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U.S.News & WORLD REPORT

NOVEMBER 3, 2003

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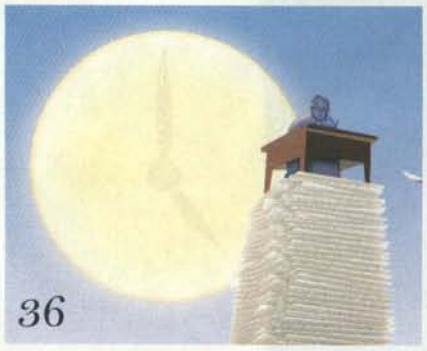

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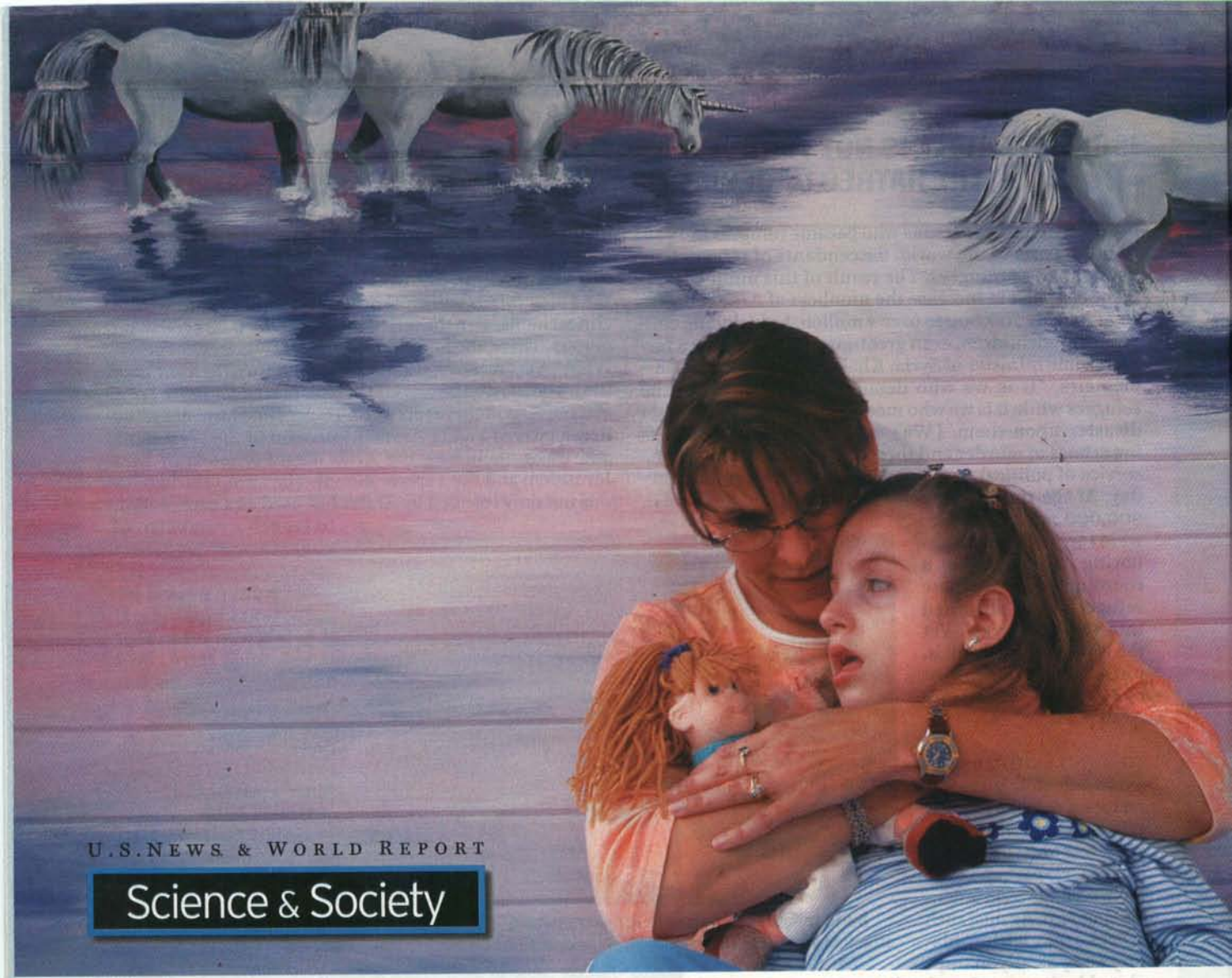


