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Last chromosome in human genome sequenced

Wed May 17, 2006 1:03 PM ET

By Patricia Reaney

LONDON (Reuters) - Scientists have reached a landmark point in one of the world's most important scientific projects by sequencing the last chromosome in the Human Genome, the so-called "book of life".

Chromosome 1 contains nearly twice as many genes as the average chromosome and makes up eight percent of the human genetic code.

It is packed with 3,141 genes and linked to 350 illnesses including cancer, Alzheimer's and Parkinson's disease.

"This achievement effectively closes the book on an important volume of the Human Genome Project," said Dr Simon Gregory who headed the sequencing project at the Sanger Institute in England.

The project was started in 1990 to identify the genes and DNA sequences that provide a blueprint for human beings.

Chromosome 1 is the biggest and contains, per chromosome, the greatest number of genes.

"Therefore it is the region of the genome to which the greatest number of diseases have been localized," added Gregory, from Duke University in the United States.

The sequence of chromosome 1, which is published online by the journal Nature, took a team of 150 British and American scientists 10 years to complete.

Researchers around the world will be able to mine the data to improve diagnostics and treatments for cancers, autism, mental disorders and other illnesses.

FINAL CHAPTER

Chromosomes, which are found in the nucleus of a cell, are thread-like structures that contain genes which determine the characteristics of an individual.

The human genome has an estimated 20,000 to 25,000 genes. The sequencing of chromosome 1 has led to the identification of more than 1,000 new genes.

"We are moving into the next phase which will be working out what the genes do and how they interact," Gregory told Reuters.

The genetic map of chromosome 1 has already been used to identify a gene for a common form of cleft lip and palate. It will also improve understanding of what processes lead to genetic diversity in populations, according to Gregory.

Each chromosome is made up of a molecule of DNA in the shape of a double helix which is composed of four

chemical bases represented by the letters A (adenine), T (thymine), G (guanine) and C (cytosine). The arrangement, or sequence, of the letters determines the cell's genetic code.

The scientists also identified 4,500 new SNPs -- single nucleotide polymorphisms -- which are the variations in human DNA that make people unique.

SNPs contain clues about why some people are susceptible to diseases like cancer or malaria, the best way to diagnose and treat them and how they will respond to drugs.

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washingtonpost.com

Genetic study reveals surprises in human evolution

Reuters
Wednesday, May 17, 2006; 4:11 PM

LONDON (Reuters) - Humans' evolutionary split from their closest relatives, chimpanzees, may have been more complicated, taken longer and probably occurred more recently than previously thought, scientists said on Wednesday.

After comparing the genomes, or genetic codes, of the two species they suggest the initial split took place no more than 6.3 million years ago and probably less than 5.4 million years ago.

The process of separation may have taken about 4 million years and there could have been some inter-breeding before the final break.

"The study gave unexpected results about how we separated from our closest relatives, the chimpanzees," said David Reich of the Broad Institute and Harvard Medical School's Department of Genetics in Massachusetts.

Instead of analyzing genetic differences between humans and chimpanzees, Reich and researchers from the Massachusetts Institute of Technology (MIT) and the Broad Institute of Harvard and MIT looked at variations in the degree of divergence between the two in different regions of the genomes.

The analysis, published in the journal Nature, shows some regions in the human genome are older than others which means they trace back to different times in the common ancestral population of the two species.

The youngest regions are unexpectedly recent, according to the researchers, which means the separation between the two species was more recent than previously thought.

"A hybridization event between human and chimpanzee ancestors could help explain both the wide range of divergence times seen across our genomes, as well as the relatively similar X chromosomes," Reich explained.

Hybridization refers to the initial separation of two species followed by interbreeding and then the final split.

The findings also raise questions about the 7 million year old fossil of a skull called "Toumai" which was thought to be the earliest member of the human family.

The skull, which has a mixture of primitive and human-like features and dates, was hailed as probably the most important fossil discovery in living memory because it was thought to belong to an ancient ancestor of modern humans.

Some scientists had argued it was a fossil of a female ape.

"It is possible that the Toumai fossil is more recent than previously thought," said Nick Patterson, of the Broad Institute and a co-author of the study.

"But if the dating is correct, the Toumai fossil would precede the human-chimp split. The fact that it has human-like features suggest that human-chimp speciation (separation) may have occurred over a long period with episodes of hybridization between the emerging species," he added.

GENOMICS

The HapMap Gold Rush: Researchers Mine a Rich Deposit

Scientists are parsing a raft of new data on genetic variation for clues to disease and evolution

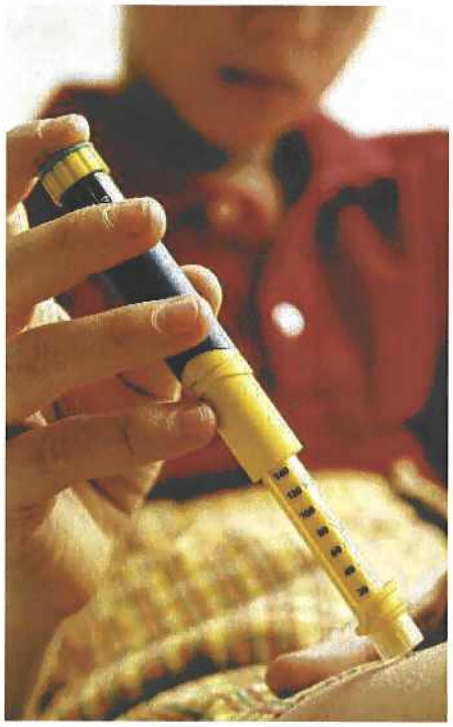
CAMBRIDGE, MASSACHUSETTS—For a conference on the next generation in genomics, the setting was just right: a pristine auditorium in a gleaming new building near the Massachusetts Institute of Technology (MIT). More than 200 people gathered here at the Broad Institute earlier this month to discuss the HapMap, a database cataloging human genetic variation. Begun in 2002, the map has been assembled primarily to boost the analysis of inheritance using pieces of DNA that are often transmitted as intact blocks.

Nearly complete, the HapMap is now being tested for a number of uses: to find genetic variants behind common diseases, to examine the genome's architecture, and to study natural selection. The human HapMap has even inspired the launch of a parallel effort for *Plasmodium falciparum*, the deadly malaria parasite.

Five countries kicked in about $138 million to fund the human project, properly known as the International HapMap Project. One early challenge was to allow for the fact that haplotypes differ somewhat across populations. To include a sweep of variants, the HapMap gathered DNA from 270 individuals of African, Japanese, Chinese, and European ancestry.

