swims. For mice, that qualifies as a permanent change in how they respond to alcohol.

Exactly how loss of the CRH receptor alters the animals' drinking habits is not clear. The mutants don't appear to be any more shaken up by the stressful situations than are the normal mice. And because they don't start drinking more right away, they're not relying on alcohol to restore their courage. Jane Stewart of Concordia University in Montreal, Canada, who studies the involvement of CRH receptors in addictive behavior, explains that "the stress may activate pathways that have nothing directly to do with fear and anxiety but which alter the approach to alcohol itself."

The researchers are now doing association studies in humans in hopes of finding out more about such pathways. Specifically, Sillaber and Spanagel will look for variations in stress-related genes in alcoholics. Some alcoholics, they say, may have defects in their CRH receptors or other anomalies that disrupt the stress response system in a way similar to that seen in the mutant mice. This research, they say, may help pinpoint some of the genes that make an individual more likely to respond to the slings and arrows of outrageous fortune by turning to the bottle.

–CONSTANCE HOLDEN

POPULATION STUDIES

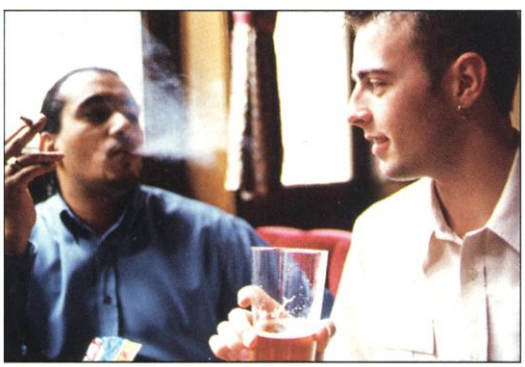
U.K.'s Mass Appeal for Disease Insights

LONDON—Plans shifted into high gear this week for a huge repository of information on the genetics and lifestyle of the population of the United Kingdom. The $66 million BioBank UK hopes to collect data from half a million middle-aged Britons over the next decade. But a public battle is looming over how much access companies should have to the database.

The project, first proposed more than 2 years ago, aims to use the trove of data on the British population's genetic makeup and way of life to flush out factors that influence common diseases such as cancer, diabetes, and heart disease (*Science*, 18 February 2000, p. 1184). On 29 April, the Medical Research Council, the Department of Health, and the Wellcome Trust, a mammoth biomedical charity, announced their financial backing for BioBank, which will collect blood samples and information on diet, smoking, and other lifestyle choices from 500,000 volunteers aged 45 through 69, then track their health for at least 10 years. Researchers will mine the database for disease-related patterns, such as genes that heighten vulnerability to the cancer-causing effects of smoking.

The study is a logical follow-up to the Human Genome Project, says Wellcome Trust director Michael Dexter. "It is part of an overall strategy to really ensure that the [sequencing] research we've done does have health benefits," he says. The human genome sequence, he says, will allow researchers to more quickly identify DNA variations in the U.K. population that correlate with disease. BioBank will stand out from a growing pack of genetic databases—including deCODE, which probes for disease genes in Iceland (*Science*, 1 January 1999, p. 13)—because it will collect detailed data on lifestyle choices and risk factors across several ethnic groups. The search for an executive director and a headquarters site will begin in the next few months.

An oversight committee, to be established by BioBank's funders, will hammer out the rules for access to the data. These are expected to come under intense scrutiny. "A lot more work needs to be done on the relationship between BioBank and industry" to ensure that benefits flow back to the public, asserts David S. King, coordinator of Human Genetics Alert in London. The watchdog group is lobbying for a ban on patents based on genetic discoveries that come out of the database. The group is also pressing for BioBank to allow volunteers to opt out of research they may object to, such as studies on behavioral genetics. Dexter argues that industry researchers must be given access for the project to succeed. "At the end of the day," he says, "they're the ones who develop the drugs."



Pay later? BioBank UK will probe the links between genes, lifestyle, and disease.

BioBank has time to address such issues: Full-scale enrollment of volunteers is not likely to get under way until 2004, says a Wellcome Trust spokesperson. The real test will come then, when doctors start pitching the project in earnest to their patients. "It is an opportunity to get people on board for this kind of new biology," says Dexter.

