moving from the list 23 other endangered salmon and steelhead populations that share waters with hatchery fish.

The decision runs counter to salmon science, according to many biologists. "A whole sheaf of scientific studies" from the past 20 years suggests that hatcheries cause problems for wild fish, says Robin Waples of the NMFS Northwest Fisheries Science Center in Seattle. Hand-reared fish may be genetically similar to their wild cousins, for instance, but they often aren't as skilled at foraging or avoiding predators. As a result, interbreeding between hatchery and wild fish can produce less fit mongrels.

Waples and other NMFS scientists hope to hash out the biological significance of these differences by next September, when the agency plans to release a new policy on the role that hatcheries should play in salmon restoration and then decide whether to delist some of the imperiled populations. NMFS will ponder whether hatcheries could help save wild populations, for instance, by rearing only eggs taken directly from wild fish. Waples says the agency will also consider whether seemingly plentiful runs that are composed largely of hatchery fish would survive on their own, as the ESA requires.

–JOCELYN KAISER

MARINE CONSERVATION

Reserves Found to Aid Fisheries

When California officials began holding public meetings last year on a controversial state plan to ban fishing in some coastal waters, some anglers raised a stink. Their anger, which included flinging dead fish at one session, stemmed in part from what they said was insufficient evidence that closing some fishing grounds would actually help boost catches in nearby areas.

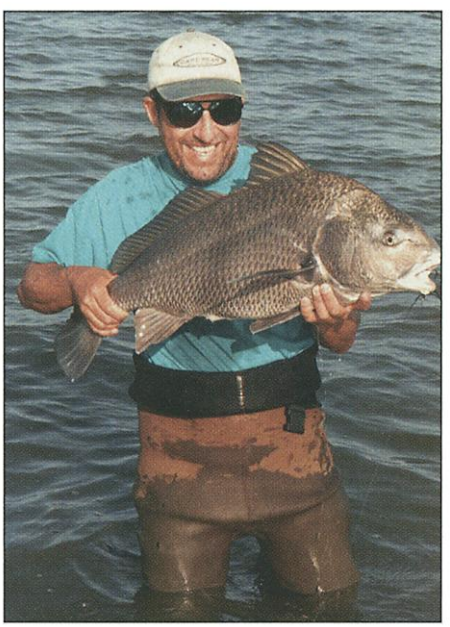
New findings presented on page 1920 could help clear the air. An international team of marine scientists reports marked increases in commercial catches and the number of trophy fish caught by sport anglers in and around small reserves in the Caribbean and off Florida. The authors say the results confirm the validity of their models and, more importantly, lend credence to global efforts to establish new reserves. The findings "will help remove a major logjam in the debate," says lead author Callum Roberts, a biologist at the University of York, U.K.

Some scientists, however, caution that closing off some areas won't be enough to restore healthy fisheries. And some influential fishing groups and politicians remain on the offensive, saying that reserves in U.S. waters threaten public access to the seas.

Studies have shown that closing swaths of the sea to human activity can produce sizable ecological benefits within the reserve, from more diverse sea life to bigger schools of fish. But "whether reserves have spillover benefits is one of the most hotly debated and least studied issues in marine reserve research," says Karen Garrison, a reserve advocate with the Natural Resources Defense Council in San Francisco, California. Such studies are difficult, she notes, because researchers must find areas where they can compare catches before and after a reserve was established, and monitor all the relevant variables, from how long fishers work to changing ocean conditions.

In their study, Roberts and colleagues from the U.S. National Marine Fisheries Service and the University of the West Indies, Barbados, focused on a 5-year-old network of reserves off the Caribbean island of St. Lucia and an area off NASA's Cape Canaveral rocket launching site in Florida that had been closed for nearly 40 years. In St. Lucia, the researchers concluded that the reserves, which cover about one-third of a long-used coral reef fishing ground, increased catches in nearby areas by up to 90%, compared to prereserve numbers. Off Florida, they found that sport anglers fishing around a 40-square-kilometer area closed in 1962 for security reasons have landed a disproportionate number of world- and state-record fish from three species. Since 1985, for instance, most Florida record red and black drum came from the area.

The study confirms that reserves can serve as sheltered nurseries for surrounding waters, say the researchers. And although the studied reserves were small, Roberts says the findings—when combined with



Drum roll. Researchers say that a Florida reserve helps produce trophy fish like this black drum.

