

the properties of exotic matter.

"It's a very big 'if' right now," says Michael Turner, a cosmologist at the University of Chicago. "But this could tell us a lot about the mass of the strange quark—it could tell us a lot about quantum chromodynamics."

A strange star, also known as a quark star, is the last incarnation of a medium-mass sun. (The heaviest stars become black holes.) When a star dies, it collapses under the influence of its own gravity. If the dead star is more than about 1.44 times the mass of the sun, its gravity squeezes together electrons and protons in the stellar material, forming neutrons. At still greater masses, in theory, neutrons might break down into their component quarks. Under enough pressure, half of the neutrons' "down" quarks might turn into strange quarks, creating a more compact type of matter. As *Science* went to press, NASA was planning to announce the possible discovery of two such strange star candidates.

The first, RXJ1856, is a neutron star about 400 light-years away in the constellation Corona Australis. When Jeremy Drake of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Massachusetts, and colleagues analyzed the light coming from the star, they were able to figure out its temperature—information that reveals how many x-ray photons should come off a hot body of any given size. Thus, Chandra's measurement of x-ray brightness reveals how big the star is. And that's the rub. "It's about 50% smaller than the range of sizes neutron stars can be," says Drake. Such dense matter, theorists believe, could exist within a strange star—and nowhere else that they can easily imagine.

The second star, 3C58, is about 10,000 light-years away in the constellation Cassiopeia. Born in a supernova explosion that Chinese and Japanese sky-watchers noted in August 1181, the star had cooled down faster than a neutron star is expected to. "It's too low by a factor of 2 in temperature

and a factor of 16 in luminosity," says David Helfand, an astronomer at Columbia University and a member of the Chandra observation team.

Although both measurements are solid, the interpretations may not be. The too-small star, RXJ1856, might be bigger than calculated if a so-far-undetected hot spot on the star's surface has messed up the calculation of size based upon brightness by making it appear too hot. The too-cool star, on the other hand, could be a neutron star after all if theorists have underestimated the cooling rate of dense neutron matter, a calculation that no one has been able to test in detail. "It's possible that there are other, more prosaic explanations," says Helfand. "I'd like to see other examples [of strange stars] and reduce the chance of an unfortunate geometric conspiracy."

If these two candidates are indeed strange stars, they should help astronomers better understand the nature of subatomic particles. "You can't produce huge chunks of matter at nuclear densities in the lab," says Turner. "There are big uncertainties here, but you take what you can get."

—CHARLES SEIFE

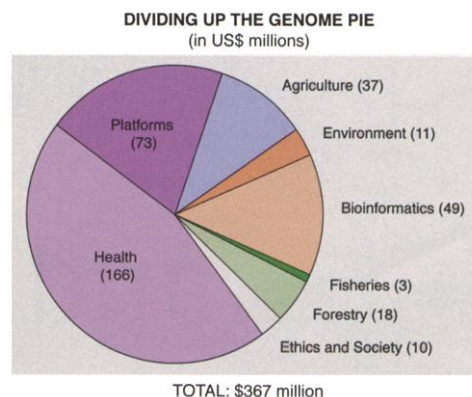
GENOME CANADA

New Awards Bolster Canada's Global Role

OTTAWA—When geneticist Tom Hudson of McGill University in Montreal learned last week that he would receive \$9.5 million to finance Canada's 10% stake in a proposed international research consortium, he wondered for a moment whether he was still living in Canada. "It's unbelievable. This is going to be one of the most high-profile genome projects in the world, and we're the first group funded," enthused Hudson, the director of the Montreal Genomics Centre.

Hudson will be participating in a project to help researchers refine their search for genes implicated in diseases by mapping long stretches of DNA called haplotypes (*Science*, 27 July 2001, p. 583). It's one of 34 projects funded last week by Genome Canada, a nonprofit agency created 2 years ago to boost Canada's capacity in genomics and proteomics likely to benefit key industrial sectors such as health, agriculture, forestry, and fisheries as well as the environment (see graphic). The agency has raised a total of \$400 million from federal, provincial, and industry sources. Combined with an earlier round of awards (*Science*, 13 April 2001, p. 186), the \$195 million committed last week will buy Canada a prominent place in a host of international research consortia, says Genome Canada president Martin Godbout.