–GRETCHEN VOGEL

GENOME RESEARCH

Venter Is Back With Two New Institutes

After 3 months of rare silence, genome scientist J. Craig Venter is back on the air. Venter, who abruptly resigned in January as president of Celera Genomics of Rockville, Maryland (*Science*, 25 January, p. 601), announced 30 April that he plans to establish two new institutes that will focus on ethics, clean energy, and the environment. Venter also made headlines last week by confirming a persistent rumor about Celera's research: "Three-fifths" of the human genome the company sequenced and published in 2001 is his own.

Venter says he is establishing an outfit called the J. Craig Venter Science Foundation. It will be the financial and legal umbrella for three nonprofit organizations whose boards he will chair, all located in Rockville. One is already well established: The Institute for Genomic Research (TIGR), a sequencing and gene analysis operation presided over by Venter's wife, microbiologist Claire Fraser. TIGR's two new siblings will be a think tank called the TIGR Center for the Advancement of Genomics (TCAG) and a research institute called the Institute for Biological Energy Alternatives (IBEA). All three will share TIGR's current endowment, which is estimated to be worth about $140 million, according to Venter. The fund was established with stocks Venter received from Celera and from an earlier partnership with Human Genome Sciences of Rockville.

The broadest of the new operations, TCAG, will enter a field already well populated with serious thinkers. TCAG will concern itself with "public policy and ethical issues related to the sequencing of the human genome," says Venter. Initially it will take up four topics: risks of discrimination and a mistaken public emphasis on "genetic determinism"; fallacies about race; genetics and medicine; and stem cell biology. Venter says, for example, that congressional efforts to "criminalize" scientific research by banning some cloning and embryonic stem cell studies are "unprecedented" and deserve much wider comment. He plans to recruit a staff of 20 to 30 people to support up to 30 visiting faculty, who will come for periods of 3 to 12 months.

TCAG's turf overlaps to some degree with that of another new center announced in April, the Genetics and Public Policy Center of Washington, D.C., backed by the Pew Charitable Trusts and Johns Hopkins University in Baltimore. The Hopkins center, headed by former National Human

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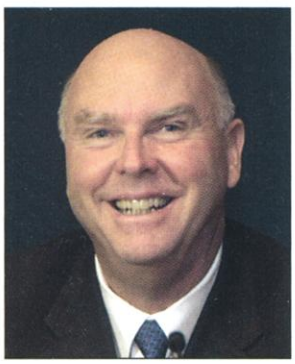
Genome Research Institute assistant director Kathy Hudson, will focus initially on reproductive genetics, as required by its 3-year, $9.9 million grant from Pew. Venter's comment: "The more voices, the better."

Bioethicist Thomas Murray, director of the Hastings Center in Garrison, New York, says the Hopkins center was carefully planned and "fills an important need." Murray hasn't seen TCAG's agenda, but he offers Venter this advice: "Define your mission clearly" and guarantee the center its independence.



Ethics and energy. Venter is moving into new research areas.

Unlike TCAG, Venter's energy and environment shop, IBEA, may rely extensively on government support. Staff scientists will explore microbial genomics to look for solutions to environmental problems, for example, by degrading toxic chemicals and sequestering carbon dioxide from the atmosphere. They will also study clean energy products, such as hydrogen. This project, according to Venter, received encouragement from Ari Patrinos, head of biological and environmental research in the Department of Energy's (DOE's) science office. Indeed, Patrinos says, IBEA's agenda matches DOE's own research goals very closely: "If [Venter's] record is any indication, we expect big things from him again."

–ELIOT MARSHALL

CLIMATE CHANGE

A Single Climate Mover for Antarctica

Weird things are afoot at the bottom of the globe. The Antarctic Peninsula's Larsen ice shelf has suffered a torrid 2.5°C warming during the past half-century (*Science*, 29 March, p. 2359). A Rhode Island–sized chunk of the ice shelf drifted away from the peninsula and broke up in recent months as glaciologists watched, some Antarctic glaciers are thinning, and sea ice is retreating—all as greenhouse warming would have it. Meanwhile, however, other glaciers are thickening. In places, sea ice is actually advancing, and most of Antarctica is not warming at all or is even cooling. What gives?

