Archaeology

Hey, Pharaoh, make way for the mayor

Pharaohs reigned supreme in ancient Egypt, but the job lacked staying power. Today, how many gold-bedecked pharaohs rule mighty kingdoms and commune with the gods from cloistered palaces? Besides Bill Gates, that is.

On the other hand, some government bureaucrats—who today seem to outnumber grains of sand in the Sahara Desert—achieved surprising prominence alongside Egypt's pharaohs. Witness the remains of a mayoral mansion in an ongoing excavation of a 3,700-year-old Egyptian town near the city of Abydos. The imposing structure included a living area for the mayor and his family, a grain-storage facility, and office space for sundry papyrus-pushers and policy makers.

Previously, archaeologists working at Egyptian sites unearthed several large houses that belonged to people with no claims to royalty. Until now, however, no such structure has been definitively identified as belonging to the mayor of a town or city, says archaeologist Josef Wegner of the University of Pennsylvania in Philadelphia, director of the Abydos dig.

Photos: U. Pa. Mus. Arch. & Anth.

Mayoral mansion emerges at ancient Egyptian site.

"From the evidence thus far, it appears that the mayor [of this ancient town] enjoyed both significant affluence and privileged political power," Wegner says.

His team found the ancient site in 1994. It was occupied during Dynastic Egypt's Middle Kingdom, from about 1850 B.C. to 1700 B.C. Archaeologists have excavated only a few towns like it.

A mortuary temple for King Senwosret III, who died in 1841 B.C., was the centerpiece of the settlement at Abydos. The town also served as a cult center of Osiris, god of the netherworld.

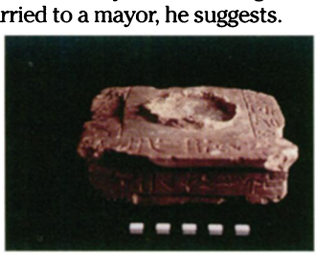
Over the past three field seasons, the team has unearthed about half of the mayor's house, which lies next to Senwosret's temple. Seal impressions on clay fragments from behind the mansion name at least four mayors who held office over a period of around 150 years.

The huge brick and plaster house matches the size of pharaohs' palaces at that time. It opens into a columned hall that spans the structure's width. A central block of rooms and courtyards includes the mayor's residential area. Household artifacts, such as jewelry and cosmetics, point to an affluent lifestyle. Toys, games, and other relics of family life have also turned up.

A granary takes up the structure's back section. This facility may have put the mayor in control of a vital food source for the town of about 1,000 people, Wegner estimates.

Thousands of seal impressions on clay fragments found in working areas of the building reflect intense official activity, Wegner says. Many of these impressions identify a Middle Kingdom princess who may have been married to a mayor, he suggests.

Either the reigning pharaoh appointed him or the mayor inherited the job from his father. Mayors supervised both religious and economic activities. The Middle Kingdom bureaucracy in which these officials operated may have wielded more power than the pharaohs did, although this possibility remains controversial, Wegner says. *—B.B.*



Inscribed limestone fragment, found by researchers last summer, identifies the town's first mayor.

Biology

Feeding hormone finds its partner

Investigators have identified the protein on brain cells that reacts to a hormone known to stimulate eating. Preventing this protein's action may offer new ways to counter obesity, and artificially stimulating it may provide treatments for eating disorders.

Brain cells sport a wide variety of receptors, surface proteins that respond to chemicals in the broth outside cells. Other surface proteins, dubbed orphan receptors, resemble receptors in shape, but scientists haven't identified chemicals that influence them.

In the July 15 NATURE, two research groups report that melanin-concentrating hormone (MCH), a brain chemical that regulates feeding, activates one such orphan receptor. Both groups, one from the University of California, Irvine and one from SmithKline Beecham Pharmaceuticals in Harlow, England, used the orphan receptor as a fishing lure, screening a mix of brain chemicals to see whether any would be snagged by the protein.

MCH was the catch of the day. "This hormone, which has been shown to regulate how much and how often an animal eats, can work only by binding to this receptor," says Olivier Civelli, who led the Irvine group. *—J.T.*

Estrogen may ward off cataracts

Forget about carrots. Estrogen replacement therapy may help preserve a woman's eyesight as she ages, say scientists who have found that the hormone prevents cataracts in rats.

In more than three-quarters of people 75 years or older, the lens of each eye slowly becomes opaque, often leading to full-blown cataracts and blindness. Beginning at menopause, women face a higher risk of the eye disorder than similarly aged men do, suggesting a role for estrogen. Several studies of people have, indeed, hinted that estrogen replacement therapy reduces a woman's risk of cataracts. Yet scientists have obtained inconsistent results when trying to determine whether estrogen protects against cataracts in animals.

In the Aug. 3 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, Robert M. Bigsby of the Indiana University School of Medicine in Indianapolis and his colleagues describe a protective effect of estrogen replacement in female rats that have had their ovaries removed. Without the hormonal treatment, 14 out of 19 rats given a cataract-inducing chemical developed the eye disorder. In contrast, among 27 rats receiving either of two versions of estrogen, only 7 developed cataracts. Bigsby and his colleagues aren't yet sure how the hormone exerts its effect, but they note that cells in rat lenses have receptors that bind estrogen. *—J.T.*

Add three genes, get one cancer cell

What makes a human cell become cancerous? The interplay of three genes can do the trick, report researchers who investigated how many genetic changes it takes to create a human tumor.

Scientists can transform mouse cells into tumor cells by adding just two oncogenes, which are genes that promote cancer by spurring cell proliferation. Yet human cells have proven resistant to such a simple transformation. Radiation, chemical mutagens, and viruses can induce human cells to create tumors, but all three cause so many changes in the cells that it's difficult to discern which are essential.

In the July 29 NATURE, Robert A. Weinberg of the Whitehead Institute for Biomedical Research in Cambridge, Mass., and his team report that two oncogenes combined with an active copy of another gene trigger cancer in several kinds of human cells.

The third gene encodes a key part of an enzyme called telomerase. The enzyme is usually available in mouse cells but not in most human cells, which may explain the need for the extra gene in the latter. By extending the ends of chromosomes, which would otherwise shrink every time a cell divides, telomerase seems to help cells proliferate indefinitely. *—J.T.*

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