CREDITS: (TOP TO BOTTOM) HST/NASA; CALLUM ROBERTS

ScienceScope

Rocky Missions Returning a Mars soil sample to Earth is an enticing prospect—and an expensive one, given its $2 billion price tag. NASA tentatively plans a 2011 launch with a 2014 return. Now a National Academy of Sciences panel argues that the agency should conduct not one, but 10, sample-return missions. In a Mars science report released 26 November, the panel, chaired by John Wood of the Harvard-Smithsonian Center for Astrophysics, concludes that one sample won't be enough to "unlock all of the planet's secrets." Instead, the first mission should be a "trailblazer" for a more extensive program. That expensive vision, however, is unlikely to win support from the Bush Administration.

Preemptive Strike? After years of resisting change, the Russian Academy of Sciences (RAS) earlier this month approved a new charter that trims the number of its divisions from 18 to 10. That will eliminate several plum positions in RAS's governing presidium. In true Soviet fashion, however, academy members reelected the only candidate on the ballot—President Yuri Osipov—to an unprecedented third term (*Science*, 2 November, p. 974).

More substantial changes may be afoot for the 325-odd RAS institutes. "We must find out which are effective and which are not," says former science minister Vladimir Fortov. That would enable the academy to funnel scarce resources to worthy institutes. Observers expect the academy to unveil other specific reforms by May.

New Chief Developmental biologist Peter Gruss has been elected president of Germany's Max Planck Society, the nation's major science group. Gruss, 52, is currently head of the department of molecular cell biology at the Max Planck Institute (MPI) for Biophysical Chemistry in Göttingen. He will take over next June from biologist Hubert Markl, who plans to return to research at the University of Konstanz after leading the society for 6 years. Although he lacks Markl's administrative experience, Gruss should bring "a fresh perspective," says Tobias Bonhoeffer, director of the MPI for Neurobiology in Martinsried. Like Markl, Gruss favors allowing research on human embryonic stem cells in Germany—an issue the government is still debating.

Contributors: Andrew Lawler, Jocelyn Kaiser, Marina Astvatsaturyan and Vladimir Pokrovsky, Gretchen Vogel

sketchier data from bigger closures—"make a compelling argument that reserves will work across a wide spectrum of scales, in many geographical locations, and for many different fisheries."

One researcher, however, cautions that reserves are just one tool available to fisheries managers. "The question is, 'What role should [reserves] play in the mix of regulatory options?' " says Ray Hilborn, a fisheries biologist at the University of Washington, Seattle. Hilborn argues that less severe moves—from closed seasons to size limits—could produce equally significant fisheries improvements. He also questions whether reserves can work wonders for some fisheries that are already highly regulated, such as those in the United States.

Some sport and commercial fishing groups have challenged the California effort and asked the Bush Administration to reconsider a major reserve in the Hawaiian Islands (*Science*, 8 December 2000, p. 1873). In August, the American Sportfishing Association (ASfA) and other groups also convinced Senators John Breaux (D–LA) and Kay Bailey Hutchison (R–TX) to introduce the Freedom to Fish Act (S. 1314), which would require government planners to make reserves as small as possible. "Blanket closures and no-fishing zones should not be the first solution, but rather the last," says an ASfA statement.

Roberts agrees that reserves should be tailored to specific ecosystems and public goals. But "fishers have nothing to fear from reserves," he says. The real danger "is a future without them." **–DAVID MALAKOFF**

GENETICS

Fragile X's Missing Partners Identified

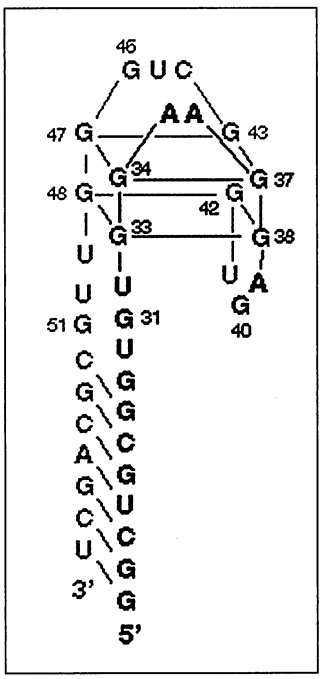
Three research teams have begun to decipher the molecular signals that lead to fragile X syndrome, one of the most common causes of mental retardation. People with this syndrome carry a mutated version of a gene on the X chromosome, and since 1991 researchers have known that this mutation blocks the production of a certain protein. But how that deficit causes the syndrome remains mysterious. Two new reports finger a set of messenger RNA (mRNA) molecules in the brain as crucial targets of the missing protein—findings that suggest these mRNAs play key roles in helping neurons communicate with each other. The third team reports that disabling a gene for one of these mRNAs eliminates symptoms of the mutation in a fruit fly model of fragile X, suggesting that eventually it may be possible to treat the syndrome.

