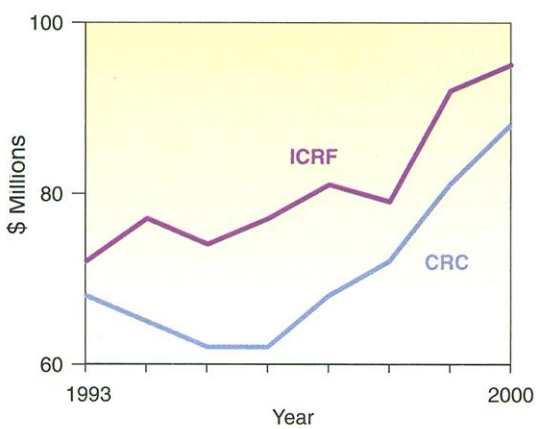
are not to be condoned but are hardly a major offense. "I don't think it's such a big deal," says Richard Reading, director of conservation biology at the Denver Zoological Foundation and co-chair of Colorado's Lynx and Wolverine Advisory Team. "They only needed to inform their superiors."

Yet that misstep may have cost the broader effort its credibility. As Forest Service Chief Dale Bosworth conceded in a statement, the scientists' actions "have called into question the scientific integrity of the interagency survey." **–ERIK STOKSTAD**

CANCER RESEARCH

Will Bigger Mean Better For U.K. Charity?

HERTFORDSHIRE, U.K.—After a long and sometimes tense courtship, the United Kingdom's two major cancer charities are ready to unite next month to form a giant funding agency similar to the U.S. National Cancer Institute (NCI). Cancer Research UK, which will be the world's biggest nongovernmental cancer research organization and the United Kingdom's largest fund-raiser, is expected to spark new collaborations at the often-frustrating nexus of basic and clinical research: turning promising test tube findings into experimental therapies.



Substantial dowries. Research funds have grown steadily for the U.K.'s two largest cancer charities.

British cancer researchers are hoping that the recipe for happiness within Cancer Research UK, as in many successful marriages, will be the complementary strengths of the partners. The Imperial Cancer Research Fund (ICRF) is a basic research powerhouse that mostly supports in-house labs, whereas the Cancer Research Campaign (CRC) focuses on prevention, treatment, and diagnostic research through extramural grants and at a handful of clinical units it underwrites.

Andrew Miller, interim chief executive for Cancer Research UK, says it didn't make sense for the two giants to compete for donations rather than collaborating. The charities had raised the bulk of their funds by vying for legacies and other private donations as well as corporate sponsorships. Both also run national networks of shops that sell goods such as secondhand clothing, bric-a-brac, and books. ICRF's 450 stores, staffed by an army of retirees, raise about $9 million a year—often in direct competition with CRC's 270 secondhand shops. "If someone came from outer space and examined this," says Miller, referring to the competition between the charities, "they would think it was a very daft situation."

An alien visitor next month may not find a land of milk and honey: Cancer Research UK's $189 million budget in 2002, although a third larger than the government's total spending this year on cancer research, is more than an order of magnitude smaller than NCI's budget. Still, an outsider would detect considerable enthusiasm for the new entity. The merger "is a very positive step," says Nick Lemoine of ICRF's molecular oncology unit at Imperial College in London. In the months since the merger plans were announced (*Science*, 26 January 2001, p. 575), Lemoine has had ample time to contemplate working more closely with CRC colleagues on gene therapy and other projects. And as a bittersweet bonus for their efforts, the 3000 scientists at Cancer Research UK can anticipate an extra $20 million or so after the elimination of 130 managerial and support jobs. Miller says the liberated funding will allow the organization to hire more researchers and boost grants in 2003.

One lingering concern in the current CRC-supported labs is that ICRF's core strengths will guide the research agenda—especially because ICRF director-general Paul Nurse, a 2001 Nobel laureate in physiology or medicine, will be Cancer Research UK's scientific chief. Nurse could not be reached for comment. Miller, however, has pledged that most research areas will be retained and that funding committees will consist of CRC and ICRF researchers in equal measure. In addition, the combined charity will remain part of a nascent coordinating body, the U.K. National Cancer Research Institute. Pressure at Cancer Research UK will come from having to do more, not less: Scientists at both ends of the research spectrum will be encouraged to team up on "translational" projects, in which the fruits of fundamental research are used to create experimental therapies.

