The effect had been seen indirectly in the 1970s, through its influence on the way a pair of pulsars—neutron stars that emit beams of radio waves—orbit each other. And in a paper soon to appear in the journal *Classical and Quantum Gravity*, a team led by Ignazio Ciufolini of the University of Rome reports detecting the minute effects of frame dragging by Earth's own gravity in the orbits of the so-called LAGEOS satellites. While that detection remains controversial, the Gravity Probe B satellite, to be launched by 2000, will attempt to measure Earth's frame dragging precisely using gyroscopes.

Stella and Vietri thought that a neutron star's powerful magnetic field might create the conditions needed to see frame dragging in the x-ray signal. Like the whirring blades of an egg beater, the magnetic field punches a hole in the middle of the accretion disk. Because the field's north and south poles are generally out of line with the neutron star's spin axis, its whirling lines of force fling matter from the accretion disk out of the disk's plane. There, like a tilted toy top, the material should wobble, or precess, at the same frequency that the reference frame—and therefore the very space in which it exists—is being dragged around the star. Being off kilter is crucial to detecting frame dragging: Within the plane, dragging would produce only indetectable changes in the disk's speed. The wobble frequencies measured by the Rossi satellite are broadly consistent with the amount of dragging expected from the neutron stars' spin rates and the distance at which the material is whirling around them.

Other experts are cautious. "All we can say is that the order of magnitude is right," says Sharon Morsink, a relativity expert at the University of Wisconsin, Milwaukee. The uncertainty stems from a lack of knowledge about the internal structure of neutron stars, which are about 20 kilometers across and roughly as dense as atomic nuclei. Astronomers don't know how that density varies with depth in the star, says Morsink, which would affect the distance of most of the star's matter to the accretion disk and the magnitude of the frame-dragging effect.

Still, a second team, led by Wei Cui of the Massachusetts Institute of Technology, reported that Rossi's measurements of low-frequency x-ray oscillations might have also detected frame dragging around far denser objects: black holes (see graphic on p. 1012). In this case, the researchers don't have a direct measure of the black holes' spins. But if the result is correct, notes Colorado's Begelman, then the whirling of space could be used to measure how fast other black holes are spinning. And that, in turn, could provide clues as to whether spinning black holes are the engines behind such spectacular displays as quasars, plasma jets, and gamma-ray bursts.

–James Glanz

GENETICS

Fast-Forward Aging in a Mutant Mouse?

When the television or the toaster oven breaks down a week after its warranty expires, it's tempting to believe the manufacturer designed it to do so. Similarly, the infirmities and breakdowns that herald old age are so predictable that some scientists believe our genes evolved with their own kind of warranty, one that runs out shortly after reproductive age. These researchers speculate that if they could figure out which genes underlie this planned obsolescence, they might be able to delay it—in effect, extending the warranty. Now, by studying mutant mice that resemble senior citizens by the time they're only 60 days old, a team in Japan may have discovered what such a gene would look like.

Whiskered and wizened. Mice with a mutated *klotho* gene resemble elderly humans.

Defects in the previously unknown gene, called *klotho*, cause mice to die prematurely with a skein of disorders commonly found in elderly humans, such as arteriosclerosis, osteoporosis, skin atrophy, and emphysema. The finding, reported by a team led by Makoto Kuro-o, a physician and molecular geneticist at the National Institute of Neuroscience in Tokyo, in this week's issue of *Nature*, suggests that a specific set of genes suppresses all these age-related conditions. The *klotho* gene, which appears to produce a protein that circulates in the blood, may be the signal that keeps this genetic program turned on while an organism is young, says Kuro-o. And because *klotho*—named after the Greek goddess who spins the thread of life—is also found in people, Kuro-o says he has high hopes "that this notion will be effective for understanding aging mechanisms in humans."

"A wonderful surprise" is how George Martin, a medical geneticist at the University of Washington in Seattle, describes the finding. Indeed, it's such a surprise that skepticism is called for, says Michal Jazwinski, a geneticist studying aging at Louisiana State University Medical Center in New Orleans. "They're fooling around with a gene that's normally expressed in the mouse and getting a syndrome that looks like aging in humans," Jazwinski notes. "That's a bizarre twist"—and a possible sign, he says, that the mutation doesn't accelerate the mice's own aging, but merely kills them prematurely.

Kuro-o started out looking for genetic changes that contribute to hypertension, not hoariness. To test whether one cause of high blood pressure in mice and humans might be overproduction of a protein that plays a role in transporting sodium across cell membranes, Kuro-o injected one-celled mouse embryos with multiple copies of the corresponding gene. These new genes took up random positions in the embryos' DNA, sometimes landing in locations where they disrupted native genes.

Kuro-o noticed that mice belonging to one such strain stopped growing 3 to 4 weeks after birth and died after only 8 to 9 weeks instead of the usual 2 to 3 years. And when Kuro-o examined tissues from the mice under the microscope, he found further changes that, in humans, would be signs of aging: Their arterial walls and other tissues had calcified, their bone density had decreased, the alveoli of their lungs had deteriorated, and they had lost hair follicles and skin thickness. Although these symptoms aren't normally part of the aging process in mice, they might at least make the *klotho* mouse a good laboratory model for the study of human aging, Kuro-o realized.

By homing in on the inserted transgene, Kuro-o's team cloned *klotho* itself and determined from its DNA sequence that it encodes a protein similar to β-glucosidase, an enzyme found in bacteria, plants, and mammals that can break apart fat-soluble molecules such as glycolipids. From its amino acid makeup, Kuro-o believes that the enzyme has an active portion that circulates in the blood, where it may break down glycolipids to generate ceramide, a compound known to help regulate programmed cell death. He's now testing mouse and human blood plasma for signs of the enzyme, and is also looking for variations in *klotho* carried by people with common age-related disorders, such as osteoporosis.

Other scientists will want to know those results, not to mention the enzyme's true function, before they'll fully believe Kuro-o's theory. The *klotho* mutants might be suffering from a metabolic disease rather than aging, says Jazwinski. "Are they seeing premature aging, or simply premature death? This is the essential question." But Kuro-o says he's found no metabolic abnormalities in the mutants. He sums up: "I think these mice died of old age."

–Wade Roush