The final version, which will be completed this fall, is slated to include more than 4.5 million single-nucleotide polymorphisms (SNPs). Although the SNPs themselves aren't necessarily contributors to disease, they may travel alongside other SNPs that are. Most of the map is already freely available online (www.hapmap.org). And it is being used "a lot," says Francis Collins, director of the U.S. National Human Genome Research Institute (NHGRI) in Bethesda, Maryland, who is one of the map's biggest proponents. Collins says various National Institutes of Health institutes are being "flooded" with funding applications that involve using HapMap data.

Despite such enthusiasm, some researchers say they're not certain just how the HapMap will aid their own genetic studies. The map's central goal is to help identify genes behind common diseases such as cancer, but it's not always clear how to apply it. When it comes to evolution studies, for example, the map may be biased because it prefers common SNPs to rare ones. "The HapMap project was not about studying population history," says NHGRI's James Mullikin. But it's being used often by researchers in that area.



CREDIT: GETTY IMAGES

Deciphering disease. Aided by the HapMap, researchers are finding gene variants that may help explain diabetes and other conditions.

As for disease genes, "it's still a bit early" to expect new findings, says Aravinda Chakravarti of Johns Hopkins University in Baltimore, Maryland, one of the project's leaders. HapMap-related studies are just ramping up, however, and a few are hitting on new results. At the meeting, for example, a postdoc at the Broad Institute, Robert Graham, reported a gene variant linked to lupus that he's found while working in the Broad lab of David Altshuler, one of the HapMap's leaders.

John Todd of Cambridge University in the U.K. and postdoc Jason Cooper described a variant associated with type 1 diabetes, discovered by scanning more than 6500 SNPs in samples from thousands of type 1 diabetes patients and controls. With help from HapMap data, Todd's group homed in on a SNP on chromosome 2 that they believe may help drive diabetes, although they couldn't rule out effects from other SNPs nearby. The work appeared 14 May in the online edition of *Nature Genetics*.

Another test for the HapMap will come this fall when David Hafler, a multiple sclerosis (MS) researcher at Harvard Medical School in Boston and the Broad Institute, and colleagues worldwide plan to complete the initial phase of the first HapMap-guided whole genome scan for a human disease, MS. The outcome "will provide a map for what to study" in MS basic research, Hafler predicts.

The massive HapMap database is inspiring large collaborations as well as projects that take a big-picture look at the genome. Some search for changes in gene expression, inherited gaps in DNA, or patterns among so-called recombination hotspots, where matching chromosomes swap DNA more often than usual. Simon Myers, formerly a postdoc with statistician Peter Donnelly at Oxford University and now at the Broad Institute, examined more than 9000 hotspots found using the HapMap and a similar map by Perlegen Sciences Inc. in Mountain View, California.

Myers and his Oxford colleagues matched each of their hotspots to a nearby "coldspot" where DNA rarely recombines. They found two DNA motifs in particular that were common in hotspots. One seven-base sequence explained 10% of hotspots examined. The motif appeared to boost the chance of a hotspot in certain DNA stretches by up to five times.

Another group at the Broad Institute is examining data that were sequenced and publicly released by HapMappers but didn't make it into the final HapMap because they were deemed erroneous. "This project is kind of a dumpster dive," says the Broad Institute's Steven McCarroll. He and his colleagues found that thousands of the flaws are actually inherited DNA deletions. They've identified 10 commonly deleted genes, including two for sex steroid hormone metabolism and three for drug metabolism. They're now studying whether those deletions might contribute to disease.

Finally, in answer to the commonly asked question, "What now?" several groups are turning from humans to parasites. Dyann Wirth of Harvard School of Public Health in Boston is leading this latest haplotyping effort, which seeks to index genetic variation in *P. falciparum* by examining DNA samples collected from South and Central America, Asia, and Africa. So far, the group has identified 55,000 potential SNPs. Data like these, if they hold up, may help uncover new drug targets and explain drug resistance and the "functional effects of mutations," says Philip Awadalla of North Carolina State University in Raleigh, who's also studying *P. falciparum*'s gene variation.

–JENNIFER COUZIN

Published by AAAS

More than 25 million Americans have puzzling disorders.

Could You Have a Rare Disease?

By Dr. Ranit Mishori
Published: July 23, 2006
Full text available online on Wednesday, July 26, 2006

23 Comments about this article.
Jump to comments

WEB EXCLUSIVE

Rare Diseases: Are You at Risk?

With a sample of your hair, blood, skin or other body part, scientists can look at your genes and examine them for abnormalities. Genetic testing may:

- Confirm a suspected diagnosis for some diseases.
- Predict the possibility of a future illness.
- Tell if you are a carrier of a disease. While you may be unaffected, your children may be at risk.

Genetic tests can be done at different times during the life cycle.

- **In the prenatal period:** via blood tests, chorionic villus sampling (CVS) or amniocentesis.
- **Immediately after birth:** Most states have newborns tested for a few genetic disorders that can benefit from prompt treatment. Parents can request additional testing.

MORE TO KNOW

On The Web

The National Organization for Rare Disorders

The Office of Rare Diseases at the NIH

NIH's National Human Genome Research Institute

For information about genetic tests: GeneTests

Save Babies Through Screening: State-by-state listing of disorders screened

Recent Feature Articles

Matt Dillon: "You Don't Shut the Door on Hope" (07/23/2006)

Life After Stroke (07/16/2006)

The Summer Sky (07/16/2006)

• **Anytime thereafter:** Testing children or adults to confirm a diagnosis when they, or other family members, become sick.

Could It Be...?

There are a few things a doctor has to go on when trying to figure out why you are sick. Your symptoms are usually the best clue. But another lead may come from your ethnic origin. Certain ethnic groups are more likely to suffer from certain diseases and conditions. Here are some examples:

Caucasians

1. Cystic fibrosis—one of the most common inherited gene disorders in Caucasians. It affects the lungs and the digestive system.

2. Factor V Leiden—a blood-clotting disorder.

3. Biotinidase deficiency—an inherited disorder that causes a severe acid build up in the body.

4. Hereditary hemochromatosis—a disorder that cases a buildup of iron in the body.

5. Celiac disease—a disorder affecting the digestive system. It is the most common genetic disease in Europe and is found mostly in people of Italian and Irish descent. It is rarely diagnosed in African, Chinese and Japanese people.

African-Americans

1. Sickle-cell anemia—an inherited disorder that affects the red blood cells' ability to carry oxygen.

2. G6PD deficiency—a blood disorder that can result in the rapid destruction of blood cells in the presence of infection or certain drugs. It is also more common in Mediterranean countries.