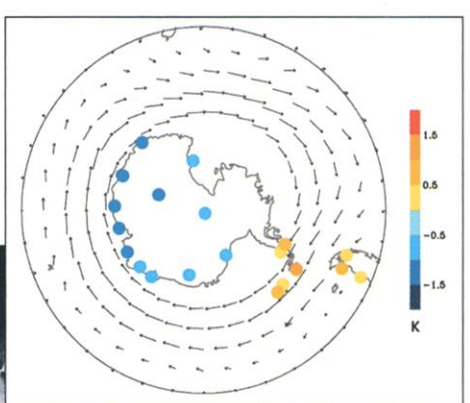
Meteorologist David Thompson of Colorado State University in Fort Collins and atmospheric chemist Susan Solomon of the National Oceanic and Atmospheric Administration's (NOAA's) Aeronomy Laboratory in Boulder, Colorado, have an explanation. On page 895, they build a case that a climate master switch in the atmosphere over the high southern latitudes is driving the wacky climate shifts of Antarctica. And the hand on the switch, they suggest, may be our own. Humanmade chemicals drive the formation of the yearly Antarctic ozone hole, which, they argue, throws the climate switch—called the Antarctic Oscillation (AAO)—in the atmosphere below.

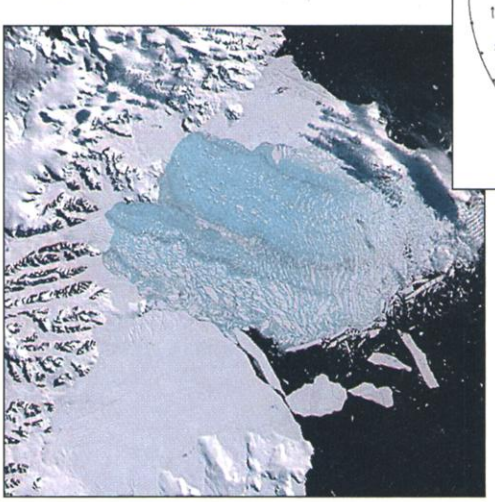
The work is "the strongest evidence yet" that a shift in the AAO "could explain a number of different components of [Antarctic] climate trends," says meteorologist David Karoly of Monash University in Clayton, Australia. The idea that Antarctic ozone loss is behind the AAO shift is getting a more cautious reception.

To link stratospheric ozone loss to climate change at the surface, Thompson and Solomon first turned to atmospheric observations from weather balloons routinely launched from seven sites around Antarctica. The instrumented balloons tracked the erratic atmospheric seesaw of the AAO, which raises atmospheric pressure alternately over the pole and in a ring passing over the Southern Ocean and the tip of South America. These pressure shifts alternately accelerate and slow the ring of westerly winds that encircle Antarctica, as Thompson and J. Michael Wallace of the University of Washington, Seattle, suggest happens in the Arctic (*Science*, 9 April 1999, p. 241). The AAO clearly swings erratically from one phase to the other week to week, month to month, and year to year, but the balloon data from 1969 to 1998 show that recently it has been spending more and more time in its positive, strong-wind phase, just as the Arctic Oscillation (AO) has.

Having shown that the AAO high above the polar region has shifted, Thompson and Solomon demonstrated that the shift could explain most of the climate change at the surface. Comparing the pattern and amplitude of the AAO trend with those of the climate change, they found that the AAO's shifts in circulation—including winds and air rising over the continent—could account for 90% of the summertime cooling over Antarctica and about half of the summertime warming over the Antarctic Peninsula and the southern tip of South America. The rest of the peninsula's warming may be linked to changes as far away as the tropical Pacific.

To trace the changes back to the stratosphere, Thompson and Solomon compared trends in stratospheric "climate" with the AAO trend. Researchers had already established that the springtime loss of ozone—which normally absorbs solar energy and warms the lower stratosphere—had cooled the lower stratosphere by 6°C each spring. That cooling, in turn, strengthens the stratospheric vortex of westerly winds, a stratospheric analog of the AAO's ring of westerlies in the lower atmosphere. Thompson and Solomon compared the timing of ozone-induced cooling and vortex intensification in the stratosphere with similar changes in the lower atmosphere and at the surface. The stratospheric shifts seemed to break through to the lower atmosphere at roughly the times of the year—late spring and early summer, and fall—when seasonal circulation changes temporarily break down the usual barrier between the wispy stratosphere and the dense lower atmosphere. That timing "seems pretty good evidence [that] ozone is important" in driving the AAO and thus climate change, says Thompson, "particularly during the late spring."





Hot times. Warming (yellow) and winds (arrows) induced by the Antarctic Oscillation doomed part of the Larsen ice shelf.

Pinning most of the contradictory Antarctic climate changes on a changing AAO "seems reasonable" to meteorologist Martin Hoerling of NOAA's Climate Diagnostics Center in Boulder. He and others are reluctant, how-

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