Shafer, rather than non-Maya folks who migrated to the area and later left or were incorporated into Maya villages.

"None of us had any reason to suppose that Colha would produce a preceramic Maya occupation," remarks Norman Hammond of Boston University, who directs excavations at Cuello, a Maya site that dates to about 1000 B.C. (SN: 10/2/93, p.212). "This is a bit of archaeological serendipity."

The earliest Central American farmers probably settled at the edges of swampland that they cleared and cultivated, Hammond says.

Excavations of preceramic Colha so far have focused on quarry and field areas, Shafer notes. Some pottery may show up in early residential structures, he remarks. *— B. Bower*

Blocking multiple sclerosis in a mouse model

"When two proteins interact, it's kind of like making love — there's movement, and a signal is sent," says molecular pharmacologist Bradford A. Jameson, who is trying to figure out how and where to interrupt the complex cascade of signals that causes multiple sclerosis (MS).

A computer-designed peptide (a sequence of amino acids too small to be a protein) blocks the development of an MS-like disease in mice, Jameson and his coworkers at the Jefferson Cancer Institute in Philadelphia report in the April 21 NATURE.

"We think we can intervene [in mice] ... at almost any stage," says coauthor Robert Korngold, "and prevent the progression of the disease."

Between 250,000 and 350,000 people in the United States suffer from MS. Their immune systems turn traitor, attacking the essential protein coverings that insulate nerves in the brain and spinal cord. The destruction of these myelin sheaths short-circuits the electrical signals that flow through the nerves, resulting in weakness, visual impairment, and loss of muscle control. In the most severe cases, MS can lead to paralysis. The disease is episodic — it attacks in bursts — and often worsens progressively.

Mice with experimental allergic encephalomyelitis (EAE) show many of the same symptoms as humans with MS, and the mouse disease progresses similarly. But researchers understand what triggers EAE while remaining in the dark about the cause of MS. "The problem is," says neurologist Moses Rodriguez of the Mayo Clinic in Rochester, Minn., "most remedies that have been successful in EAE have not worked in MS."

In the normal course of any disease, white blood cells called T cells replicate when presented with foreign substances and enlist the help of other cells in the immune system to attack the invader. In MS, a T cell gets a message that the myelin sheath is foreign.

Jameson wanted to prevent this message from getting through. Other researchers have implicated T cells carrying CD4 proteins in myelin sheath inflammation. Jameson theorized that if he designed a peptide to mimic the shape of a piece of the CD4 protein, that inactive peptide could replace the protein piece and no signal would connect. A simple copy of the peptide worked in test tubes but was quickly broken down in the mice.

So Jameson synthesized a reverse mirror image of the CD4 segment. This tiny peptide resists being broken down by the body yet still mimics the shape — and thus can block the function — of the CD4 protein piece. Surprisingly, the peptide does not stop the T cell from recognizing the myelin sheath as foreign, but it prevents the T cell from replicating. It may also cause the T cell to self-destruct, Jameson adds.

Only 20 to 40 percent of the mice treated with the peptide developed EAE, compared to between 71 and 83 percent of the untreated mice. Inoculated mice that did develop EAE developed it later, says Korngold, and with less severe symptoms. Because the peptide causes the death of all activated T cells, not just the ones that trigger MS, it shouldn't be given to anyone fighting off other diseases, he says. The peptide is excreted by the body less than 2 hours after inoculation, Korngold adds, so it is unlikely to compromise overall immune function.

One injection may not get rid of EAE (or MS) permanently, says Jameson, because the immune system might produce more cells that react to myelin. Jameson and Korngold hope to begin human MS trials in 12 to 18 months.

"One of the advantages of this drug is that you don't have to redesign it for other diseases," says Korngold. They are looking at other autoimmune diseases, such as rheumatoid arthritis, and at AIDS, and they speculate that the synthesized peptide might also increase the success of bone-marrow transplants and skin grafts. *— D. Christensen*