Southeast Asians

1. Hemoglobin E syndromes—which may result in a variety of blood disorders.

Eastern European Jews

1. Tay Sachs disease—a fatal disorder in children that causes a progressive degeneration of the central nervous system.

2. Other genetic disorders—including: Bloom syndrome, Canavan disease, familial dysautonomia, Fanconi anemia, Gaucher disease, mucolipidosis type IV, Niemann-Pick disease.

Other groups:
1. Thalassemia—a group of blood disorders that mainly affect people of Mediterranean (Italian, Greek, North African), African and Southern Asian descent.

2. Maple syrup urine disease—a life-threatening inherited disorder that is more common in the Mennonite population.

3. Tyrosinemia—an inherited disorder that causes severe liver disease in infancy. It is very rare except in certain populations, including Canadian Inuits.

A word of caution: Any of these disorders can affect any individual regardless of the ethnic group to which he or she belongs.

On the Web

For more information about rare diseases and how to test for them:

The National Organization for Rare Disorders: rarediseases.org

The Office of Rare Diseases at the NIH: rarediseases.info.nih.gov

NIH's National Human Genome Research Institute: genome.gov

For information about genetic tests: genetests.org

Save Babies Through Screening: State-by-state listing of disorders screened: savebabies.org

Read, print and e-mail the full text of this article on Parade.com starting Wednesday, July 26

PARADE Home

READER COMMENTS | Add a comment

Intracranial Hypertension

By mirra_of_swd on 7/24/2006 12:19:AM

There are alot of rare conditions. The one I suffer from is Intracranial Hypertension. Intracranial Hypertension (IH) is the general name for the disorders in which the cerebro-spinal fluid (CSF) pressure within the skull is too high. A lot of people are treated for years for headaches and depression. It can cause blindness as well as in rare cases death if it is left untreated. There has yet to be a drug develpoed strickly for this condition and very few doctors who study it. When I was diagnosed my nero hadn't even heard of it(my othro dignosed me).It is diagnosed by normal cat and elevated spinal fluid checked by a spinal tap. more info go to www.ihrfoundation.org

Long-term effects of acute viral encepahlitis

By kingrex53 on 7/23/2006 10:29:PM

Some of the long-term effects of having acute viral encepahlitis are gi motility problems, hypoadrenalism, focus problems, migraines, decrease in IQ, difficulty remembering,, and an immune system unable to rid by body of viruses, Candida, etc.In addition, I have Type 2 diabetes, a genetic hypercoagulation defect,(lipoprotein a), and a mood disorder. Dr. Jay Goldstein MD, and Dr. John Martin, with the CCID in Southern California have studied the effets of acute viral encephalopathy and stealth viruses. Dr. Joseph BrewerMD and Dr. Carol Ryser MD are up to date on rare diseases and are excellent diagnosticians also. I am pleased and grateful for their help.

Mal de Debarquement

By DrM on 7/23/2006 10:24:PM

Have you ever felt like you were still on the boat when you got off a cruise or sailboat? The ground seems to move up and down, you rock and sway, and it goes away after a good night's sleep. Or, it stays with you for years. Some people, who suffer from Mal de Debarquement rock and sway 24/7 for years. They are fatiqued, some have brain fog and feel like they are walking on a trampoline. This can follow any motion activity including cruises, plane flights, amu******t park rides, rocking chairs, car rides. There is no known cause or cure, and it affects people with different levels of debilitation. Sometimes it goes away only to return after another motion activity. There is no research in progress at this point so many people go for years without a diagnosis. Often, it is a diagnosis by exclusion and one of thing that is common to most sufferers is that while riding in a car the feeling of movement disappears only to return with a vengenence when the car stops.

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REUTERS

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Home DNA tests 'snake oil,' U.S. congressman says

Fri Jul 28, 2006 4:33 AM IST

By Maggie Fox, Health and Science Correspondent

WASHINGTON (Reuters) - Home DNA kits that claim to warn people of their risk of diseases ranging from cancer to osteoporosis offer little real guidance and are often misleading, according to a Congressional report released on Thursday.

An investigation into 14 companies that sell the tests showed many gave meaningless information, and some then tried to sell consumers expensive "customized" supplements that were little different from grocery store vitamin pills.

The Food and Drug Administration and the Centers for Medicare and Medicaid Services said they were investigating the companies and checking to see whether more oversight was needed.

"Clearly consumers are being misled and exploited by this modern day snake oil and I am shocked to learn how little the federal government is doing to help consumers make informed decisions about the legitimacy of these tests," said Oregon Republican senator Gordon Smith.

Smith, who is chairman of the Senate Special Committee on Aging, held a hearing to release a report from the Government Accountability Office on the home test products.

Often called nutrigenetic tests, they usually involve a customer taking a swab from inside the cheek and sending it to the lab for analysis. The GAO took samples from two people and sent them to 14 different labs under fictitious names.

The GAO report found that the tests were not clinically valid and that companies mislead consumers by making unproven and ambiguous health-related predictions.

RISING DEMAND

"Demand for this type of service appears to be on the rise; one company estimates that it has sold over 35,000 nutrigenetic tests to consumers since it began selling the tests in the United States in 2003," the GAO's Gregory Kutz told the hearing.

Officials from the Food and Drug Administration and from the Centers for Medicare and Medicaid Services agreed with the GAO's findings that the tests were largely meaningless and said they were investigating.

Kathy Hudson, Director of the Genetics and Public Policy Center at Johns Hopkins University in Washington, said the FDA had not been clear on what its role should be in regulating the labs.

"The current regulatory environment fails to ensure the quality either of the laboratories performing genetic testing or of the tests they are offering," Hudson told the hearing.

She recommended that the Center for Medicare and Medicaid Services enforce regulations that might affect the

labs and make new rules under the 1988 Clinical Laboratory Improvement Amendments, under which laboratory tests are regulated.

Officials of some of the companies investigated by GAO defended their products and services.

"I support your efforts here and I believe we need to have more regulation," said Howard Coleman, chief executive officer of Genelex Corporation in Seattle, Washington.

But when asked why the companies returned 14 different profiles for DNA samples taken from just two people, Coleman answered: "Senator Smith, I would like to see those reports and go over them ...There may be explanations for this."

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PalmBeachPost.com

Genetic breakthroughs usher in personalized medicine

By Stephen Pounds

Palm Beach Post Staff Writer

Sunday, August 06, 2006

Matthew Green shaves, does his own laundry and can cook. He wiped down tables at the Beverly Hills Cafe in his hometown of Pembroke Pines until recently.

At 22, that would be normal. But some of Green's behavior isn't.