Together, the studies could help explain the role certain fragile X–related proteins play in the brain. "This is a major step toward understanding the biology of fragile X and understanding the biology of higher cognitive functions," says geneticist Harry Orr of the University of Minnesota, Twin Cities.

The genetic mutation in fragile X syndrome gives the chromosome a distinctive look: One arm appears to be hanging on by a thread. The mutation prevents the gene from producing the so-called fragile X mental retardation protein (FMRP). In addition to mental retardation, people with the disorder often have elongated faces, autism, and attention deficit disorder, among other symptoms. On the microscopic level, people with the syndrome have misshapen dendrites, neural projections that receive signals from other neurons.

Scientists have struggled to find a link between the missing protein and these symptoms. In normal brain cells, FMRP binds to strands of mRNA, molecules that transmit blueprints for protein production. But with millions of differently shaped RNA molecules floating around in the cell, researchers have had trouble figuring out which are controlled by FMRP.

To crack this problem, a group led by neuroscientist Robert Darnell at Rockefeller University in New York City manufactured trillions of different versions of mRNA. By seeing which stuck to FMRP and washing away the rest, the team narrowed its search down to a family of RNAs called G quartets, which are shaped like cubes on a stick—imagine square Popsicle flavored ices. The team then searched databases of the human genome to find sequences that produce similarly shaped mRNAs, suspecting that these mRNAs are targets of FMRP in the brain.



Syndrome subjects. Some mRNAs with this G quartet shape are misregulated in fragile X syndrome.

Working independently, geneticist Stephen Warren of Emory University in Atlanta and his colleagues ground up mouse brains and mixed them with antibodies that specifically grab FMRP out of the mush. The team counted more than 400 mRNAs that came along with FMRP for the ride. When the two teams compared results, they identified a dozen mRNAs that have both a G quartet structure and cling to FMRP.

Warren's group then looked in the brains of people with fragile X syndrome and found that eight of those mRNAs were either overexpressed or underexpressed. "These are the first eight mRNAs ever identified that are really different in fragile X patients," Darnell says. Both teams report their results in the 16 November issue of *Cell*. Figuring out what these mRNAs do is the next step, but Darnell speculates that they build proteins that help brain cells communicate with each other.

Knowing which mRNAs FMRP interacts with could potentially lead to treatments for the disease, suggests a third study. *Drosophila* have a gene similar to the FMRP-producing gene, and scientists can disable it to induce a version of fragile X syndrome. Neurogeneticist Kendal Broadie of the University of Utah in Salt Lake City and colleagues made fragile X fly mutants and noticed an increase in concentrations of the fly version of map1b—a protein built by one of the mRNAs Warren's team found to be overexpressed in people with fragile X syndrome. When the team increased the levels of map1b in normal flies, fragile X symptoms appeared—a sign that map1b is behind the fly syndrome. But the clincher came when the team simultaneously disabled the fragile X gene and, to compensate, hobbled the gene that makes map1b. The researchers ended up with normal flies, they report in the 30 November issue of *Cell*.

"This was completely startling," Broadie says. "There were no defects left over whatsoever." That suggests, he says, that a single mRNA target might be the key to explaining what causes fragile X syndrome, at least in *Drosophila*. If the same proves true in humans, "that takes us to the point where we might be able to treat and cure" fragile X syndrome.

Other experts are more circumspect about treatment possibilities. "At this time," says Orr, "it is a bit simplistic to think that the whole story of fragile X syndrome is controlled by the ability of this protein to regulate a single mRNA," especially considering the hundreds of potential targets found by Warren's group. Still, Orr agrees with other experts that the recent convergence of fragile X research is a major step toward understanding what causes the disorder.

–EMILY SOHN

Emily Sohn is a writer in Minneapolis, Minnesota.

CREDIT: R. DARNELL/ROCKEFELLER UNIVERSITY