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

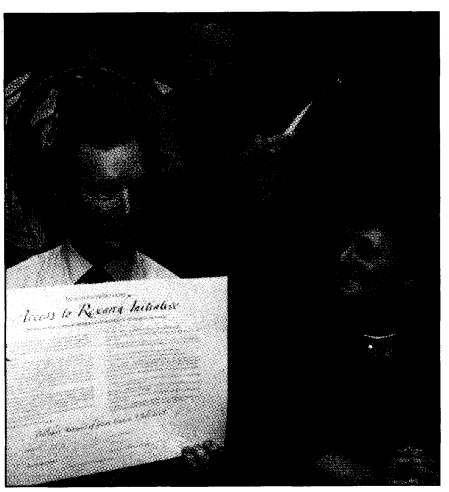
Journals Offered Free To Poorest Nations

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.

VIROLOGY

New Finding Heats Up The Hot Zone

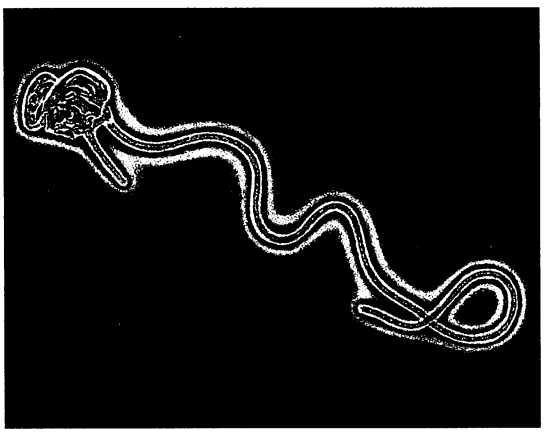
Ebola and its equally gruesome cousin, Marburg, fascinate the public. They've even played starring roles in best-selling books and Hollywood movies. But these terrifying viruses, which typically cause death by hemorrhage within weeks of infection, have attracted relatively few research groups (and scant funding) since they were discovered some 25 years ago. As a result, the modus operandi of these viruses is largely unknown. Now a team led by Mark Goldsmith at the Gladstone Institute of Virology and Immunology in San Francisco, California, has uncovered an intriguing lead: a molecule that helps both viruses infect cells.

As Goldsmith, Stephen Chan, and co-workers describe in the 13 July issue of *Cell*, these viruses have an intimate relationship with folate receptor–α (FR-α), which is found on the surface of many cell types. "It's an important finding," says Paul Bates of the University of Pennsylvania in Philadelphia. "We're now going to get into the nitty-gritty of taking [the discovery] apart and trying to figure out the nature of the interaction," says Bates, whose own lab also studies how Marburg and Ebola establish infections. If FR-α does turn out to be an important accomplice for viral entry, this insight might point the way to novel treatment and vaccine strategies.

Many viruses cause infections by first docking onto receptors on cell surfaces. But the Goldsmith team carefully avoids calling FR-α a "receptor" for Ebola and Marburg. Rather, they describe it as a "cofactor for cellular entry"—which is another way of saying that they don't yet know the precise role that FR-α plays in opening the cellular door to these invaders. "At this point we're happy to have any clues about how [these viruses] enter cells," says Gary Nabel, who heads the Vaccine Research Center at the National Institutes of Health and is part of the small club of investigators who study Ebola.

The Gladstone researchers found FR-α by exploiting an earlier finding—that Ebola and Marburg do not infect T cells. These cells thus provide an ideal system for testing, one by one, possible factors that control viral entry. Specifically, the researchers made a library of DNA from cells that the viruses *can* infect, reasoning that these genes produce the missing factor (or factors). They next engineered new T cells to contain one or more of those genes, then "challenged" the modified cells with a chimeric virus that combined a surface protein from Marburg with HIV. (Researchers routinely use such "pseudotype" viruses of both Marburg and Ebola because few labs have the biosafety capabilities to work with the real pathogens.) They soon found that the pseudotype Marburg virus could infect T cells that contained FR-α. The same held true for a pseudotype Ebola virus.

"We did many, many, many experiments to try and convince ourselves that the results were real," says Goldsmith. For example, they found that blocking FR-α inhibits viral entry. And, working with Alan Schmaljohn at the U.S. Army Medical Research Institute of Infectious Diseases in Fort Detrick, Maryland—which has one of two labs in the country equipped to handle the hottest viruses—the researchers demonstrated that wild-type Marburg virus could infect T cells only if they were modified to contain FR-α.



Breaking and entering. Researchers have found a molecule that helps Ebola (above) and Marburg viruses enter cells.

CREDIT: BARRY DOWSETT/PHOTO RESEARCHERS

Several caveats remain. Nabel cautions that rather than serving as a receptor for Ebola and Marburg, FR-α might send signals that tell the cell to let the viruses enter. Pennsylvania's Bates adds that he'd like to see more studies done with other cell types. "The three most important targets [for these viruses] aren't addressed in this paper: macrophages, hepatocytes, and endothelial cells," says Bates. (All have FR-α, to varying degrees.) And both Goldsmith's group and Bates have unpublished data that Ebola and Marburg can infect cells that do not have FR-α—evidence that the viruses may use different pathways to enter different types of cells.

Goldsmith, too, is cautious about the FR-α findings. "We don't know how things will play out," he says. But for the small cadre of Ebola and Marburg researchers, the new work offers a tantalizing clue about two diseases that, famous though they are, largely remain a mystery. **–JON COHEN**

ScienceScope

New Faces A trio of prominent research institutions is getting new leaders. The European Molecular Biology Laboratory (EMBL) in Heidelberg, Germany, this week appointed two women to lead key outposts. Janet Thornton, a structural biologist at University College London, will become research director at the European Bioinformatics Institute near Cambridge, U.K. Nadia Rosenthal, a biomedical researcher at Harvard University, will take over EMBL's mouse biology program in Monterotondo, Italy. Both programs are emerging reinvigorated from financial crises (*Science*, 18 May, p. 1275, and 15 June, p. 1985).

In the United States, veteran virologist Edmund Tramont has taken the helm of the government's largest AIDS program. Tramont, former head of the U.S. military's AIDS research program and the University of Maryland Biotechnology Center, will lead the National Institute of Allergy and Infectious Diseases' $1 billion Division of AIDS.

Celera, NIH Make a Deal After 7 months of painstaking review, the National Institutes of Health (NIH) has agreed to let some of its scientists use private DNA databases maintained by Celera Genomics of Rockville, Maryland. NIH's largest unit, the National Cancer Institute (NCI), signed a memorandum of understanding with Celera in late June that permits NCI scientists to pay for Celera's data from their own budgets. The decision was controversial because NIH has already used public funds to create its own DNA data archive, GenBank (*Science*, 12 January, p. 223).

NCI's decision does not signal a loss of confidence in the public system, says NIH intramural chief Michael Gottesman. "Some people at NCI were interested" in seeing Celera's data, he said, so NCI managers made a deal that would give them access and serve as a model for other NIH institutes. He doubts many researchers will sign up but hasn't taken a "head count" of potential users.

Celera president J. Craig Venter says NIH was pushed into the decision by its own researchers, especially those who want to check out the company's assembled and annotated mouse genome. (The public mouse genome may not be available for years.) The agreement "will put behind us" old battles, Venter says. He predicts Celera will bill NCI "less than $20,000 per lab" for use of all its databases, including the mouse genome.

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