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A victim of a genetic malfunction called Fragile X syndrome, he still watches children's programs such as *Blue's Clues*, *Power Rangers* and *The Wiggles*. He avoids looking at people who speak to him, becomes hypersensitive to even small changes and has a poor attention span.

"Mentally, he's more like 6," said his mother, Randy Green of Pembroke Pines. "He can do a lot independently, but he still needs supervision."

Fragile X occurs when an errant gene on the X chromosome deprives the brain of a particular protein, causing autistic behavior, mental retardation and in some cases, epilepsy.

"It's relatively rare, and what that means is that there isn't a lot of impetus at drug companies to do anything about it," said Karen Fay, executive director of the Palm Beach-based Conquer Fragile X Foundation, which hands out grants for research into the genetic defect.

There's no cure for Fragile X syndrome, but recent advances in science may allow people such as Matthew Green to lead more normal lives. In a future of so-called personalized medicine, doctors will customize medical care to a person's genetic code for better diagnosis, more effective treatment and fewer unwanted drug side effects.

Still, there are many obstacles.

"We'll get there as fast as the science and technology permit, which in turn will encourage changes in the business model," said Edward Abrahams, president of the Washington D.C.-based Personalized Medicine Coalition. "There's a chicken-and-egg thing here."

The routine of plying a patient with different drugs until he or she responds has been the tried-and-true method for decades. It's what doctors practice, patients expect and the pharmaceutical industry feeds with its drug pipeline.

Enter the Human Genome Project. In 2003, scientists completed mapping the 3 billion bits of DNA in the human genome, the term applied to the 23 pairs of chromosomes in which genetic information is stored. With that done, researchers have begun to look for disease-causing flaws in the sequence and ways to treat the resulting health problems.

Cancer research has been an early beneficiary of personalized medicine. Genetic testing allows doctors to pinpoint the precise variety of cancer involved and the treatment that targets it.

"From the patient's point of view, they don't have a lot of time," said Mara Aspirall, president of the genetics unit at Genzyme Corp., a Cambridge, Mass.-based biotech giant. "It's all very good to say, 'There are a lot of drugs available. Let's start with the traditional treatment and move to newer ones.' But only 40 percent of lung patients last a year to try new medications."

Treatment was becoming more personalized, even before the human DNA was spelled out.

Under a program offered by the U.S. Food and Drug Administration's Office of Orphan Drug Development, a Bridgewater, N.J.-based company called Enzon came up 10 years ago with a drug called Adagen to treat a group of children with a shortage of an enzyme called adenosine deaminase. People who lack the enzyme have a severe immune-deficiency disorder known as "bubble-boy disease," which makes them mortally susceptible to a host of common infections.

"There were only 14 children in the U.S. with the disease. That's fairly personalized," said Marlene Haffner, who directs the FDA's orphan products division.

In 23 years, the FDA orphan program has shepherded 289 drugs to market, all for patient populations of fewer than 200,000 people.

In 2004, the agency launched an effort called the Critical Path Initiative to revive Big Pharma's drug pipeline. Today, many in Congress are calling it the next crucial step toward personalized medicine.

An FDA report issued in March said spending on biomedical research increased from $37 billion to $94 billion between 1994 and 2003 with no increase in product applications before the FDA. It said 2004 represented a 20-year low in new medical therapies launched on the market worldwide.

The FDA said genetic tests can solve Big Pharma's pipeline problem by weeding out of clinical trials the patients who won't respond to a new drug. But it won't be easy to win over the big drug makers.

Drug development for smaller populations is better suited to companies the size of Nabi

Biopharmaceuticals Inc. in Boca Raton than to drug giants such as Pfizer Inc. and Merck & Co. They focus on blockbuster drugs for large swaths of patients and pull in billions in sales, Nabi Senior Vice President Henrik Rasmussen said.

"For Pfizer or Merck, $200 million to $300 million would be useless," he said. "That's never going to compensate for the mass-market drug."

Still, Big Pharma's trade group, the Pharmaceutical Research and Manufacturers of America, says it endorses personalized medicine.

Smaller biotech firms, on the other hand, have embraced it.

Monogram Biosciences Inc. of South San Francisco extracts a sample of an HIV patient's virus to see how it reacts to any of the 20 or so drugs that are given in combination for AIDS treatment.

"A patient might be given a cocktail of three drugs and as the virus mutates, we can tell what drugs might fail and what they could be replaced with," said Alf Merriweather, Monogram's chief financial officer.

NitroMed Inc., a biotech firm in Lexington, Mass., markets a drug called BiDil to blacks with failing hearts. In 2003 and 2004, the company, with help from the Association of Black Cardiologists, held the first clinical trial of heart patients who identified themselves as blacks. Some of the participants were given typical heart medications while others were given those drugs plus BiDil.

A panel of doctors monitoring the studies stopped the trial after six months and suggested all the patients receive BiDil because of the striking results. The FDA approved BiDil for sale in 2005, heralding it as a step toward personalized medicine.

Even so, NitroMed still struggles to get Medicare Part D as well as state Medicaid payments for the use of BiDil. Company spokeswoman Jane Kramer said Medicare Part D gives patients "generally poor access to BiDil." Florida offers it to its Medicaid patients but neighboring Alabama and Georgia do not.

"Florida has been a leader, but other states have lagged behind... even though the FDA thinks that this is a great advance," Kramer said.

Gail Javitt, law and policy director of the Genetics and Public Policy Center in Washington, said the government must contend with several policy issues including reimbursement before personalized medicine takes off.

Some genetic tests have FDA or other government approval but many of the 900 tests now in use do not. A report last week by the Government Accountability Office said some direct-to-consumer genetic test kits that claimed to provide consumers with personalized diet and lifestyle recommendations don't work.

At the same time, labs that do legitimate testing aren't always reimbursed by medical insurance plans, though they can cost less than $50.

"Reimbursement... hasn't kept up with the growth in testing," Javitt said.

On the other side, Walter Hollinger, a senior medical director with Blue Cross and Blue Shield of Florida, said the insurer pays for genetic testing if a physician is using it to establish a diagnosis, based on two criteria — medical necessity and proven effectiveness.

Blue Cross relies on the doctor to determine medical necessity and refers to the FDA, the Centers for Medicare and Medicaid Services or even published medical journals to determine if the test has any history of success.

"The main determination is whether it's used to manage someone's care," Hollinger said.

In the public debate over personalized medicine, there's also the concern about privacy, said Julie Johnson, a pharmaceutical sciences professor at the University of Florida. Critics worry that if patients take genetic tests to determine their risk of disease, they might have trouble finding insurance coverage.

