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Naughty or Nice, We Give Presents To the Deserving

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**Naughty or Nice, We Give Presents To the Deserving**

By Sharon Begley, The Wall Street Journal, 845 words
Dec 23, 2005

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IN THE SPIRIT of the season, we offer gifts to special men, women and machines for their contributions to science in 2005, even though not all have followed the song's advice to be good for goodness sake.

To atmospheric scientist Kerry Emanuel of MIT, whose prescient study linking global warming to greater hurricane intensity appeared weeks before Katrina and the rest of this record storm season -- beachfront property far from storm zones.

To paleontologist Mary Schweitzer and colleagues: for discovering the first soft tissue, complete with bone cells and red blood cells, from dinosaur fossils -- a "Jurassic Park" DVD.

To stem-cell pioneer Hwang Woo Suk: for cloning (he said) the first dog and for creating (he claimed) stem cells that precisely match the DNA of each of the 11 people providing donor nuclei, raising hopes for tailor-made cell therapy -- bioethics lessons. He admitted that some of the eggs he used were paid for, or donated by lab underlings, an ethical no-no, and the cloning claim is now under a cloud.

To the Huygens space probe: for becoming the first craft to land on another planet's moon when it touched down on Saturn's Titan, finding conditions like those on Earth when life first arose as well as complex organic molecules in Titan's clouds -- five golden rings.

To the orbiting Mars Express: for spying a flat area near the Martian equator that may be a huge frozen sea, raising the odds that life might exist below the planet's surface -
- little green men.

To the Dover, Pa., school board: for embracing intelligent design and creationism, which got it voted out of office and sued -- a pair of Darwin's finches. This week a

judge ruled against the board, citing the "breathtaking inanity" of its policy and finding it "ironic" that board members "who so staunchly and proudly touted their religious convictions in public, would time and again lie to cover their tracks" and disguise the fact that their real purpose was to foster religion, not intelligent science.

TO ANTHROPOLOGIST Michel Brunet: for discovering additional pieces of Sahelanthropus, the oldest known hominid (ancestor of humans and not apes), in Chad -- a pair of cuff links, including a missing one.

To Sen. Larry Craig of Idaho: for killing federal funding for the Fish Passage Center of Portland, Ore., because he disagreed with scientific findings that led a judge to order the release of water from dams in the Northwest to save endangered salmon -- a stale bagel, no lox.

To Spirit and Opportunity, the Mars Rovers: for zipping around the red planet, finding signs of life-sustaining water, seven times as long as they were expected to -- an Energizer Bunny.

To BiDil: for winning FDA approval for use by blacks only, making this heart drug the first race-based medicine and pushing the dialogue on the important question of whether race has any biological validity -- a guest appearance on "Oprah" with cardiologist Mehmet Oz.

To RHIC, the particle collider at Brookhaven National Laboratory: for producing a quark-gluon stew more like liquid than gas, different enough from what theorists believed first emerged from the big bang to send them back to their calculations -- primordial soup.

To Deep Impact: for pinpoint accuracy in reaching Comet Temple 1 and sending a probe into its heart to fathom the origins of the solar system -- sunblock.

To the ivory-billed woodpecker: for hanging in there after all -- protected habitat.

To geneticist Jody Hey: for using mitochondrial DNA analysis to discover that a mere 70 adults of reproductive age crossed the Bering Strait millennia ago to found the population of North America -- a turquoise compass.

TO BIOLOGIST Michael Krutzen: for discovering that bottlenose dolphin mothers teach offspring to use sponges to forage for food, the first evidence of cultural transmission of behavior in nonprimates, and to Thomas Breuer and his team for discovering that gorillas in the wild use tools -- Swiss army knives.

To astronomers Geoff Marcy and Paul Butler: for discovering the most Earth-like planet beyond our solar system, a solid orb that's too hot for life but which improves the odds that there are life-friendly planets out there -- ET's phone.

To the international team that sequenced the chimp genome: for showing that chimps differ from us by only 1% at the level of DNA, and for paving the way to discoveries about which of the 40 million genetic differences account for the key distinctions

between the two species: a private screening of "Bedtime for Bonzo."

