Tugen Hills team, which has dubbed its specimen *Orrorin tugenensis*.

The Ethiopian fossils were found between 1997 and early this year by an Ethiopian-American team led by Yohannes Haile-Selassie and Tim White of the University of California, Berkeley. The dates—determined by argon-argon dating and confirmed by paleomagnetic measurements and analysis of animal bones found in the same sediments—place the teeth and bones around the time when most geneticists believe that humans and chimpanzees split from a common ancestor, between 6 million and 9 million years ago. "But the closer you get to the branching point, the harder it is to say what the characters are that define hominids," says Rick Potts of the Smithsonian Institution in Washington, D.C.

Describing their find in two papers in this week's issue of *Nature*, the team has named its specimen *Ardipithecus ramidus kaddaba*, as it may represent a subspecies of 4.4-million-year-old *Ardipithecus ramidus* fossils first reported from Ethiopia in 1994. The researchers argue for hominid status for *Ardipithecus ramidus kaddaba* partly because of its lower canine teeth, which in cross section are diamond-shaped like those of later hominids rather than V-shaped like those of apes. They also note that the foot bone has features—such as a joint's orientation—similar to those of later hominids, including the famous 3.5-million-year-old Lucy. This orientation "suggests bipedality," says Juan Luis Arsuaga of the University of Madrid, although "the evidence is still weak" because so far it is based primarily on a single bone. Fred Spoor of University College, London, agrees that the jury is still out on hominid status: "Neither this nor the *Orrorin* paper make a really watertight case," he says.

The *Orrorin* and *Ardipithecus* teams assert that each other's fossils could represent an ancestor of chimps or other apes, rather than one of our early human ancestors or cousins. Figuring out who's right is hard: Although numerous hominid species have been unearthed over the years, no fossils representing the chimp evolutionary line have ever been discovered. "Our obsession over the earliest hominids is a bad habit," says Daniel Lieberman of Harvard University. "Finding the earliest known chimpanzee would be just as exciting." To help resolve the debate, Haile-Selassie says the team will go back into the field in November to search for more fossils.

Whatever they dig up could offer an important piece to the evolutionary puzzle. "It is a mistake to feel that one has to squeeze this [find] into the category of human or chimp ancestor," says Bernard Wood of George Washington University in Washington, D.C. "Just to have a fleeting glimpse of these creatures is exciting."

–MICHAEL BALTER AND ANN GIBBONS

CREDIT: (BOTTOM) ULRIKE PREUSS/*BMJ*

SCIENTIFIC PUBLISHING

Journals Offered Free To Poorest Nations

Researchers and doctors in poorer nations will get free or low-priced electronic access to nearly 1000 biomedical journals. The six largest commercial journal publishers agreed this week to open their Internet vaults to universities, laboratories, and health agencies in nearly 100 nations under an initiative led by the World Health Organization (WHO).

Scientists and health workers in the developing world have long struggled to obtain timely, affordable access to information on new findings and therapies. Many journals are too expensive or arrive months after publication. The 3-year pilot project, set to begin early next year, "is perhaps the biggest step ever taken towards reducing the health information gap between rich and poor countries," WHO director Gro Harlem Brundtland said at a 9 July press conference in London announcing the deal.

The six publishers—which publish 80% of the world's top 1240 biomedical journals—have agreed to let WHO set up an Internet portal through which approved institutions can retrieve papers. Initially, says Barbara Aronson, a librarian at WHO's Geneva headquarters, the portal will be free to more than 600 institutions in 63 of the world's poorest nations, mostly in Africa, with per capita incomes of less than $755 annually. Later, WHO hopes to arrange deeply discounted subscriptions for institutions in about 40 nations, including some in Eastern Europe, with per capita annual incomes of up to $3000.

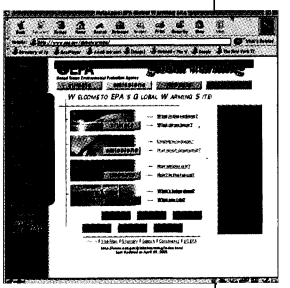
Health InterNetwork, a United Nations program led by WHO, will help institutions get the necessary hardware, Internet connections, and training. Participants declined to put a price tag on the project, estimated by

Free deal. WHO's Brundtland, right, and Blackwell's Jon Conibear, left, unveil the plan.