And, Johnson said, physicians accustomed to using the trial-and-error approach resist genetic testing.

"They've always done it a different way," she said.

Meanwhile, scientists are delving further into the genetic mysteries.

Researchers around the world are now taking part in the International HapMap Project, an effort to create a catalog — the HapMap — of genetic similarities and differences in human beings.

The goal is to compare the genetic sequences of different population groups to identify haplotypes, or chromosomal chunks where DNA differences occur. With the HapMap, researchers can compare haplotypes in groups that have specific diseases.

"We'll have a catalog of all those things. Before we didn't have the phone book. We were just dialing numbers randomly," said Chris Austin, director of the Chemical Genomics Center of the National Institutes of Health.

Some scientists, such as Claes Wahlestedt of Scripps Florida in Jupiter, are focusing on RNA as the next logical step toward personalized medicine. If DNA is a reference book for human life, RNA is a throw-away copy. It carries a duplicate of a person's DNA code out of a cell's nucleus and translates it into protein.

But much of the RNA doesn't carry any code. Wahlestedt believes this non-coding RNA is connected to cell and body functions.

"For many years, RNA was thought to be a boring middleman," Wahlestedt said. "We're thinking it is actually a new type of drug target."

Through the study of haplotypes and RNA, scientists hope to dig deeper into the makeup of cells to create more personalized drug treatments.

"Prevention is the key. That's the real dream for medicines. In the future, treating disease might be a drug or it might be a lifestyle change," Wahlestedt said. "The idea is to understand diseases better."

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The New York Times
nytimes.com

August 10, 2006

Ga. Researchers Begin New Gene Mapping

By THE ASSOCIATED PRESS

Filed at 10:20 p.m. ET

ATLANTA (AP) -- Atlanta-based researchers say they've begun mining a new kind of genetic data in a project that may help explain why some people are susceptible or resistant to certain diseases.

A summary of the work by Emory University researchers was released Thursday. It's being published in a scientific journal, Genome Research.

"The topic is quite exciting" and the Emory researchers are breaking new ground, said Lisa Brooks of the National Human Genome Research Institute, a federal agency. She is director of the Institute's genetic variation program.

Scientists think that in as soon as 10 years, doctors will be able to examine the DNA of newborn babies and compare it to a reference code of human DNA to make predictions about each infant's susceptibility to disease. Such information could help doctors know which medicines will work most effectively on that person if illness develops.

"We're entering an exciting new era of predictive health," said Scott Devine, an Emory assistant professor of biochemistry who co-authored the paper.

The Emory work should contribute to that, he added.

In 2000, scientists announced they had substantially finished mapping the genetic blueprint for all human cells. The breakthrough was heralded as ushering in a new era of medicine, which would -- and to some extent has -- led to new ways to test for and treat disease.

The finished human genome sequence was published in 2003, and was based on DNA from about a half-dozen people.

The mapping showed the human genome is built from billions of chemical building blocks that appear in pairs. The blocks come in four types: adenine (A), thymine (T) cystosine (C) and guanine (G).

Since then, scientists have been focused on mapping tiny variations in the genetic code of 36 additional people, in

an attempt to understand why, for example, some nonsmokers develop lung cancer while some lifelong smokers never get sick.

The variations are called "snips" -- or SNPs -- an abbreviation for "single-nucleotide polymorphisms." They involve single-block replacements. That is, part of one person's genetic sequence might read A-T-C, but a SNP might replace a G for the C in the next person, resulting in A-T-G.

A federally led mapping of the SNP variations was published last year.

In the new work, Emory researchers used the SNP mappers' data, but applied a new kind of computer-based analysis to look for another type of variation called an INDEL -- for insertion and deletion polymorphism.

In an INDEL, building blocks are added or deleted, not just switched on a one-for-one basis. And an insertion or deletion can involve thousands of blocks.

INDELs represent as much as 25 percent of all genetic variations, Emory researchers said. They already have been shown to be the cause of several genetic diseases, including cystic fibrosis, Devine said.

HEALTHCENTRAL

Some Dogs Carry 'Contagious' Cancer

Medical anomaly poses no threat to humans, experts say.

By Ed Edelson
HealthDay Reporter

THURSDAY, Aug. 10 (HealthDay News) -- Researchers are describing what seems to be a real-life medical nightmare: A cancer that spreads from animal to animal like an infection.

Luckily for humans, this malignancy occurs only in dogs, and there's no need for people to be worried about it, experts say.

"It's a scientific curiosity," said Robin Weiss, professor of viral oncology at University College London, and a member of the team reporting the discovery in the journal *Cell*. "There is no evidence of transfers of human cancers from one person to another, except in very special circumstances, so we should not say that a human cancer patient is dangerous to others."

The cancer, called canine transmissible venereal tumor (CTVT), was first isolated from 16 dogs in Italy, India and Kenya. In each case, a study of the tumors' genetic material showed that it differed from that of the dog in question -- suggesting that it had been passed from another dog.

Further study of cancers from 40 other dogs in five continents found that the tumors were almost genetically identical, meaning that they originally came from a single source and had somehow spread across the globe.

Working with geneticists and computer experts in Chicago, the researchers compared the genetic material of tumors to that of specific breeds of dogs. They concluded that the cancer most likely arose more than 250 years ago -- perhaps as long as 1,000 years ago -- in a wolf or Asian dog such as a Husky or Shih Tzu.

CTVT is transmitted primarily through sexual contact, but experts believe it can also be picked up as dogs lick, bite or sniff tumor-affected areas. It is seldom fatal and usually disappears in three to nine months, just long enough for the dog to pass it on.

"One aspect where this is related to human cancer is not in the mode of transmission, but what it tells us about the nature of cancer," Weiss said.

Generally, as cancers become more aggressive, they become less stable genetically, he said. But CTVT has had the same genetic makeup for centuries and is "the oldest tumor cell lineage known to science," which means that it has become genetically stable, Weiss said.

"This questions the theory of instability," he said. "I don't think that instability is inevitable as a tumor gets worse and worse."

The report also raises wildlife conservation issues, added Elaine Ostrander, chief of the cancer genetics branch at the U.S. National Human Genome Research Institute, who wrote an accompanying commentary.

Similar cancers are known to exist in two other species, the Tasmanian devil and the Syrian hamster, Ostrander said. For these types of endangered species, exposure to CTVT might endanger the population's survival, she wrote.

There appears to be no danger to humans from the sort of cancers seen in these animals, Ostrander said. While CTVT may occur in stray dogs, pedigreed dogs are usually not allowed casual sex, and the cancer "can't be transmitted to humans by handling dogs," she said.