Observers expect that Cancer Research UK will have an easier time wooing donors than the two charities had as swinging singles. "There were concerns early on that one

CREDITS: (TOP TO BOTTOM) CRC/ICRF; DIANE CLARIDGE/AP

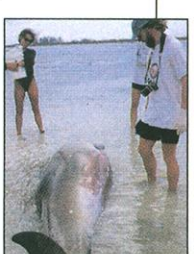
ScienceScope

Research Injection Work on infectious diseases got a boost last month with the opening of a new vaccine center at the University of Texas Medical Branch (UTMB) in Galveston. Scientists there plan to develop vaccines for a range of pathogens—from bioterror threats to sexually transmitted diseases—and ponder policy issues, such as the growing public resistance to vaccination.

The center was kick-started by a $3.75 million grant from the John Sealy Memorial Endowment, a charity that gives exclusively to UTMB. It will be directed by herpes vaccine researcher Lawrence Stanberry, who says he has lured Martin Myers, director of the U.S. National Vaccine Program Office, to be the resident policy wonk.

The new center will allow UTMB—already noted for infectious disease research (*Science*, 28 April 2000, p. 598)—"to make some very important contributions" to vaccine development, predicts John La Montagne, deputy director of the National Institute of Allergy and Infectious Diseases in Bethesda, Maryland.

Victims of Sound The U.S. Navy has concluded that a sonar training exercise caused a mass whale stranding in the Bahamas in March 2000 that killed several rare beaked whales (*Science*, 26 January 2001, p. 576). In a report released 20 December 2001, the Navy and the National Marine Fisheries Service conclude that the strandings were caused by an "unusual combination" of factors, including sea-bottom contours and water conditions that may have channeled and magnified sonar pings. The researchers could not pinpoint exactly how the sound energy injured the whales, but the acoustic assault appears to have left some dazed and confused, causing them to swim ashore. The Navy says that it will try to avoid using sonar in similar situations during training runs. But Naomi Rose, a marine mammal expert with the Humane Society of the United States in Gaithersburg, Maryland, says the report is "carefully worded" so that it does not give ammunition to critics of SURTASS LFA, a new, lower frequency sonar system the Navy plans to deploy.



Contributors: Jeffrey Mervis and Elizabeth Pennisi, David Malakoff, Martin Enserink

plus one would not equal two, but market research seems to suggest that merging could in fact raise our profile," says Neil McDonald, a structural biologist at ICRF's headquarters in London. Indeed, predicts Steve Jackson, deputy director of the Wellcome-CRC Institute of Cancer and Developmental Biology at the University of Cambridge, Cancer Research UK should have more clout among both politicians and scientists. Coupled with a solid research program, that would amount to far more than a marriage of convenience.

–JOHN PICKRELL

John Pickrell is a science writer in Hertfordshire, U.K.

ASTHMA RESEARCH

Missing Gene Takes Mice's Breath Away

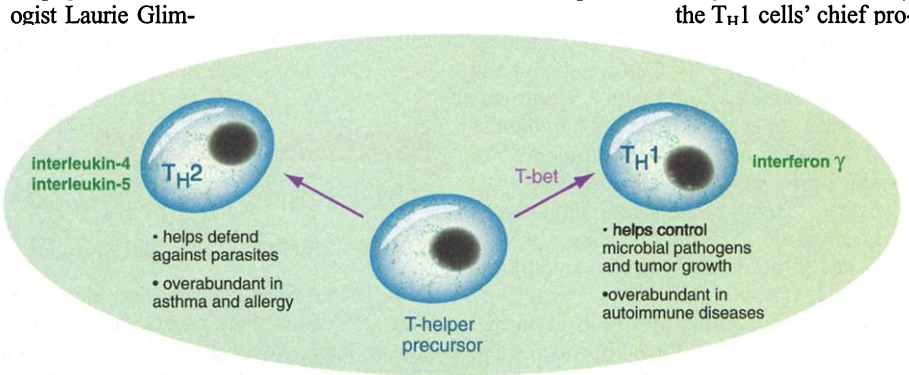
A strain of mice with a tendency to wheeze may help scientists get closer to the root of asthma in humans. Asthma constricts airways in patients' lungs and leaves them short of breath, sometimes fatally; it afflicts tens of millions of people worldwide and seems to be on the rise in many areas. Scientists are still struggling to decipher the cellular signals at the root of the attacks. Now, in reports on pages 336 and 338, immunologist Laurie Glimcher and her colleagues describe a mouse strain that mimics the human condition and might provide a better model system for studying the disease.

