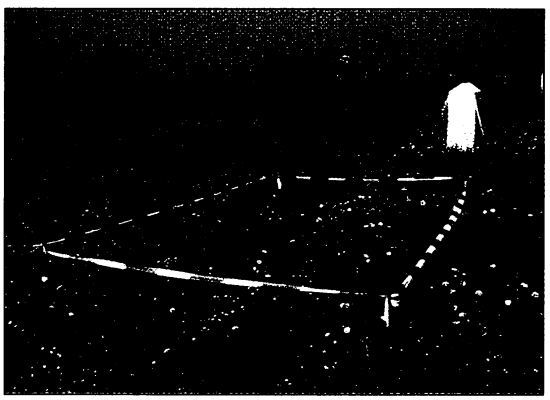
plot, wheat seeds engineered to resist the stinking smut fungus. Smuts and bunts—a related pest—devastated European wheat in the 18th century and continue to plague crops in many developing countries. The diseases are hard to detect and are spread mainly through planting infected seeds.

Sautter modified two Swiss spring wheat lines to express a viral gene, *KP4*, that encodes a protein that inhibits fungal growth. In greenhouse experiments, the transgenic plants proved 30% less susceptible than controls to infection with stinking smut. In 1998, Sautter was ready to take the next step: petition the Swiss Agency for the Environment, Forests, and Landscape (BUWAL) to grow the transgenic plants on a plot "twice the size of a double bed," he says.



Waiting for Godot? Christof Sautter displays his dormant 8-square-meter plot with safety measures, including a tent to prevent pollen from escaping.

But Sautter hesitated, worried about the outcome of a national referendum that would ban transgenic research (*Science*, 12 June 1998, p. 1685). The referendum was defeated, but the climate remained uncertain as parliament launched a debate—which is still going on—about how to legislate gene technology. According to Wilhelm Gruissem, director of ETH's plant biotechnology laboratory, BUWAL representatives requested an "informal" meeting at the Bern train station in December 2000 to discourage him and Sautter from submitting their field trial petition. BUWAL by then had already rejected two applications from other teams and appeared to be tipping its hand to the ETH duo: Gruissem claims they were told that their experiment would be "politically inopportune." BUWAL spokesperson Andreas Stuber confirms that the meeting took place but insists that its purpose was constructive.

Sautter and Gruissem went ahead with their application on 19 January, after which BUWAL requested additional greenhouse tests. They got a boost on 5 September when the biosafety commission ruled that the experiment posed no "appreciable" risk to people or the environment. But at a press conference on 20 November, Philippe Roch, director of BUWAL and former head of the Swiss World Wildlife Fund, announced that the department had rejected the application. Roch argued that it was impossible to assess the experiment's risks because too little is known about the KP4 protein and because the transgenic wheat contains a foreign antibiotic resistance gene. Although this gene is dormant and not known to pose a risk, Swiss legislators are moving to outlaw trials of plants that contain it anyway.

Gruissem rejects Buwal's rationale, arguing that the field trial would have been "the perfect risk-assessment experiment." The proposal included such restrictive safety measures—wire mesh to keep out field mice, for example, and a tent cover to prevent pollen from escaping—that members of the biosafety commission, Wittek recalls, joked whether it could still be called an open field trial.

ETH announced on 29 November that it will appeal the ruling to the Department of Environment, Transport, Energy, and Communications. In the meantime, Sautter's continued funding from the Swiss National Science Foundation stipulates that he must obtain approval by February for field trials of his wheat. Failing that, he says, he could pack up and go to the United States, although he says he would prefer to remain in Switzerland to argue the case for GM field trials.

Beat Keller, a plant biologist at the University of Zürich who coordinates the Swiss National Science Foundation program on wheat, sees the decision as a culmination of nonscientific approaches to the regulation of GM plants. "It is so obviously wrong," he says. And it is not likely to be righted anytime soon: Wittek says there are no other field-trial applications pending or in sight.

–GISELLE WEISS

Giselle Weiss is a writer in Allschwil.