U.S. NEWS & WORLD REPORT

Science & Society

Energy Crisis

Failing 'power plants' inside cells give rise to debilitating diseases

BY EMILY SOHN

Jennifer Lyman will never forget the moment she first realized that something was very wrong with her baby girl. Granted, she'd had her suspicions: Unlike other 3-month-olds, Caroline didn't smile, and she couldn't track moving objects. At times her body seemed as floppy as a rag doll. But that August day, as she looked into her daughter's luminous blue eyes, trying to figure out why she was so fussy, Jennifer suddenly was gripped by the terrifying feeling that her daughter had somehow, deep inside, gone missing. "It was almost like she was staring right through me," she says.

The Lymans, of Cuyahoga Falls, Ohio, rushed Caroline to the pediatrician, who was baffled by her symptoms. Over the next year, they saw "gazillions" more doctors, as their daughter's condition slid inexorably downhill. Caroline was 18 months old and experiencing breath-

Jennifer Lyman's daughter, Caroline, suffers from a mitochondrial illness.

SCOTT GOLDSMITH FOR *USN&WR*

ing problems and violent seizures before the Lymans finally encountered the physician who diagnosed her illness. Biopsies of her skin and muscles revealed defects in her mitochondria, the sausage-shaped organelles, inside cells, that convert food and oxygen into energy. Although rare, her condition had a name: complex 3-4 deficiency.

At the time, four years ago, the Lymans had a hard time getting information about the disease. But that is changing, as new research unravels how defects in these cellular powerhouses trigger a host of illnesses characterized by widely varying symptoms, including muscle weakness, kidney failure, and loss of vision. Experts estimate that fully one in 2,000 babies may inherit some kind of mitochondrial illness. Researchers have also uncovered intriguing clues suggesting that mitochondrial failure may play a role in Alzheimer's disease, stroke, diabetes, and heart disease, among other aging-related ills. Although therapies remain elusive, says Robert Naviaux, codirector of the Mitochondrial and Metabolic Disease Center at the University of California–San Diego, the field is on the verge of any number of breakthroughs. "This is a revolution in medicine that is cutting across all disciplines."

This month, Naviaux and some of his colleagues petitioned the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention in Atlanta, to officially recognize nearly 400 newly described mitochondrial disorders. Currently, the NCHS recognizes only four. That's in part because the field is so new. Scientists first linked a mutation in mitochondrial genes to a disease just 15 years ago.

Today, some 120 defect-causing mutations have been isolated. Mutations can occur in mitochondrial DNA itself—each of the organelles has its own rings of DNA—or in genes in the cell's nucleus that control mitochondria (diagram). These tiny power plants pile by the thousands into every cell in the human body, except adult red blood cells. They are especially prevalent in body tissues that require lots of energy, like the brain, the heart, and other muscle tissue. "You can have a tremendous combination of tissues affected," says Columbia University neurologist Salvatore DiMauro, "You can see . . . heart disease, endocrine problems, hearing loss, you name it."

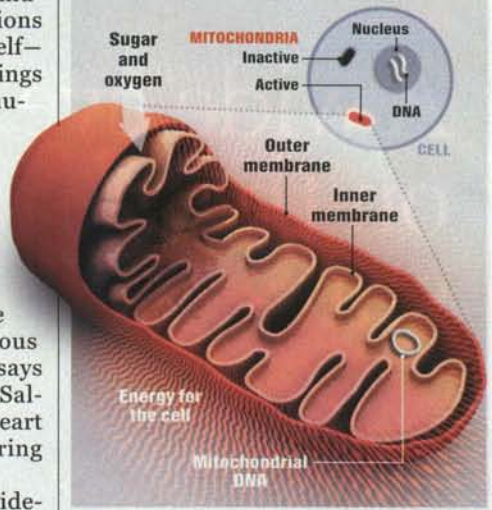
Telltale symptoms. With such wide-ranging symptoms, these illnesses can challenge the diagnostic abilities of even the most knowledgeable physicians. Still, most of the illnesses share a few telltale characteristics: First, problems almost always crop up in mitochondria-rich organs. Typically, at least three organs are affected. And the problems usually appear to be unrelated, at a first glance. Once physicians suspect a mitochondrial disease, they can confirm it with biopsies of affected tissues.