ScienceScope

Data-Quality Jitters A federal proposal that would allow citizens to critique data disbursed by government agencies is troubling some researchers. Its backers in Congress and industry make no bones about wanting to use the rules to pick apart reports and Web sites (below) on hot-button topics such as global warming and toxic chemicals.

The guidelines, proposed by the White House Office of Management and Budget (OMB) in the 28 June *Federal Register*, call for agencies to ensure the "quality, objectivity, utility, and integrity" of information they disseminate, including "opinions." Agencies would have to set up "mechanisms" for "citizen review" so the public can "obtain correction of information." OMB crafted the plan in response to language tucked into a funding bill by Representative Jo Ann Emerson (R–MO) and other lawmakers last fall.



Researchers are particularly alarmed by a requirement that any scientific results "be substantially reproducible upon independent analysis of the underlying data." That could force academics to turn over their data to anyone who asks, worries Wendy Baldwin, extramural grants chief at the National Institutes of Health. Adds one academic lobbyist: "It's an open invitation to industry to come in and trash" the work of scientists. Comments are due by 13 August.

Planet Finders The ongoing battle over whether to send a spacecraft to Pluto (*Science*, 17 November 2000, p. 1270) is the most obvious sign that U.S. planetary scientists are at odds over how to spend limited dollars. Next week, senior researchers will kick off a sweeping 10-month review of solar system exploration aimed at deciding which missions are most needed.

The two dozen planetary scientists want to come up with "a plan written by the community" rather than NASA or White House officials, says retired astronomer Michael Belton, who will lead the panel. Modeled on the astronomy decadal survey, the National Research Council study is funded by NASA and due next May. The results could replace NASA's current planetary science plan, which researchers criticized last year for lacking a clear set of science goals.

publishing industry analysts as being worth millions of dollars at normal market rates.

The list of available journals does not—so far—include those produced by smaller publishers, including such prominent publications as *The New England Journal of Medicine*, *Science*, and *Nature*. "We decided to go for the largest publishers first [rather than] the most prestigious journals," Aronson says. Many big-name publications already have their own low- or no-cost distribution programs, but Aronson said that their participation in WHO's project would be welcome. Donald Kennedy, editor-in-chief of *Science*, says the journal would consider all invitations and is already weighing whether to join one effort to make scientific information more easily available in the developing world.

The six cooperating publishers are Reed-Elsevier, Wolters Kluwer, Blackwell, Harcourt General (which is merging with Reed-Elsevier), Springer-Verlag, and John Wiley & Sons. The Open Society Institute, a charitable group founded by finance billionaire George Soros that already runs its own journal distribution program, will help identify eligible institutions. The *British Medical Journal*, published by the British Medical Association, also played a role in developing the initiative.

–DAVID MALAKOFF

CELL BIOLOGY

Fresh Molecule Whets Appetite

As appetite researchers feast on a banquet of molecules that control eating behavior, yet another joins the spread. This one comes from a field that has produced samplings of other proteins that transmit signals—but none before that governs hunger.

"It's a different kind of molecule for influencing these pathways," says Jeffrey Flier, an endocrinologist at Beth Israel Deaconess Medical Center in Boston. The 20 to 25 players identified so far are either neuropeptides that transmit messages or cell surface receptors that register incoming information. By contrast, this new molecule, called syndecan-3, apparently helps one of these neuropeptides activate a receptor. Fiddling with the newfound regulator, researchers suggest, could open therapeutic avenues for controlling appetite.

Although syndecan-3 is an unexpected player in appetite control, it works with a molecule well known to regulate body weight, report Ofer Reizes, a postdoc in Merton Bernfield's lab at Harvard Medical School in Boston, and their colleagues in the 13 July issue of *Cell*. Syndecan-3 belongs to a family of proteins that grab signaling molecules and attach them to receptors. Long thought to act as molecular glue, these proteins—and in particular, their side chain appendages—have recently been shown to help send signals, some of which are crucial for embryonic development. And that's what the Bernfield team was exploring when it discovered syndecan-3's appetite-control powers.

The researchers were curious about the developmental role of a cousin of syndecan-3, called syndecan-1. To test its function, they engineered a mouse to overproduce the molecule. "We expected to see some sort of developmental anomaly—perhaps an extra digit," says Reizes. But the mice grew normally, at least for the first 6 weeks. Then they began to gain excess amounts of weight. By adulthood, they were obese.