FUNCTIONAL GENOMICS

Pathogen Researchers Get Help From TIGR

Immunologist Pam Baker is getting the backup she needs. As a professor at Bates College, a small undergraduate institution in Lewiston, Maine, Baker doesn't have easy access to the advanced gene research tools that could help her understand how the bacteria *Porphyromonas gingivalis* helps spark gum disease. So she was pleased when The Institute for Genomic Research (TIGR) in Rockville, Maryland, recently offered to provide her with the specialized glass microarrays that can document how *P. gingi-*

ScienceScope

Pluto Power NASA says it doesn't have the money, and the White House insists it won't back the mission, but Congress is getting its way—for now. The space agency last week chose a team led by Alan Stern of the Southwest Research Institute in Boulder, Colorado, and the Applied Research Laboratory (APL) in Laurel, Maryland, to start designing a spacecraft for a 2006 flight to Pluto.

The push to go to the solar system's farthest planet comes from Congress, which allocated $30 million for the flyby in the recently approved 2002 NASA budget. Underlining the importance of politics, APL director Richard Roca praised the work of "avid space science supporters, such as Senator Barbara Mikulski [D–MD]," who leads the Senate subcommittee that oversees NASA's budget. But the Administration continues to insist that there's just not enough money for a launch, presaging another showdown next year.

No New Toys Geophysicists hoping to unveil parts of "the most complete, highest resolution, digital topographic map of Earth" at next week's meeting of the American Geophysical Union will probably have to contain their excitement a few more months. In the aftermath of 11 September, the Defense Department's National Imagery and Mapping Agency (NIMA) has "requested" that NASA not release any of the data returned by the Shuttle Radar Topography Mission (SRTM, right), in which NIMA was a major partner.

After almost 2 years of processing the 10 terabytes of data, researchers had topographic maps of Oregon, California, and the Philippines' Mount Pinatubo volcano ready to show their colleagues how geologic hazards such as landslides, coastal erosion, and volcanic mudflows can be better understood and anticipated. NIMA is "talking to NASA about how to start releasing the data," says SRTM deputy project scientist Tom G. Farr of the Jet Propulsion Laboratory in Pasadena, California. "They're just trying to be cautious, to do the right thing. I don't think it'll be longer than a few months"—just long enough to spoil the holiday fun.

CREDIT: (RIGHT) NASA

for any violations. Some hospitals in Minnesota, which passed a law 5 years ago that imposes strict rules for releasing records, have banned external researchers—those not on hospital staff—from using their databases, Korn says. Having more health care systems put their databases off-limits, the AAMC letter warns, could "paralyze vital public health research." –JOCELYN KAISER

OBESITY RESEARCH

Pot-Bellied Mice Point To Obesity Enzyme

Words linking fruit and the human anatomy have long sweetened sonnets and love letters. But lately the term "apple-shaped" has gained renown on the pages of medical texts. People who carry excess fat around their waists—the so-called apple-shaped body type—are more prone to obesity-related maladies than their equally overweight but pear-shaped counterparts, who pack weight around their hips. Physicians have observed the connection for decades, but no one could explain it, let alone search for a therapy to right the scales.

Now on page 2166, researchers at Beth Israel Deaconess Medical Center in Boston suggest a reason for the disease–body type relationship, and a possible new target for treatment. The culprit is an obscure enzyme that works to recycle a steroid stress hormone called cortisol. Through delicate genetic engineering, endocrinologist Jeffrey Flier and his colleagues overexpressed the gene for this enzyme solely in the fat of mice. These rodents look and act a lot like overweight apple-shaped people: They eat more than normal mice and gain fat disproportionately around their middles. As adulthood sets in, the animals develop the early biochemical symptoms of heart disease and diabetes. Blocking the enzyme in people, the researchers suggest, might thwart obesity-related illnesses.

"This was really the first proof that manipulating steroid conversion in fat alone is enough to lead to all these abnormalities," says endocrinologist Stephen O'Rahilly of Addenbrooke's Hospital in Cambridge, U.K., who studies the genetics of obesity and diabetes. "I wish I'd done the experiment myself."