Hudson won't know which chromosome

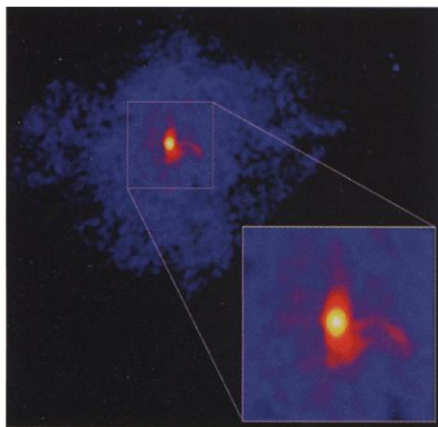


A healthy lead. Health-related research tops Genome Canada's agenda, followed by other economically important sectors.

his group will be mapping until his expected partners, including the Whitehead Institute for Biomedical Research/MIT Center for Genome Research in Cambridge, Massachusetts, and the Sanger Centre in Hinxton, U.K., line up funding from U.S. and U.K. sources. But it's a heady experience to be leading the pack. Except for a few financially modest individual efforts, Canadian scientists sat on the sidelines during the torrid race to sequence the human genome and lamented government cutbacks that tied their hands.

The new funding will allow Canada to move ahead on several fronts. In addition to the haplotype map, the second group of awards includes \$15.75 million for a massive public database on protein interactions (*Science*, 8 June 2001, p. 1813), \$4 million for Marco Marra and Steven Jones of the British Columbia Cancer Research Centre in Vancouver to study the regulatory elements of gene expression, and \$6.3 million for University of Calgary, Alberta, molecular biologist Christoph Sensen to develop a new software program for analyzing genomics data.

The money will also let Canada carry its weight in international circles, says Godbout. A grant of \$6.73 million was awarded to molecular biologist David Baillie of Simon Fraser University in Burnaby, British Columbia, to determine protein function in the soil nematode *Caenorhabditis elegans*, and microbiologist Sherif Abou Elela of the University of Sherbrooke, Quebec, received \$3.75 million to test modified nucleic acid technologies in determining gene function. Both projects will be done jointly with the Karolinska Institute in Stockholm. Genome Canada is negotiating with two other nations to build a consortium to map the genome of the potato, Godbout says, and with Norway to develop a consortium in fisheries. Negotiations are nearly complete on collaborative agreements with the Netherlands and Spain



Odd ball. Born in a supernova's blast, 3C58 seems too cool to be made of normal matter.

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under which scientists from those countries will compete for funding on topics of mutual interest. Talks have also been launched with Germany, Japan, France, and the United Kingdom. "Our vision for the next 5 years will be focused on top-down strategic initiatives," Godbout says. "But we had to first build up the base."

An analysis for the government of published papers showed Canada clinging to the third tier, along with Italy, Australia, and Switzerland, while the United States led the way and the United Kingdom, Japan, Germany, and France were bunched in second place. The investments by Genome Canada should help it move up the ladder, says Francis Collins, director of the U.S. National Human Genome Research Institute, which has recently announced a \$32 million competition to work on the haplotype map. "Until Genome Canada, Canada did not have available the kind of funding capabilities that make it possible to be a player on the big stage," Collins says. The money has been especially useful in providing world-class facilities and equipment, adds Thomas Caskey, president of Houston's Cogene Biotech Ventures Ltd. and head of the 32-member international peer-review panel that waded through the \$1.1 billion worth of applications in the second round.

—WAYNE KONDRÓ

Wayne Kondro writes from Ottawa.

TOXICOLOGY

Fruit Bats Linked to Mystery Disease

Like many scientists who have spent time on Guam, Paul Alan Cox was intrigued by a mysterious malady that stalks the South Pacific island. Victims of the disease that the indigenous Chamorros call "lytico-bodig" may become paralyzed, develop tremors and move sluggishly, or slide into dementia. Unmasking the cause of this invariably fatal neurodegenerative disorder could offer insights into major killers such as Parkinson's and Alzheimer's. But lytico-bodig is dying out—and threatening to take its secrets to the grave. "I felt we were missing a chance to solve an enigma," says Cox, an ethnobotanist who runs the National Tropical Botanical Garden in Kalaheo, Hawaii. Now, he has fashioned a provocative hypothesis around a pair of remarkable coincidences.

