RANDOM SAMPLES

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Turtle Project Scores a Success

They may be the most expensive baby turtles ever: Last week, biologists released turtle hatchlings from eggs laid in two nests along the Padre Island national seashore in Texas, nests made this spring by the only two Kemp's ridley sea turtles known to return to their experimental hatchery so far during the 18-year history of the $4 million project.

"It is a first glimmer of hope that we may be able to see a secondary nesting colony," says biologist Donna Shaver of the National Biological Service. But researchers caution that the return of a few turtles doesn't mean the project is a proven tool for restoring turtle populations.

The controversial program was launched in a desperate effort to save the animals, the most endangered of all sea turtle species. Decimated by egg robbers and shrimpers' nets, only an estimated 3000 adults remain today. Between 1978 and 1988, scientists collected more than 22,000 eggs from the species' only nesting colony at Rancho Nuevo, Mexico, and shipped them to Padre Island. They released hatchlings on the beach so they would "imprint" the location, then raised them in captivity for 9 months to give them a head start against predators before releasing them, hoping they would return after they reached sexual maturity in 10 to 15 years.

NOTTINGHAM/TEXAS PARKS AND WILDLIFE DEPT.

Pricey turtles. Kemp's ridleys hatch on Padre Island.

The experiment bogged down because of early missteps in raising and releasing the turtles and an inability to judge its effectiveness. Eventually even its leading scientists—worried when the shrimp industry began touting such programs as a way to make up for turtles killed in its nets—turned against the project (*Science*, 1 May 1992, p. 614). Former project leader Jack Woody remains skeptical: "We get more for our dollar by far by protecting beaches and ensuring the use of turtle excluder devices" to help turtles escape from the shrimp nets, he says.

Tay-Sachs Protein Modeled

An enzyme that helps bacteria digest complex sugars has—with some window-dressing from a computer—allowed scientists to model the long-elusive protein behind Tay-Sachs disease.

Tay-Sachs, an inherited degenerative brain disorder, usually appears in infancy and is often fatal by age 3. But some people get a less severe version that occurs later in life. Scientists have long known that the various forms of the disease can be traced to different mutations in an enzyme important in the brain. But the guilty protein has proved difficult to crystallize, a necessary step for determining its structure and the effects of the mutations.

Researchers from Germany, Israel, and Greece report in the July *Nature Structural Biology* that they have gotten around that problem by crystallizing a bacterial enzyme that is closely related to the Tay-Sachs protein. Ivo Tews and Constantin Vorgias of the European Molecular Biology Laboratory in Germany (Hamburg outstation) and their colleagues superimposed a computer's best estimate of the human protein onto their structure of the bacterial protein, then adjusted the human model to achieve the best fit.

The results support what scientists have surmised: The mutation for the severe version of Tay-Sachs appeared in the centrally located active site of the enzyme model, while the mutations causing the less severe forms were in less critical regions on the outside of the protein.

McGill University geneticist Feige Kaplan has used the computer model in research probing the Tay-Sachs enzyme and says it's consistent with experiments with the real protein. She cautions, though, that the model is one step forward on a long road: "A cure is not just around the corner."

NSF Awards Program Goes Electronic

No paper. That's what the National Science Foundation (NSF) promised this month as it plunged into its first awards program to be handled entirely on-line, a scheme that could be a first for a federal agency. If the $5 million Recognition Awards for the Integration of Research and Education (RAIRE) program succeeds in its electronic efforts, other NSF grant programs could be paperless by year's end.

Earlier this month over 100 institutions sent a 3-page application to RAIRE electronically (*Science*, 28 June, p. 1868); next reviewers will access them using a password and record their comments electronically. Their reactions will be transmitted to universities to help them decide whether to submit a longer application in November.

NSF's David Schindel says the "main unknown" is whether people will find downloading materials too bothersome and ask NSF to mail them instead. But so far, they seem to like the project. "A tenth of a second after he pressed the send key," Schindel says one administrator told him, "a proposal number appeared on the computer screen, and he received e-mail confirmation that the proposal had been received. That would ordinarily take 3 weeks." NSF had already begun weaning research grant applicants from paper through its FastLane project, but no program has yet tried an all-on-line solicitation. That may happen this fall, Schindel says, if RAIRE works out.

Jigsaw Puzzle Gene

They are socially adept, musically oriented, and able—and eager—to read, but people with Williams syndrome are also mentally retarded and can't do spatial tasks such as jigsaw puzzles. The inherited disease causes a bewildering mosaic of symptoms, including a narrowed aorta, shortness, fine bones, and elfin faces.

Now the discovery of a gene that affects spatial cognition and lies next to a gene already implicated in the vascular symptoms is helping geneticists make sense of the illness. "[The gene] explains some of the clinical features," comments Jerome McCombs, a cytogeneticist at the University of Texas Medical Branch in Galveston. "And what is remarkable is how specific the cognitive defect is," adds Mark Keating, a molecular geneticist at the University of Utah in Salt Lake City, who led the groups reporting the work in the 12 July issue of the journal *Cell*.

The newly discovered *LIM-kinase1* gene sits on chromosome 7, right next to the gene for elastin, a protein that makes blood vessels, skin, and lung tissue resilient. In 1993, Keating's group showed that the *elastin* gene was missing or mutated in people who had only the vascular problems associated with Williams syndrome. People with the cognitive symptoms lack a larger piece of chromosome 7, so his team looked in that piece for a kinase gene, because they knew that mice with mutated kinase genes have problems with spatial learning.

"*LIM-kinase1* is the next gene down the line," Keating reports. Its DNA sequence actually merges coding for two protein elements: a kinase part, key in intracellular signaling, and a LIM part, which resembles proteins associated with development, Keating explains. The group now hopes to find people with a different subset of symptoms so they can home in on more Williams syndrome genes.