Inspiration for the study came indirectly from a rare illness called Cushing syndrome. Its sufferers have too much cortisol coursing through their bloodstreams and become diabetic and severely obese. For decades, endocrinologists hypothesized that common forms of obesity may represent very mild cases of Cushing syndrome. If so, most obese people should have higher than normal blood levels of cortisol—but researchers found that they don't and discounted the hypothesis.

The theory was resurrected by Paul Stewart of the University of Birmingham in Edgbaston, U.K., whose group found that people have pockets of high cortisol activity. The team compared stress hormone production in two types of fat in 16 patients undergoing surgery, most of whom were of normal weight. One sample came from underneath the skin, the other from adipose tissue in the abdomen. In the belly fat, the researchers found higher activity of an enzyme called 11β hydroxysteroid dehydrogenase type 1 (11β HSD-1), which regenerates active cortisol from its inactive form, cortisone.

Flier read a 1997 paper in *The Lancet* on the research and thought, "If we could make a mouse that overexpresses the enzyme only in fat, we could ask the question, 'Will that mouse get the apple-shaped body type and all its ill effects?' " he recalls. Visiting scientist Hiroaki Masuzaki engineered the mice; he linked a rat 11β HSD-1 gene to a promoter that turns on only in fat. The mice had 2.4 times more enzyme activity in their belly fat than did normal mice. Stress hormone levels in stomach fat tissue rose by 15% to 30%, but, as in most obese humans, bloodstream levels of the hormone were normal. As adulthood set in, the transgenic mice ate more, got fatter than normal mice, and carried the fat in their abdomens. Even when fed low-fat diets, the transgenics carried a spare tire that accounted for 37.9% of their total body fat compared with 27.5% in normal mice. The mice showed the hallmarks of early diabetes and hypertension: insulin resistance, renegade blood glucose levels, and other biochemical abnormalities. And a high-fat diet accelerated the pot-bellied rodents' downward spiral.

"It is really the whole picture of what we refer to as the metabolic syndrome," says Flier, citing a term now in vogue in en-

Belt loosener. Activating an enzyme in fat gives mice a syndrome seen in apple-shaped people.

CREDIT: PHILIP HARVEY/CORBIS

ScienceScope

War's First Casualty The British government wants to stop publicizing the locations of U.K. labs working with genetically modified (GM) organisms. In October, the government's Health and Safety Executive (HSE) temporarily stopped releasing a list that pinpointed government, university, and commercial labs doing GM research on grounds that terrorists might use the list to locate ready sources of virulent superbugs. And last month, the HSE proposed to permanently strike labs working with potential bioweapons from the public list. If parliament agrees, the agency would release a sanitized version in January.

Observers disagree on whether the censorship is a good idea. "Any other position would be irresponsible," says Tom Loeffler of the Biotechnology and Biological Sciences Research Council, a grant-giving body. But because "GM organisms currently pose little more threat than existing ones," delisting the labs does little to improve security, says Alastair Hay, a bioweapons expert at the University of Leeds. Clever readers, he adds, can discover out what labs are doing by trolling through journals.

Wayward Brains? Scientists at the U.K.'s Institute for Animal Health (IAH) have come out swinging against two government-sponsored audits that conclude that they mixed up cattle and sheep brains in a high-profile study. The IAH had carried out a 4-year investigation into whether Britain's sheep flock was infected with "mad cow disease." But last October, an independent laboratory reported that sheep brain samples used in experiments actually came from cows, calling the study's results into question (*Science*, 26 October, p. 771).

The audits, made public last week, blame the fiasco on IAH's poor sample labeling and record keeping. But they produced "no clear evidence" for mistakes at IAH, argues institute chief Chris Bostock. The samples originally came from another government facility, he notes, meaning a mix-up or contamination could have occurred either before the IAH took custody or after it sent out the tissues for independent analysis.

IAH researchers complain that auditors spent just 1 or 2 days visiting their lab in Edinburgh, where much of the work was done, and did not interview the scientists who first worked with the samples. Says one IAH staffer: "Everybody is furious at the way this has been handled."

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