The animals' behavioral and biochemical abnormalities resembled those found in obese mice with defects in an established appetite-control network called the melanocortin system. A key protein in the system, called the melanocortin-4 receptor (MC-4R), receives competing signals that tell an animal whether to eat. The receptor can bind a "satiety peptide" that activates the receptor and produces a feeling of fullness. Or it can bind an "antisatiety peptide" that obstructs the satiety peptide and induces hunger.

The researchers suspected that syndecan-1 was interfering with MC-4R, so they tested how cultured cells operate with and without the syndecan. They already knew that the antisatiety peptide by itself inhibits activation of the receptor. But when the researchers added syndecan-1, the receptor's activity took a deeper dive.

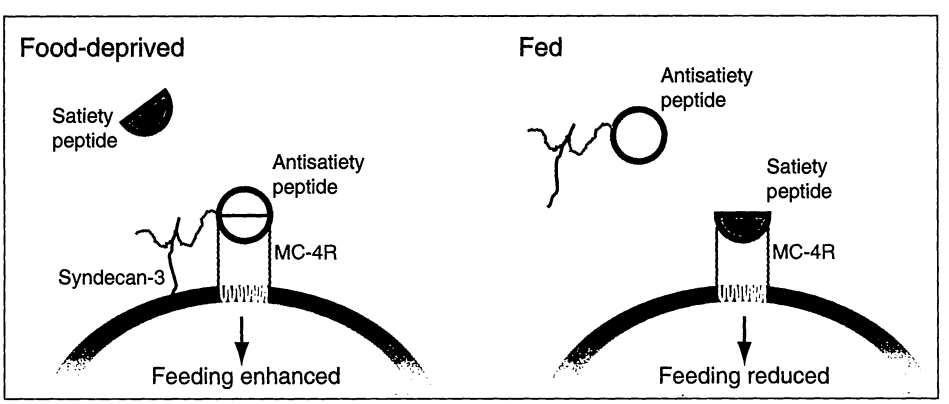
While intriguing, the results had a major limitation: Syndecan-1 normally resides in skin and related tissues, not in the brain. The transgenic mice were an exception, the team found—they produced syndecan-1 in the brain's hypothalamus, the part that regulates feeding behavior. "My initial response was, it's probably not interesting," says Flier, who advised the team. He suspected that the transgene was damaging the hypothalamus somehow rather than playing an active role in appetite control.

To get at the "real physiology," says Reizes, the group went after a relative of syndecan-1 that normally dwells in the brain: syndecan-3. They tested whether syndecan-3 concentrations rise when mice are hungry, as would be expected if the molecule works in concert with an antisatiety peptide. "Sure enough, that's exactly what we saw," says Reizes. And without syndecan-3, engineered mice don't eat even after fasting all night. "It's as if they don't perceive that they're hungry," says Reizes. This result "suggests that syndecan-3 may actually be a normal factor that modulates body weight," says Flier.

The researchers propose that when normal mice eat, they shed syndecan-3 from the surface of cells in the hypothalamus; the syndecan-3, in turn, takes the antisatiety peptide along for the ride, removing it from the MC-4R and liberating the receptor so it can bind the satiety peptide (see figure). To test this theory, they engineered two strains of mice. In one, syndecan-1—whose structure they understood well enough to tweak in the appropriate ways—was stuck to the cell membrane; in the other, it wasn't. Only membrane-bound syndecan-1 caused animals to gain weight. That result "strongly suggests that only the membrane-anchored form can potentiate the activity of the antisatiety peptide," says Carl Blobel, a cell biologist at the Memorial Sloan-Kettering Cancer Center in New York City. Perhaps, he suggests, a future therapy might clip syndecan-3 from the cell surface.

The new finding adds another molecule to the smorgasbord of biochemical factors that might predispose a person to obesity or leanness. "Studies of mechanisms that regulate feeding are at an exciting juncture," says Jeffrey Friedman, a molecular biologist at Rockefeller University in New York City. "A rudimentary wiring diagram can be drawn now that includes a number of molecules known to regulate feeding behavior. Syndecan-3 is a new and important element in this system."

–EVELYN STRAUSS



Shedding molecules, shedding pounds? Syndecan-3 collaborates with an antisatiety peptide.

ILLUSTRATION: MODIFIED FROM REIZES *ET AL.*