In the 26 March issue of *Neurology*, Cox and neurologist Oliver Sacks correlate a sharp rise in flying fox consumption among the Chamorros after World War II with a presumed crest in the disease. They also found that much of the decline in lytico-bodig happened as flying foxes were hunted nearly to

oblivion. Cox and Sacks, of the Albert Einstein College of Medicine in New York City, speculate that Guam's flying foxes may be biological weapons with wings, chock-full of neurotoxins accumulated in their tissues from a favorite food: cycad seeds.

The perplexing Guam disorder first intrigued scientists in the early 1950s, when U.S. investigators reported that the Chamorro population was afflicted with a form of amyotrophic lateral sclerosis (ALS), or Lou Gehrig's disease, at a rate roughly 100 times the global average. Some patients displayed the tremors and rigidity of parkinsonism often coupled with an Alzheimer-like dementia—a unique malady called parkinsonism-dementia complex (PDC). Eventually, researchers concluded that the disparate symptoms were all part of the



Recipe for disaster? Guam's flying foxes, like this one prepared in coconut cream for a traditional Chamorro feast, may be laced with cycad neurotoxins.

same disease, lytico-bodig. The U.S. National Institutes of Health set up a center on Guam in 1956 to search for a cause.

Nearly a half-century later, the culprit remains elusive, with suspects ranging from faulty genes to mineral deficiencies and parasites to neurotoxins. Only a handful of Guamanians born after 1960 are known to have developed the disease, and according to neurologist John Steele of the University of Guam, the principal manifestation now is of dementia in women late in life.

Cycad seeds have long been among the suspects. In the traditional Chamorro diet, the seeds of *Cycas rumphii* Miquel, a tree native to Guam, are ground into flour for a kind of tortilla. The Chamorros know that the seeds are toxic and rinse the flour several times to remove the poison. In the lean years after World War II, however, cycad tortillas were a staple of the Chamorro diet, and researchers have speculated that the large quantities ingested, coupled with incomplete detoxification of the flour, might have delivered enough cycad neurotoxin to trigger ALS-PDC. But lab animals fed the most

likely cycad neurotoxin, cycasin, fail to develop an illness resembling ALS-PDC.

"I was puzzled like everybody else," says Cox, "but thought there might be something that ethnobotany could bring to the table." He learned that the Chamorros relish the meat of the flying fox, a kind of fruit bat that on Guam subsists largely on cycad seeds. Cox noted that after World War II, hunting with firearms largely replaced the traditional technique of snaring the animals in thorny vines. The shooting coincided with an apparent sharp increase in ALS-PDC. The hunting took a heavy toll: One of Guam's two species of flying foxes had vanished by the mid-1970s, and the other had dwindled to fewer than 100 individuals. As the bats grew scarce, so did cases of the disease. Last year Cox met Sacks in New

York and sketched out his scenario. "My first thought was that it was charming, ingenious—and unlikely," says Sacks. But Sacks eventually agreed that at least part of the fatal disease's decline could be tied to the demise of a furry flying alembic for neurotoxins.

Some veteran ALS-PDC researchers, however, view the hypothesis as speculation built on a shaky foundation. "If we *knew* that cycad was the cause of the disease, the paper would be enticing," says Ralph Garruto, a biomedical anthropologist at the State University of New

York, Binghamton. Peter Spencer, a neurotoxicologist at the Oregon Health and Science University in Portland, argues that if cycasin was indeed the culprit, the bat theory won't fly: The toxin is soluble in water and thus would not have built up in flying fox tissue. And preliminary inquiries by researchers on Guam suggest that some ALS-PDC patients may never have consumed flying fox.

Sacks says that the flying fox hypothesis was not conceived as an "exclusive cause" for ALS-PDC. He points to unpublished data on a fat-soluble neurotoxin in cycad that may be a candidate for biomagnification. If that were to pan out, "a 1-pound bat is as good as half a ton of seeds," he says.

Cox's group has launched a feeding trial with a common species of flying fox in American Samoa. "We'll see what does bioaccumulate," he says, and compare the toxicological profile to decades-old archival tissue from flying foxes taken on Guam. Whatever they find, notes Spencer, "it's essential that work continue on this tremendously important disease." Time is clearly running out.

—RICHARD STONE