Tomb of Key Maya Ruler Found

In recent weeks anthropologists in Guatemala, relying on hieroglyphic clues, have discovered the tomb of a man who may have been one of the greatest rulers of Maya civilization. Beyond the ruler's possible stature, the discovery is remarkable in several ways. Reflecting rapid recent advances in understanding Maya writing, it is the first time excavators in the Americas have been guided to such a tomb by written inscriptions. In addition, the find may help to illumine the mysterious collapse of Maya civilization—putting warfare at the center of that process.

Arthur Demarest, an anthropologist at Vanderbilt University and director of the Petexbatun Regional Archeological Project, found the tomb and bejeweled skeleton after sinking a vertical shaft through a 10-meter pile of loose rubble. Several large pottery vessels covered with hieroglyphics and hundreds of obsidian blades surrounded the skeleton. The skull, says Demarest, "was crowned with a beautiful headdress with a mosaic of shell, mother-of-pearl, and jade."

Some of the jade pendants may be incised with the king's name, although Demarest and other anthropologists believe they already know who he was: Ruler 2 of Dos Pilas, the Maya city Demarest is excavating. If so, the team will have found the skeleton of a man Demarest credits with changing the nature of Maya warfare.

Says Demarest: "Prior to Ruler 2, the Maya only captured members of the elite society for sacrifice. But he changed the rules and began conquering other cities, extending his territory." By A.D. 740, 25 years after Ruler 2's death, Dos Pilas formed the center of a kingdom stretching 1500 square miles along Guatemala's Pasión River. Demarest believes this expansion came at a great cost: five decades of warfare, the construction of walled cities and fortified agricultural fields, and ecological tragedy.

"The soil in the rain forest won't sustain the kind of intensive agriculture that walled fields imply," he added. "But the siege type of warfare forced people to concentrate their fields behind walls." Dos Pilas itself fell in A.D. 760, with the citizens apparently huddled behind walls constructed from the stones that once formed the facade of Ruler 2's pyramidal tomb. Only 70 years later, with the land ravaged, the area was abandoned.

Demarest gives considerable credit to a small group of epigraphers (researchers who translate hieroglyphics) that, in the last 15 years, has made great strides in reading what the Maya wrote. "Their translations of the hieroglyphics [which carried the tales of war and inter-city rivalry] are the foundation for our excavations," he said.

One of those epigraphers, Stephen D. Houston of Vanderbilt, is codirector of Demarest's project; the team began looking for Ruler 2's tomb 2 years ago, after Houston and others translated a stela that once stood in front of a pyramid on Dos Pilas' Great Plaza. "It was an extraordinary monument," says Houston, "because it referred to both the king's burial and the place where he was buried"; the location of the tomb is not often included in Maya inscriptions.

Demarest's theory that warfare was central to the Maya collapse is far from universally accepted. As the excavation of the new tomb—and the rest of the project—proceeds he will be looking for further data to buttress that view. In addition to excavating at Dos Pilas his team is digging at six other sites that were conquered by Ruler 2. In fact, he says, that extensive survey is the project's real work; the tomb is merely "icing on the cake." ■ VIRGINIA MORELL

Virginia Morell is a free-lance writer based in Ashland, Oregon.

A New Tumor Suppressor Gene?

Until recently, cancer biologists had difficulty answering a seemingly simple question: Why doesn't everybody get cancer? Over the past few years, however, they've come up with one likely reason—several gene changes appear to be necessary to cause most common cancers. Not only do one or more growth-stimulatory oncogenes have to be activated, but the growth-inhibitory genes that would otherwise suppress tumor formation have to be inactivated.

Now, a team of researchers from the Fels Institute in Philadelphia and the New York University Medical Center has identified a possible new type of tumor suppressor gene that may act to prevent development of lung and kidney cancer. If so, the discovery could one day lead to improved therapies for lung cancer, currently the leading cause of cancer death in the United States.

The candidate suppressor gene codes for an enzyme called phosphotyrosine phosphatase. In the last year or two, the study of these enzymes, which remove phosphate groups from the amino acid tyrosine in proteins, has become one of the hottest areas in cell biology. That's because they may be key components of the machinery that keeps cell growth in check—and therefore likely candidates to be tumor suppressors (also see *Science*, 15 February, p. 744). But until the new results, described by Carlo Croce of the Fels Institute at this year's meeting of the American Association of Cancer Research,* there was no evidence that they play such a role.

The first clue came, Croce says, when his Fels colleague Kay Huebner showed that the tyrosine phosphatase gene, which was originally cloned in Joseph Schlessinger's lab at New York University Medical center, maps to chromosome 3 in a region already suspected of containing one or more suppressor genes because it is frequently deleted in lung and kidney carcinomas. "It's exciting that [the gene] localizes in a region that's deleted in the cancers," says biochemist Edmond Fischer of the University of Washington, the leader of the team that isolated the first tyrosine phosphatase.

Croce and his colleagues also have direct evidence that the tyrosine phosphatase gene is among those that go missing in the cancers. At least one copy was deleted in half of the 12 lung cancer samples they examined and in three of five lines of kidney carcinoma cells. The gene was not deleted, however, in normal lung and kidney cells.

"It's an excellent candidate gene," says John Minna of the University of Texas Southwestern Medical Center in Dallas, whose research also focuses on the genetic changes leading to lung cancer. He adds, however, that it's still a candidate, not a proven tumor suppressor. Croce and Schlessinger agree. Says Schlessinger: "We don't have a proof yet, just a correlation."

Such proof would come, Minna says, if it could be shown that in cancer cells with the deletion, the second copy of the tyrosine phosphatase gene is inactivated by mutations, wiping out all the gene's activity. It would also help, Croce adds, if lung cancer cells can be induced to behave normally by introducing a functional copy of the gene into them.

If that kind of proof is obtained, it might point the way to better therapy for lung cancer, perhaps drugs that can compensate for the missing tyrosine phosphatase activity. And here the enzyme would have a leg up. It's the only one of the possible tumor suppressors identified so far for which the biochemical activity is already known. ■ JEAN MARX

*The results are also in press in the June *Proceedings of the National Academy of Sciences*.