were all made of local materials. Neither did agriculture. When Saunders and his colleagues gleaned charred seeds from the sediments and sent them to Kristin Gremillion, a specialist on ancient agriculture at Ohio State University in Columbus, she found no signs of domesticated plants. However, she did identify the wild ancestors of three domesticated plants—*Chenopodium berlandieri*, *Iva annua*, and *Polygonum* spp.—first cultivated in the American Southeast some 4000 years ago. Clearly, Archaic mound builders were already gathering and consuming the wild plants' starchy seeds.

In fact, Saunders says, the rich environment at Watson Brake may deserve much of the credit for making the construction project possible. The site perches on a terrace that, 5400 years ago, overlooked what was then the Arkansas River and an extensive wetland. Animal remains at the site suggest that the mound builders took full advantage of the varied habitats. In addition to hunting deer, turkey, raccoon, and other upland species, Watson Brake's inhabitants collected freshwater mussels and snails and fished both the main channel and the backwaters.

Indeed, the mound builders were adept fishers. Northeast Louisiana University paleontologist Gary Stringer and faunal expert Edwin Jackson of the University of Southern Mississippi in Hattiesburg identified at least nine fish species. The most abundant were freshwater drum, which weigh up to 27 kilograms. Catching these fish, especially during spring and summer when they are spawning and easily captured in nets, is a highly efficient way of obtaining food, notes Stringer.

But even though the team's work provides ample evidence of how the Watson Brake people mustered the food surpluses needed for the construction project, another big mystery remains. Saunders has so far unearthed few clues about what purpose the giant enclosure might have served. Soil sampling inside the earthwork retrieved few artifacts, even though team members screened sediments through fine geological sieves. This suggests that the builders did not conduct ceremonies or other activities within the enclosure.

Stranger still, the excavations to date show little evidence that people occupied the area once the complex was completed. It's as if the builders had nothing to keep them there once the job was done. "I know it sounds awfully Zen-like," Saunders concludes, "but maybe the answer is that building them was the purpose."

–Heather Pringle

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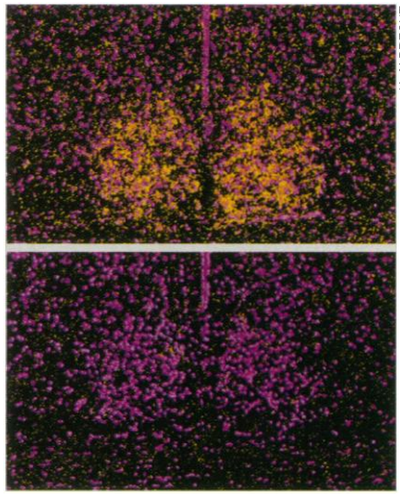
CHRONOBIOLOGY

Gene for Mammals' Body Clocks Found

As anyone who has suffered from jet lag knows, the body's 24-hour biological clock delivers a powerful timekeeping signal. In recent years, clock researchers have made significant progress in understanding the biochemical gears and springs that keep this clock running, largely by identifying a handful of genes that appear key to the process in a few animal and plant species. Now, a team at the Baylor College of Medicine in Houston, Texas, has come up with the first evidence that some of these genes may have been conserved over the course of evolution, indicating that universal mechanisms across all species might keep the clock ticking.


U. ALBRECHT

Night and day. 11 a.m.: New gene, *m-rigui*, is expressed in a mouse's brain (*top*, yellow). 11 p.m.: All is quiet.

In today's issue of *Cell*, molecular geneticist Cheng Chi Lee, developmental biologist Gregor Eichele, and their co-workers report isolating a gene in mice and humans similar to the *period* (*per*) gene of the fruit fly *Drosophila melanogaster*. The *per* gene, which is turned on and off in a daily cycle, appears to work with other genes to create an oscillating mechanism that runs the fly's internal clock (*Science*, 3 November 1995, p. 732). "This is an extremely interesting piece of work," says clock researcher Joseph Takahashi at Northwestern University in Evanston, Illinois. "This is really the first molecular link between the *Drosophila* clock gene story and the emerging mouse gene story."

The Baylor group found the new gene during a hunt for DNA sequences that code for regulatory proteins on human chromosome 17. Out of five such sequences they identified, one was found to code for a protein that shares 44% of PER's amino acid sequence and showed greater similarity in a region of the protein, called the PAS domain, which is a common feature in most clock genes identified so far. They dubbed this gene *RIGUI* and in a similar study in mice found the same gene on chromosome 11, which they dubbed *m-rigui*.

This finding is likely to be bolstered by another paper, from Hajime Tei of the University of Tokyo and co-workers in Japan and California—expected to be published shortly in *Nature*—which will also report the identification of putative human and mouse homologs of *Drosophila*'s *per* gene. Takahashi says that this group's results are so similar that he believes "it's the same gene."

In a clue to the function of *RIGUI*, Lee and his colleagues found that expression of the gene rises and falls according to a circadian pattern, like that of *per*. For example, Lee and his colleagues measured the production of *m-rigui* messenger RNA (mRNA)—a necessary intermediate in protein synthesis—in a part of the mouse brain called the suprachiasmatic nucleus, thought to be the master clock regulator in mammals. They found dramatic swings in mRNA levels over a 24-hour period, even when the animals were kept in the dark. Moreover, the timing of *m-rigui* expression could be altered by shifting the timing of the animals' light and dark cycle. Both of these effects are key tests of an internally controlled circadian pattern.

These findings imply, Lee and his colleagues say, that *RIGUI* may play the same role that *per* does in the fruit fly. They caution, however, that the sequences are not close enough for them to be sure. Steven Reppert, a neurobiologist and clock researcher at Massachusetts General Hospital in Boston, echoes this caution, saying that the partial homology with *per* is not conclusive evidence that *RIGUI* has the same function in mammals that *per* does in insects. He adds that the oscillating expression of *RIGUI* in brain tissues does not prove that it is central to the clock's regulation. The only way to prove this, he says, would be to "knock out the [mouse] gene and see what happens to the circadian rhythms." If they are disrupted, Reppert concludes, the Baylor group's results would represent "a profound finding."

The Baylor group is now embarking on just such experiments. And despite their reservations, Reppert and other researchers agree that the new results are likely to open new doors in clock research. "We will now be able to test molecular models of the clock in mammals," says Takahashi. "Once we get a couple of these genes, the next ones will start falling into place."

–Michael Balter