To geneticist Manel Esteller: for discovering that as identical twins go through life, their genomes randomly acquire different "on" and "off" tags, the best explanation yet of why identicals diverge with age -- Doublemint gum.

To the two teams that sequenced the genome of the 1918 pandemic flu and partially reconstructed the virus: for finding clues to how it became so deadly, but also raising fears that bioterrorists might do the same thing -- Tamiflu.

And to all, a good night.

You can email me at sciencejournal@wsj.com.

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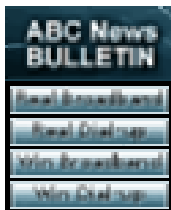
JULY 2005

Identical twins grow apart with age



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Identical twins grow apart with age

Identical twins grow apart genetically as the years pass, according to a team of European and US researchers.

Their study of identical twins shows the genetic code itself does not change, but rather chemical changes after birth alter the way the gene is expressed, a process known as epigenetics.

The study, which involved researchers and twins in Spain, Denmark, Britain, Sweden and the United States, can help shed light on how environment and genes interact to produce disease and ordinary differences between people.

They studied 80 twins from Spain and found significant epigenetic differences in 35 per cent of them.

The younger pairs of twins were identical, while the older pairs were more likely to differ from one another.

"Most importantly, we found a direct association between the remarkable epigenetic differences observed and the age of the monozygotic (identical) twins: the youngest pairs were epigenetically similar, whereas the oldest pairs were clearly distinct," the researchers wrote in this week's edition of the *Proceedings of the National Academy of Sciences*.

"Our study reveals that the patterns of epigenetic modifications in (identical) twin pairs diverge as they become older."

Identical twins occur in one out of every 250 births around the world.

But although their genetic codes are virtually identical, there are clear differences that are obvious to more than just their mothers.

For instance, psychiatric diseases such as schizophrenia and bipolar disease do not occur

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uniformly among identical twins, and there are often physical differences.

"There are several possible explanations for these observations, but one is the existence of epigenetic differences," Manel Esteller of the Spanish National Cancer Centre in Madrid and colleagues wrote.

The researchers say this supports theories that environmental factors, such as smoking, diet and exercise, affect DNA directly.

They say it is also possible that just as DNA mutations occur with simple aging, the epigenetic effects on genes also "drift" with age.

-Reuters

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J U L Y 2 0 0 5

Explaining Differences in Twins

The New York Times

July 5, 2015

Explaining Differences in Twins

By NICHOLAS WADZ

Identical twins possess exactly the same set of genes. Yet as they grow older, they may begin to display subtle differences.

They may start to look different, develop different diseases or slide into different personalities. Women who are identical twins may differ in their fertility or in the age at which they reach menopause.

These discrepancies are usually attributed to ill-defined differences in environment.

But a whole new level of explanation has been opened up by a genomic survey showing that identical twins, as they grow older, differ increasingly in what is known as their epigenome. The term refers to natural chemical modifications that occur in a person's genome shortly after conception and that act on a gene like a gas pedal or a brake, marking it for higher or lower activity.

Identical twins have the same set of epigenetic marks on the genome when they are born. But differences in the epigenome emerge as the twins grow older and become greater the longer they live apart, say a team of researchers led by Dr. Manel Esteller of the Spanish National Cancer Center in Madrid.

Their report appears in today's issue of *The Proceedings of the National Academy of Sciences*.

"This is one of the most fascinating things I have read," said Dr. Nancy Segal, a psychologist who studies twins at California State University at Fullerton and the author of "Indivisible by Two," a forthcoming book on twins. By giving us a handle on something specific, it opens up many new avenues of inquiry as to why twins are different.

There are two possible explanations for Dr. Esteller's findings. One is simply the well-known fact that epigenetic marks are lost as people get older. Because the marks are removed randomly, they would be expected to occur differently in two members of a twin pair.

A second possible explanation is that personal experiences and elements in the environment - including toxic agents like tobacco smoke - feed back into the genome by changing the pattern of epigenetic marks.

Dr. Esteller believes he is seeing both processes at work. The evidence for the second process, he said, is that twins who reported that they had lived apart the longest also had the greatest differences in their epigenome.

"This is a way for the genome to be responsive to the environment," he said, noting that it is easier for chemical marks on the genome to change than for the genome itself to mutate.