Symptoms experienced by children with a mitochondrial illness often mimic those seen in the elderly, leading some experts to speculate that mitochondrial defects may underlie some of the illnesses of old age. If true, the mechanism by which failing mitochondria causes symptoms would be essentially the same for both groups: defective organelles kill off cells, weakening tissues and organs. The key difference would be in the source of the mitochondrial defects. Children inherit disease-causing mutations; the elderly's arise from a lifetime of accumulated insults to DNA.

New studies suggest that some inherited mitochondrial mutations may actually help people to live longer. Last February, in the journal *Proceedings of the National Academy of Sciences,* California Institute of Technology molecular biologist Giuseppe Attardi and colleagues reported that centenarians were five times more likely to possess a particular mitochondrial "defect" than were younger people. "This is the first well-identified genetic marker associated with longevity," says Attardi.

POWERHOUSE

Mitochondria are tiny structures within cells that convert sugar and oxygen into energy. Defects in mitochondrial DNA or DNA inside the nucleus can cause them to fail.



STEPHEN ROUNTREE—*USN&WR*

As the body metabolizes food, water, and air, it produces "free radicals," which damage DNA, whether in mitochondria or the cell nucleus. Attardi theorizes that the way the mutation might work to prolong life is by helping cells sense which mitochondria contain damaged genes, allowing cells to stitch them back together again.

For now, the biggest challenge in mitochondrial medicine is finding better treatments. Doctors currently prescribe exercise regimens, vitamin cocktails to boost cell function, and various medications to control symptoms. Caroline Lyman, for instance, takes about 20 medications a day to control her seizures among other symptoms. But currently there are no FDA-approved drugs targeted specifically to mitochondrial diseases, nor cures. "As we speak," DiMauro says, "therapy is dismal."

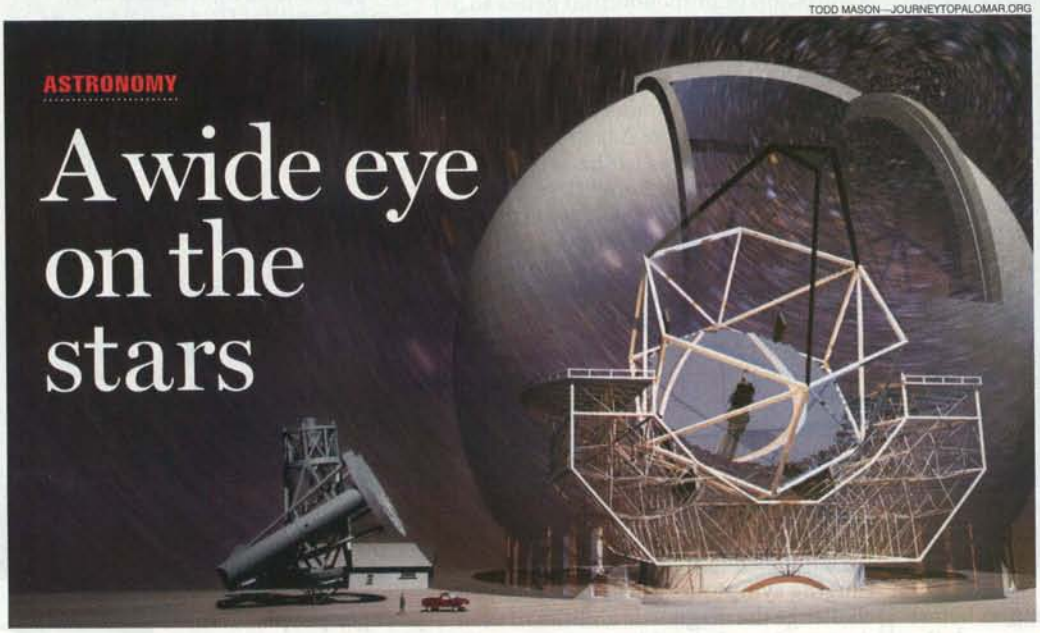
Some small-scale clinical trials are be-