"We always wonder when we see something in the animal kingdom if we will see the same thing in humans," Ostrander said. "We don't see any human evidence in this case."

More information

There's more on the genetics of cancer at the U.S. National Cancer Institute (www.nci.nih.gov).

This article can be accessed directly at:
/newsdetail/408/534299.html

BCM
Baylor College of Medicine

- Ross Tomlin713-798-7973 htomlin@bcm.tmc.edu

- E–mail this article to a friend

Advanced bovine genome assembly, genetic resources released: Cow genome project heads for home

HOUSTON -- (August 14, 2006) -- Researchers from the Bovine Genome Sequencing Project recently announced the release of a comprehensive set of genome resources into freely available public databases. These new assets for bovine researchers include the most complete and accurate genome sequence to date, an upgraded genetic map, and a new set of two million DNA base differences for use as DNA sequence polymorphisms.

Contributors to the $53 million international effort to sequence the genome of the cow (Bos taurus) include: the National Human Genome Research Institute, which is part of the National Institutes of Health; the U.S. Department of Agriculture's Agricultural Research Service and Cooperative State Research, Education, and Extension Service; the state of Texas; Genome Canada via Genome British Columbia, The Commonwealth Scientific and Industrial Research Organization of Australia; Agritech Investments Ltd., Dairy Insight, Inc. and AgResearch Ltd., all of New Zealand; the Kleberg Foundation; and the National, Texas and South Dakota Beef Check-off Funds.

The sequencing of the bovine genome was conducted at the Baylor College of Medicine Human Genome Sequencing Center in Houston. The new genome sequence is 2.9 billion DNA base pairs, similar to the human and other mammalian genomes. It incorporates about one-third more data than earlier versions. The new information comes from sequencing 20,000 clones containing large pieces of bovine DNA (BACs), created at the BACPAC Resource Center at Children's Hospital Oakland Research Institute in Oakland, Calif. and mapped at the Michael Smith Genome Sciences Centre at the British Columbia Cancer Agency in Vancouver.

The use of the BAC data allows a more structured assembly of DNA sequences into a complete genome, more effectively addressing challenges posed by repeated sequences. New techniques developed at the BCM-HGSC allowed the BACs to be sequenced in groups rather than individually, reducing cost and effort. The new high-quality bovine sequence, which covers 95 percent of the genome, will enable researchers to make accurate gene predictions and evolutionary comparisons for this important animal.

The Hereford breed was selected for the bulk of the sequencing project. Although an inbred animal was used, heterozygosity in the sequence was detectable. The sequence data was analyzed to identify likely single nucleotide polymorphisms (SNPs), representing positions where members of each chromosome pair of the diploid animal differ in DNA sequence. About two million SNPs were found, and will be of great utility for high-resolution studies of the differences between breeds and relating associated traits to the genome.

DNA sequencing to sample other breeds to detect genetic differences was also performed on Holstein, Angus, Jersey, Limousin, Norwegian Red and Brahman animals. DNA differences between these breeds will expand the discovery of traits for better meat and milk production and to model human disease.

The genome sequence was also used to order over 50,000 genetic markers, creating the most dense and complete genetic map of the bovine genome to date. This genetic map will be invaluable in identifying heritable traits and conditions of interest in agriculture and medicine. About 17,000 of the markers were contributed by several groups within the BGSP and combined at the U.S. Department of Agriculture's Meat Animal Research Center in Clay Center, Nebraska before being integrated with the genome sequence to contribute to the map. Another 35,000 markers were

selected from the SNP collection, and from ESTs previously aligned to the genome. Mapping reagents for about 9,000 of these are already commercially available and are being used in bovine genetic studies around the world.

Sequencing of the bovine genome began in December 2003. With the release of these new data, the project now heads into the final analysis phase. The bovine genome sequence will aid agricultural researchers to improve health and disease management of cattle and enhance the nutritional value of beef and dairy products. Medical researchers will also use the bovine information to interpret the human genome and thereby develop better ways of treating and preventing disease.

Researchers can access the sequence data through the following public databases:

- Baylor College of Medicine Human Genome Sequencing Center (www.hgsc.bcm.tmc.edu)
- GenBank (www.ncbi.nih.gov/Genbank) at NIH's National Center for Biotechnology Information
- EMBL Bank (www.ebi.ac.uk/embl/index.html) at the European Molecular Biology Laboratory's Nucleotide Sequence Database
- DNA Data Bank of Japan (www.ddbj.nig.ac.jp)

The data will also be viewable through NCBI's Map Viewer (www.ncbi.nlm.nih.gov), UCSC Genome Browser (www.genome.ucsc.edu) at the University of California at Santa Cruz, and the Ensembl Genome Browser (www.ensembl.org) at the Wellcome Trust Sanger Institute in Cambridge, England.

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washingtonpost.com

Scientists Find Brain Evolution Gene

By SETH BORENSTEIN
The Associated Press
Thursday, August 17, 2006; 6:48 PM

WASHINGTON -- Scientists believe they have found a key gene that helped the human brain evolve from our chimp-like ancestors. In just a few million years, one area of the human genome seems to have evolved about 70 times faster than the rest of our genetic code. It appears to have a role in a rapid tripling of the size of the brain's crucial cerebral cortex, according to an article published Thursday in the journal Nature.

Study co-author David Haussler, director of the Center for Biomolecular Science and Engineering at the University of California, Santa Cruz, said his team found strong but still circumstantial evidence that a certain gene, called HAR1F, may provide an important answer to the question: "What makes humans brainier than other primates?" Human brains are triple the size of chimp brains.

Looking at 49 areas that have changed the most between the human and chimpanzee genomes, Haussler zeroed in on an area with "a very dramatic change in a relatively short period of time."

That one gene didn't exist until 300 million years ago and is present only in mammals and birds, not fish or animals without backbones. But then it didn't change much at all. There are only two differences in that one gene between a chimp and a chicken, Haussler said.

But there are 18 differences in that one gene between human and chimp and they all seemed to occur in the development of man, he said.

Andrew Clark, a Cornell University professor molecular biology who was not part of Haussler's team, said that if true, the change in genes would be fastest and most dramatic in humans and would be "terrifically exciting."

However, the gene changed so fast that Clark said that he has a hard time believing it unless something unusual happened in a mutation. It's not part of normal evolution, he said. Haussler attributed the dramatic change to the stress of man getting out of trees and walking on two feet.

And it's not just that this gene changed a lot. There is also its involvement with the cerebral cortex, which is responsible for some of the more complex brain functions, including language and information processing.