His study suggests that the epigenome may be involved in many diseases that can affect identical twins differently, like schizophrenia, bipolar disorder and cancer. Although schizophrenia evidently has a genetic component, the epigenome may hold the clue to its nongenetic aspects.

Differences between identical twins could also help pinpoint the epigenetic differences that

contribute to cancer. "We think that epigenetic changes are very common in cancer," said Dr. Peter A. Jones, the president of the American Association for Cancer Research and a professor at the University of Southern California.

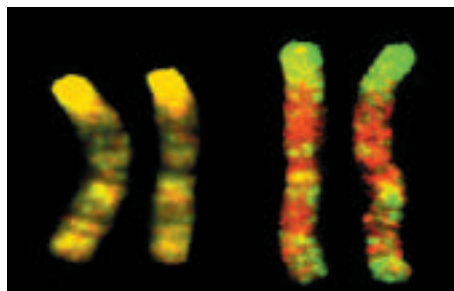
Dr. Jones said Dr. Esteller's finding "is exceptionally interesting in that it underlines the importance of epigenetic changes in human development and disease."

Dr. Jones recently convened a workshop to discuss starting an international human epigenome project. The proposal could rival the Human Genome Project in complexity because the human genome is the same in every cell of a person's body, while the epigenome is expected to be different for each of the 250 or so human cell types.

Among the most important components of the epigenome are small chemical handles known as methyl groups, which are added directly to the chemical units of DNA.

A wave of demethylation occurs in a sperm's genome shortly after an egg is fertilized, followed by the extensive readdition of methyl groups in early embryonic development.

These methyl groups, which generally inhibit the activity of the genes in which they occur, tend to be lost during aging. Dr. Esteller's team studied the total amount of methylation in the twins' genomes, as well as another kind of epigenetic modification, the addition of acetyl groups to the histone proteins that act as a scaffolding and as a control system for DNA.



Chromosome 1 in 3-year-old identical twins, left, and, showing differences, right, in 50-year-olds.

J U L Y 2 0 0 5

Twin Data Highlight Genetic Changes

washingtonpost.com

Twin Data Highlight Genetic Changes

Minor Differences Increase With Age

By Rick Weiss
Washington Post Staff Writer
Tuesday, July 5, 2005, A02

A mysterious biological mechanism that subtly changes the way people's genes behave may account for many of the surprising differences between identical twins, researchers announced yesterday.



These identical twins were part of a scientific study documenting subtle changes in people's genes caused by environmental influences, such as diet or stress.

(By Mario F. Fraga – Spanish National Cancer Center)

Geneticists said the new work, by an international team of scientists who studied the DNA of more than 100 pairs of twins, strengthens the case that a fledgling research field called epigenetics holds the long-sought answer to one of biology's toughest questions: How do environmental influences, such as exposure to pollutants, consumption of certain foods or perhaps even powerful emotional experiences, produce lasting and potentially life-altering changes in a person's DNA?

Beyond its potential importance for understanding differences in identical twins, epigenetics could explain many of the twists of fate that affect ordinary people -- why one person may be struck by cancer, for example, while another is spared, even though neither's DNA harbors a cancer-causing mutation.

"This is how the environment talks to the genome," said Rudolf Jaenisch, a geneticist at the Whitehead Institute for Biomedical Research in Cambridge, Mass., who was not part of the project. "This paper says lifestyle, or environmental influences or whatever you want to call them, have a real influence on your DNA."

Stephen Bayliss, a professor of oncology and medicine at Johns Hopkins Medical Institutions in Baltimore, said the new work points to the roots of a host of chronic diseases. "This could lead to far-reaching revelations about how our environment breeds predispositions for lots of diseases, like diabetes, cancer and heart disease."

Human cells have tens of thousands of genes inside them, each with its own job, such as producing energy or overseeing cell division. But only certain genes are active at any given time or in any cell type while the rest are appropriately dormant -- a grand orchestrator that adds up to a smooth-running life.

The new research, led by Mario F. Fraga and Marel Esteller of the Spanish National Cancer Center in Madrid, focused on two biological mechanisms that influence gene activity. In one, called DNA methylation, enzymes inside a cell attach a minuscule molecular decoration to a gene, deactivating that gene. In the other, called histone acetylation, a dormant gene is made active again.