ginning to produce marginally encouraging results. Supplements of creatine, a chemical that occurs naturally in the body, may help certain patients. Other ideas are less conventional. DiMauro's group is experimenting with a technique that aims to take a mutated mitochondrial gene, fix it, then put it back into the nucleus to fool the cell into making proteins usually made in the mitochondria. "It sounds like science fiction," DiMauro says, "but it's been done *in vitro* and it works." It will be many years, however, before these kinds of treatments can be tested in people.

As research continues, Naviaux is heading an international task force aimed at devising diagnostic standards to help doctors recognize the illnesses more quickly. He and others also are putting together an Internet-based registry of patient information.

For her part, Jennifer Lyman is doing her best to raise the profile of mitochondrial disease. As president of the Ohio chapter of the United Mitochondrial Disease Foundation, she organizes an annual 5K race, sells UMDF paraphernalia to raise money for research, and sets up support groups for parents. Caroline, now 6, relies on a feeding tube and can't walk or talk. Her doctors say there's a strong possibility she won't live past the age of 10. Still, her mother takes her along on trips to the grocery store and on other errands. She pushes her in a wheelchair, often with the help of her full-time nurse. When they go out, mother and daughter usually wear UMDF sweatshirts. "So many people say, 'What is mitochondrial disease?' " Lyman says. "That's exactly what I want them to ask." ●

TODD MASON—JOURNEYTOPALOMAR.ORG



LOOKING UP. An artist's view of the Thirty-Meter Telescope shows the existing Palomar for scale.

ASTRONOMY

A wide eye on the stars

BY CHARLES W. PETIT

Ask an astronomer to make a wish and it will be for a bigger telescope. It's been that way since 1610, when Galileo used a lens an inch wide to see mountains on the moon.

England's William Herschel built scopes with light-gathering mirrors up to 48 inches wide in the late 1700s. In the 1840s Irish astronomer William Parsons erected the Leviathan of Parsonstown, a 56-foot-long tube with a 72-inch mirror. And bigger they got: a 100-incher in 1917, the 200-inch Mount Palomar telescope in 1948, and several since the mid-1990s with mirrors 10 meters wide (about 33 feet). Each jump brought waves of new discoveries. Now, eyes are set to open even wider as a project called TMT or Thirty-Meter Telescope picks up speed.

TMT was born earlier this year when two competing U.S. programs merged: the California Extremely Large Telescope project backed by Caltech and the University of California, and the Giant Segmented Mirror Telescope sponsored by the National Optical Astronomy Observatory in Tucson, Ariz. A Canadian consortium is also joining in. A boost came in recent weeks when the Gordon and Betty Moore Foundation—he cofounded Intel—granted $17.5 million in start-up money to Caltech, with an identical grant in the works for the University of California. Set to fulfill a top astronomy priority of the National Academy of Sciences, the behemoth could parse starlight by 2012 from a peak in Hawaii or Chile.

Construction cost is estimated at $600 million. The cash drive is in full swing, with astronomers seeking private and government funding. But the Moore Foundation grant jump-starts detailed design and testing, says Richard Ellis, Caltech's director of optical observatories. "We want to know exactly what we can build. If we can make it bigger for $600 million, we will."

With roughly the area of a baseball diamond, the new scope's mirror will see objects one tenth as faint as the 10-meter Keck, in Hawaii. New optical methods known as adaptive optics will compensate for air turbulence and offer a focus sharper than the Hubble Space Telescope's. "A 30-meter telescope will knock your socks off," says project scientist Jerry Nelson, a University of California astronomer who designed the revolutionary Keck telescope mirrors, made of 36 separate hexagonal segments (the new one may have more than 1,000). Its quarry will include images of the first galaxies that formed and maybe Earth-size planets around other stars.

Of course, that won't be the end of it. European astronomers have a more distant gleam in their eye they call OWL, for Over-Whelmingly Large Telescope, with a mirror 100 meters across. ●

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MIRROR, MIRROR

As telescopes have grown, one-piece mirrors are giving way to segmented designs.