"It looks like in fact it is important in the development of brain," said co-author Sofie Salama, a research biologist at Santa Cruz who led the efforts to identify where the gene is active in the body.

The scientists still don't know specifically what the gene does. But they know that this same gene turns on in human fetuses at seven weeks after conception and then shuts down at 19 weeks, Haussler said.

On the Net:

http://www.nature.com/nature

World

Half a million Britons set for DNA disease quest

REUTERS

By Reuters
Tuesday August 22, 07:31 AM

By Ben Hirschler

LONDON (Reuters) - A project to collect DNA samples from half a million Britons to unpick the genetic basis of killer diseases including cancer got the go-ahead on Tuesday, marking the start of the world's biggest medical experiment.

A team of international scientific and medical experts said the success of a local three-month pilot phase, involving 3,800 participants around Manchester, meant the UK Biobank project could now be rolled out nationwide from the end of 2006.

Over the next four years, blood and urine samples will be collected from volunteers aged 40 to 69, to help scientists unravel the genetic foundations of common diseases, including cancer, heart disease, diabetes, dementia and joint problems.

"For decades to come, the UK Biobank resource should provide researchers around the world with vital insights into some of the most distressing diseases of middle and old age," principal investigator Professor Rory Collins said in a statement.

The mapping of the human genome in 2000 opened the door to the detailed analysis of genes but experts are still grappling to understand how they interact with lifestyle and environment to determine why some people become sick and others do not.

In the long term, scientists believe the project could improve prevention, diagnosis and treatment of diseases and help to explain why certain people

react differently to medications.

The 61 million pound ($115.6 million) project will be funded by the British government, the Wellcome Trust medical research charity and other sources.

Some researchers have expressed concerns about the design, size and cost of the project, but Collins said he was confident it would produce valuable medical breakthroughs.

A total of around 10 million samples are expected to be collected from the half million volunteers. That genetic data will be cross-referenced against information about patients' subsequent health, obtained with their permission.

Researchers from around the world will be able to apply to UK Biobank for access to the resource but there will be strict security systems to protect participants' privacy.

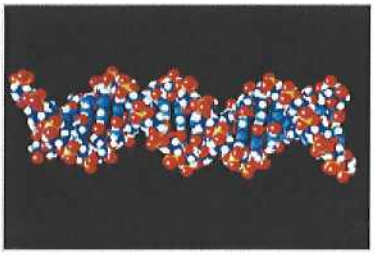
Health Minister Andy Burnham said Britain was "leading the world" with the project, which would underpin the country's academic and industrial research capability.

No one organisation or commercial body will have exclusive access to the data but pharmaceutical companies are expected to be able to use the results to help design new drugs and diagnostic tests.

The Quest for the $1,000 Human Genome

By NICHOLAS WADE
Published: July 18, 2006

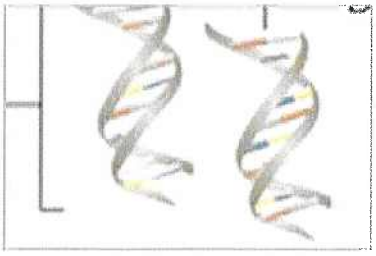
As part of an intensive effort to develop a new generation of machines that will sequence DNA at a vastly reduced cost, scientists are decoding a new human genome — that of James D. Watson, the co-discoverer of the structure of DNA and the first director of the National Institutes of Health's human genome project.



LLNL/Science Source, via Photo Researchers

Decoding a person's genome is at present far too costly to be a feasible medical procedure. But the goal now being pursued by the N.I.H. and by several manufacturers, including the company decoding Dr. Watson's DNA, is to drive the costs of decoding a human genome down to as little as $1,000. At that price, it could be worth decoding people's genomes in certain medical situations and, one day, even routinely at birth.

Multimedia



Graphic: Genomes Decoded Here

Low-cost decoding may bring the genomic age to the doctor's office, but it will also raise quandaries about how to safeguard and interpret such a wealth of delicate and far-reaching personal information.

The first human genome decoding, completed by a public consortium of universities in 2003, cost more than $500 million. With the same technology, dependent on DNA sequencing machines made by Applied Biosystems, a human genome could probably now be decoded for $10 million to $15 million, experts say.

John Dunn for The New York Times

CLINICAL PROMISE A DNA sequencing machine at Cold Spring Harbor Laboratory.

Much greater efficiency is expected from the new generation of DNA sequencing machines, based on different, highly miniaturized technologies. One machine, made by 454 Life Sciences, has been on the market since March 2005. Another, made by Solexa, will start shipping this summer. Applied Biosystems will start marketing its own next-generation machine next year.

Last month, at a training course organized by the Cold Spring Harbor Laboratory on Long Island, researchers were learning how to use the DNA decoding machines made by 454 Life Sciences. Looking like a hybrid between a washing machine and a giant iPod, the machines cost $500,000 each, not counting the computer software needed to analyze the results.

At their heart lies a plate of light-sensitive chips, the same as those used in telescopes for detecting faint light from distant stars. On top of the plate sits a glass slide pitted with thousands of tiny wells, each containing a fragment of the DNA to be decoded.

As each unit of DNA is analyzed in a well, a flash of light is generated by luciferase, the enzyme that fireflies use to make themselves glow. The telescope plate records the twinkling lights from each well and, at the end of the run, which lasts four or five hours, the sequence of units in each well's DNA fragment has been recorded. The fragments are about 100 units in length, and from their overlaps a computer can then be set to piece together the entire genome they come from.

In the training course, the project was to analyze DNA from a Tasmanian devil, a marsupial afflicted with a mysterious malady called devil facial tumor disease. The researchers found that the genome was laden with a virus that had integrated its sequence into the devil's DNA.

The 454 machine can assemble small genomes like those of bacteria, which perhaps accounted for the presence at the course of three scientists from the Department of Homeland Security. But the human genome is about 600 times larger than a bacterium's and includes many repetitive sequences that, like identical pieces in a jigsaw puzzle, make the solution much harder.

At the Cold Spring Harbor course, researchers heard Dr. Watson, the laboratory's chancellor, say that 454 Life Sciences had asked to sequence his genome with their new machine. Only two human genomes have been sequenced to date. The genome sequenced by

the public consortium was a mosaic of DNA from several anonymous people. The consortium's rival, Celera Genomics, prepared a draft sequence, most of it from the genome of its former president, Dr. J. Craig Venter.

Dr. Watson told the students that he had given the company permission to publish the sequence of his genome, "provided they didn't release to the world that I have some disease I don't want to know about."

Genomic information can already reveal a lot and will reveal much more as the roles of new genes are discovered.