Researchers have created asthmatic mice before, but through a process involving injections of allergens and irritants. That scenario doesn't match the situation of many patients with chronic asthma, whose attacks are not triggered by known allergens. The new mice resemble those human asthmatics in several key ways, such as having characteristic chronic lung inflammation and thickened airway walls. What's more, the gene responsible for the mice's affliction appears to be misregulated in human asthmatics as well. "It's an exciting model," says immunologist William Paul of the National Institute for Allergy and Infectious Diseases in Bethesda, Maryland.

The engineered mice lack a gene called T-bet, which codes for a transcription factor, a protein that controls the expression of other genes. In previous experiments, Glimcher and her colleagues had shown that T-bet affects the development of immune system cells, encouraging the development of so-called T_H1 cells. These cells help organize attacks on unfamiliar cells such as invading microbes. They also make proteins that discourage overproduction of a sister cell type called T_H2 cells, whose main job is to help the body defend against parasites.

In most people, a complex set of feedback loops keeps the two cell types in balance. But many scientists suspect that in asthma patients something skews the balance, allowing T_H2 cells to predominate. The proteins those cells produce can lead to some of the changes seen in asthma patients' airways, including high numbers of trigger-happy immune cells that spark inflammation.

To find out more about T-bet's role in the immune system, the team created mice lacking the gene. As expected, the mice produced fewer T_H1 cells. Immature immune cells taken from the animals' lymph nodes produced very little interferon γ, the T_H1 cells' chief protein product, compared to their wild-type littermates. The cells also produced higher levels of interleukin-4 and interleukin-5, two products of T_H2 cells—and prime suspects in fueling asthma.



Proper balance. The T-bet gene prompts immature immune cells to become T_H1-type cells, keeping the number of T_H2 cells in check.

The animals' lungs resembled those of chronic asthma patients, with unusually thick layers of collagen and extensive networks of the musclelike cells that constrict airways. And even before exposure to an irritant, the animals' lungs showed signs of inflammation: They had significantly more immune system cells called eosinophils and lymphocytes than their littermates with functioning T-bet. Mice lacking T-bet were also extremely sensitive to the irritant methacholine; their airways narrowed and it took more effort to breathe. Although it is difficult to really hear a mouse wheeze, Glimcher says, "these mice have asthma."

T-bet might play a role in human asthma as well. The researchers found that asthma patients had significantly lower levels of T-bet expression in their lungs than people without asthma. Although the genetic causes of asthma are complex, the T-bet gene is in a region of the genome that has been implicated in asthma susceptibility.

The mice will be especially useful for fingering the proteins that interact with T-bet to encourage the development of T_H1 cells, says asthma specialist Jack Elias of Yale University School of Medicine. Such proteins might help scientists track down the still-mysterious cause of asthma. Although any treatments are years away, Glimcher says there may be ways to tweak the T-bet system in human lungs to discourage asthma attacks. Any such hints should help asthma patients breathe a little easier.

–GRETCHEN VOGEL

STEM CELL RESEARCH

Stem Cells May Shore Up Transplanted Hearts

Can a broken heart be mended? Perhaps, says a new report, which shows that after a heart transplant, cells migrate to the donated organ, possibly helping it recover. These migrants show signs of being stem cells, those multitalented cells that have the capacity to develop into a multitude of tissues.

Some parts of the body, such as the skin, regenerate readily when damaged. But "we all thought that once you lose a chunk of heart, it's gone," says cardiologist Roberto Bolli of the University of Louisville in Kentucky. One of the first indications that the heart can bounce back came in July 2001, when researchers reported that heart muscle cells can divide after a heart attack. Transplanted hearts are often similarly damaged: Many heart cells die during the hours the organ is out of the body.

Cardiovascular researchers Federico Quaini and Piero Anversa of New York Medical College in Valhalla and colleagues at the University of Udine, Italy, wanted to find out whether the transplant recipient's body pitches in to help heal the new organ. The team examined eight hearts transplanted from female donors into male patients. Up to 10% of cells in the transplanted hearts contained the male Y chromosome—a clear sign that cells from the recipient had taken up residence in the new heart, the group reports in the 3 January issue of *The New England Journal of Medicine*.

Cardiologist Philip Binkley of Ohio State University, Columbus, calls the study an "ingenious and novel demonstration that the heart can recruit new cells that may be a