These altered genetic settings can last a lifetime (though they are not passed down to a person's offspring) and can be important if, say, the gene turned off is one that protects against cancer. The extent to which epigenetic changes are preprogrammed from birth or spurred by factors outside the body has been unclear.

In the new work, described in today's issue of Proceedings of the National Academy of Sciences, researchers measured the extent to which twins of various ages, from 3 to 74, differed in the number

and variety of genes that had been either turned on or shut down by epigenetic processes. They found that young twins had almost identical epigenetic profiles but that with age their profiles became more and more divergent.

In a finding that scientists said was particularly groundbreaking, the epigenetic profiles of twins who had been raised apart or had especially different life experiences -- including nutritional habits, history of illness, physical activity, and use of tobacco, alcohol and drugs -- differed more than those who had lived together longer or shared similar environments and experiences.

Small epigenetic events before birth probably account for many of the minor distinguishing differences in the appearance, personality and general health of young twins, Estella said, and a lifetime of further epigenetic changes gradually increases individuality.

"Both nature and nurture are acting on these twins," Estella said. "Epigenetics is the bridge or the intermediary between them."

Francis Collins, director of the National Human Genome Research Institute, said the work adds to an emerging recognition that scientists must move beyond their long-standing focus on the inherited spelling of people's DNA code and the occasional mutation or outright "misspelling." Epigenetic changes do not alter genetic spellings but may account for as many cases of cancer and other diseases as full-blown gene mutations, he said.

"Here's something where Mendel, Watson and Crick et al seem to have missed some crucial goodies," said Collins, referring to pioneers of genetic research. Collins's institute, along with the National Cancer Institute and others, intends to launch a major effort to identify the many epigenetic changes that contribute to cancer.

Scientists want to identify the epigenetic changes wrought by specific environmental exposures -- whether dietary factors, environmental toxins or more nuanced influences such as persistent stress -- and develop drugs that can reverse those alterations.

Peter Jones, director of the Norris Comprehensive Cancer Center at the University of Southern California in Los Angeles, likened epigenetic alterations to the clothes worn by genes. "The clothes are really what determine how the genome works," he said. "The clothes can get tattered by these changes, which can lead to different susceptibilities to disease."

The supermice that resist cancer





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The supermice that resist cancer

Scientists have bred a family of "supermice" that are highly resistant to cancer.

The mice have three instead of two copies of genes that keep cell division in check.

It might be possible in the future to make a drug for humans that would confer the same protection against cancer, the Spanish scientists hope.



Supermice were resilient to cancer

The team at the Spanish National Cancer Centre in Madrid report their findings in *Genes and Development*.

Super resistant

Cell growth and division is normally kept under control by a group of gatekeeper genes called tumour suppressors.

In cancer, cells grow out of control and invade, erode and destroy normal tissue.

Dr Manuel Serrano used DNA technology to breed mice that had an extra copy of part of the tumour suppressor genes called Ink4a/ARF locus.

This locus controls the production of two proteins that together appear to stop most human cancer cells developing.

It might be anticipated that an extra working copy would reduce the risk of this disease

These 'supermice' were found to be extra resistant to things known to trigger cancer, called carcinogens, in normal mice.

Hazel Nunn of Cancer Research UK

When the animals were exposed to various carcinogens they developed tumours at a much lower rate than normal.

What's more, the presence of the extra copy of the locus and increased cancer resistance had no apparent effect on the lifespan or fertility of the 'supermice'.

Dr Serrano attributes the increased tumour resistance to the modest increase in the levels of the two proteins gained by the extra copy.

He suspects some humans might inherit extra resistance to certain cancers.

He said it might be possible to induce similar tumour protection in humans using a drug, although this would be some time away.

He said: "This may translate in a big benefit regarding cancer susceptibility."

Hazel Nunn of Cancer Research UK said: "As people who inherit a defective copy of this gene are susceptible to cancer, it might be anticipated that an extra working copy would reduce the risk of this

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disease.

"This is exactly what the research has shown in mice.

"It is very encouraging to have such clear confirmation on which to base future research into how this gene works.

"Ultimately, we hope this knowledge can be developed for the benefit of the cancer patient."

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