"I think that personal genetic information should ordinarily be kept secret," Dr. Watson said. "But I have said that 454 can put mine out there, even though it's saying something about my sons."

So far, however, 454 Life Sciences has not published Dr. Watson's genome, and it is not clear how much progress the company has made. Christopher K. McLeod, its chief executive, said, "Technically, we've done a lot of good work on it." But, he added, "I don't think we want to discuss where we are."

Mr. McLeod expressed reservations about releasing personal genetic information, despite having Dr. Watson's permission to do so. "Jim feels there are certain things he'd be comfortable releasing," he said. "I'm not sure we would agree."

Another factor may be that the company is developing a more powerful model of its machine that will be able to read DNA fragments that are 200 or even 400 units in length. These longer-read lengths should make it more feasible to decode large genomes, like those of people.

The 454 machine is at present being bought chiefly by researchers and by the large genome sequencing centers established by the public consortium. But it has begun to show promise for the clinic. One new use is in screening tumors for genes known to be mutated in cancer, a task that existing machines do not do well. Spotting which mutation has occurred in a patient's tumor can help in the choice of chemotherapy.

Although the 454 model is the only next-generation DNA sequencing machine on the market, it will be joined this summer by the machine from Solexa. The Solexa instrument, which will cost $400,000, works on somewhat similar principles but uses fluorescent dyes to visualize the structure of DNA. And next year Applied Biosystems will introduce its next-generation machine, based on a technology developed by George Church of Harvard, said Dennis A. Gilbert, the company's chief scientific officer.

Each of the manufacturers claims special advantages for its technology, ensuring that researchers will have a rich choice.

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Gibbon, It's Time for Your Close-Up

By Elizabeth Pennisi
*Science*NOW Daily News
20 July 2006

The National Human Genome Research Institute (NHGRI) in Bethesda, Maryland, is getting its primate house in order. First, the institute played a major role in sequencing the human genome. Then the chimp's DNA got the all-star treatment. And when comparing the two genomes proved incredibly useful for understanding our own DNA, NHGRI set its sights on the rhesus macaque, marmoset, orangutan, and gorilla. Now the gibbon is getting in line.

Next!
The gibbon genome will be the seventh primate genome sequenced.

Credit: JIM ZUCKERMAN/GIBBON CONSERVATION CENTER

Yesterday, NHGRI added the northern white-cheeked gibbon (*Nomascus leucogenys*)--and 15 other species--to its burgeoning list of genomes to decipher. Once the gibbon genome is in hand, evolutionary biologists will have a sequence for each of the major branches of the primate family tree. The work should help researchers understand primate evolution and the role of genes in disease.

From the moment the first complex organism--a nematode--was sequenced in 1998, researchers have struggled to make sense of a veritable alphabet soup of A's, T's, G's, and C's. Sequencing other genomes has helped: Comparing the DNA of related organisms has been key to identifying regulatory regions of DNA and other essential genome components. To continue its quest to understand how genomes work, NHGRI has regularly solicited proposals from researchers asking them to recommend the next candidates for sequencing.

The gibbon won out because it's a second cousin to humans and, as such, will eventually help biomedical researchers pinpoint the genetic bases of disease, says NHGRI Director Francis Collins. The institute expects to have the genome sequenced within 3 years. NHGRI also agreed to improve on the sequences of the elephant, cat, bat, rabbit, armadillo, guinea pig, and tree shrew, mammals for which NHGRI had previously decided to produce just a small amount of sequence.

But at the same time that NHGRI is working on species at the tips of the evolutionary

tree, the institute is also reaching down to the roots. It announced that its sequencing centers will decipher the genomes of 10 protists, some of which have closely related colonial and unicellular species. In doing so, researchers hope to pinpoint the genes key to the evolution of multicellular life. Also new to the sequencing pipeline are five common fungal pathogens and 50 yeast strains.

As each new genome sees the light of day, it should help researchers better understand our own genome. "There's so much about the genome that we have relatively little information about," says David Anderson, director of the Washington National Primate Research Center in Seattle, Washington. "You want to get as much comparative sequence as you can."

Related site

- List of organisms NIH is sequencing

Leadon guilty of scientific misconduct, it announced on 8 June. Leadon, formerly a professor at the University of North Carolina at Chapel Hill, was previously found guilty of falsifying research findings by a university panel in 2003. The US federal finding said that eight publications and several grant applications were affected.

Previous retractions of Leadon's papers left the field in disarray (see *Nature* 435, 1015; 2005). He has now signed an agreement that forbids him from receiving federal grants or consulting for the government for five years. He must also ask to retract three more articles.

Leadon denies any misconduct. "I did not engage in scientific misconduct — an error was discovered in the experimental protocol," he wrote in a statement e-mailed to *Nature*. He says he entered into a settlement because he cannot afford the legal costs of fighting his case.

NIH cash boosts bid for more knockout mice

Two public mouse repositories last week received $800,000 from the National Center for Research Resources at the US National Institutes of Health (NIH) in Bethesda, Maryland. The move is part of a plan to make genetically modified 'knockout' mice more easily accessible to researchers.

M. BARTLETT/NHGRI

It's a knockout: NIH funds will make another 300 types of mouse mutant available to researchers.

Knockout mice have become a powerful tool in basic and medical research. But three-quarters of the approximately 4,000 knockout mouse lines described in the scientific literature have not yet been placed in public repositories. The money will be distributed to the Mutant Mouse Regional Resource Centers at the University of California, Davis, and the Harlan facility of the University of Missouri, Columbia, with the goal of making available more than 300 existing mouse mutants. These repositories will help an NIH project that aims to create a knockout mouse for each of the 20,000 genes in the mouse genome.

Japan steps up attempt to overturn whaling ban

Japan has won an unprecedented, if largely symbolic, victory at the International Whaling Commission (IWC).

On 18 June, members passed a resolution noting "concern that the IWC has failed to meet its obligations". Japan feels that a 1986 moratorium on commercial whaling has continued "irrespective of stock conditions" and in opposition to the original goal of the commission, says Hideki Moronuki, a spokesman at Japan's fisheries ministry.

The resolution calls for a "normalization" of the commission's operations, and notes that "the moratorium which was clearly intended as a temporary measure is no longer necessary". But support for the resolution, a narrow 33–32 vote, falls far short of the three-quarters majority needed to overturn the moratorium. "It's a first step," says Moronuki.

Correction

The News story 'Born or made? Debate on mouse eggs reignites' (*Nature* **441,** 795; 2006) should have said that the mice in Jonathan Tilly's study expressed GFP in egg cells whereas the mice in Amy Wagers' study expressed GFP in all cells.