Before Big Bang: Light Shed on "Previous Universe"

Mason Inman
for National Geographic News
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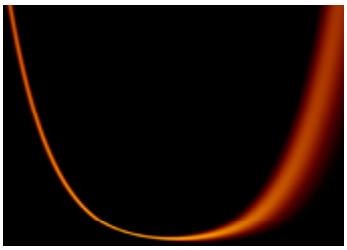
We may be able to get a glimpse of what happened before the big bang, thanks to a new study—but only a glimpse.

The big bang has traditionally been seen as the beginning of everything—space, time, matter, and energy.



But researchers are developing sophisticated new theories to look ever further back in time, to what happened just fractions of a second after big bang itself.

In the new research, Martin Bojowald of Pennsylvania State University pushes one of these theories back even further—to the time of a purported previous universe that contracted and "bounced" to form our own.

The new study comes with some bad news, though.

It suggests that the universe suffers from "cosmic forgetfulness," so that we can never be able to find out too much about what came before our big bang.

The new study appeared online this week in the journal *Nature Physics.*

Theory of Everything

Our universe is expanding outward in every direction, implying that it originally exploded out from a single point about 14 billion years ago.

The further we look back in time, the smaller and hotter the universe gets. At the beginning of time, most traditional theories speculate, the universe was infinitely hot and had no size at all.

But no one knows for sure, since textbook physics suffers a meltdown and gives nonsensical answers when used to describe what the universe was like at moment of the big bang, Bojowald says.

These theories "tell us energies were infinitely large," Bojowald said. "It doesn't have any meaning for us."

This is one reason why researchers have been toiling for decades to unite two main branches of physics—gravity and quantum mechanics.

Gravity rules on cosmic scales, while quantum mechanics dictates the behavior of tiny particles like electrons and quarks.

While each theory has been wildly successful, they remain contradictory.

Uniting these two branches of physics would peel back time further and allow scientists to figure out exactly what the big bang was.

But creating such a "theory of everything" has been a longstanding and difficult goal that has stumped every physicist who has attempted it, including Albert Einstein.

Big Bounce, Not Big Bang

Bojowald used a leading approach to this quandary known as loop quantum gravity, a competitor to the more popular approach known as string theory.

Both theories are still incomplete and unproven, and each suggests very strange ideas about the fundamental nature of the universe.

In loop quantum gravity, for instance, space and time are not smooth and continuous but rather divided up into tiny chunks.

In this mathematical approach, everything is jerky and blocky—although on such a tiny scale that it doesn't affect daily life.

Nothing can occupy a space smaller than the smallest chunk of space, and nothing can happen any faster than this shortest moment of time.

This implies that the universe could never shrink down beyond a certain size. So when it was at its most compact, where did that tiny ball of energy and matter come from?

It could have come from the universe before our own, Bojowald argues. Unlike our expanding universe, this earlier universe was contracting back toward a point, he says.

When it reached its most compact, it hit the barrier dictated by loop quantum gravity. Then it "bounced back" outward, forming a new, expanding universe.

Certain Uncertainty

So if our universe came from an earlier universe, it's natural to wonder what that

ancestral universe was like.

But there's a problem: Quantum physics must have played a key role in the hot, dense state around the time of the "big bounce."

Things behave very oddly in the quantum world. An object that appears to be in one spot when you first glimpse it can be in another spot when you look again.

This jumpiness, known as uncertainty, is built into quantum physics. Building better measuring devices won't get around it.

If the whole universe suffered from these jitters, "it could be impossible to have life," Bojowald said.

In our universe, however, such weirdness only happens on very, very tiny scales.

But what about the universe that came before us?

When the universe goes through a big bounce, Bojowald showed, the amount of uncertainty before and after the bounce have little relation to each other.

So there's a veil that screens out much of what we would want to know about the earlier universe.

This also implies that a universe is never the same before and after a bounce.

"The eternal recurrence of absolutely identical universes would seem to be prevented by the ... cosmic forgetfulness," Bojowald said. (Related: "Universe Reborn Endlessly in New Model of the Cosmos" [April 25, 2002].)

Even that kind of cycle might be coming to an end, since scientists now believe that the universe is expanding faster every day, not slowing down as would be expected. So a re-contraction seems extremely unlikely under our current understanding.

Question of Accuracy

Whether Bojowald's model is believable or not, however, depends on whether the version of loop quantum gravity that he used is accurate.

Thomas Thiemann, of the Perimeter Institute for Theoretical Physics in Waterloo, Canada, called Bojowald's approach a "drastic simplification."

But it may turn out to be fairly accurate anyway, Thiemann said.

If so, then it is "the cleanest derivation of a pre-big bang scenario that any physical theory has delivered so far," he added.

It's "much cleaner than in string-theory-inspired models."

Oldest Known DNA Found in Greenland Ice Core

Mason Inman
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The oldest known strands of DNA have been recovered from frozen mud taken from the base of Greenland's ice sheet, according to a new study.

The discovery could rewrite what was thought about Greenland's ecological past—and could alter current predictions about how global warming will affect the island's ice.

Today most of the Danish-owned island is covered with an ice sheet up to two miles (three kilometers) thick.

But the newfound DNA—genetic material from pine trees, butterflies, and other organisms that lived as much as 800,000 years ago—tells a story of a much greener and vibrant past. Hundreds of thousands of years ago southern Greenland had thriving forests similar to those in northern Canada today, says an international team of 30 scientists led by Eske Willerslev of the University of Copenhagen in Denmark.

The find surpasses the previous record for the oldest DNA, which came from mammoths and other animals frozen in Siberia about 300,000 to 400,000 years ago.

The study also implies that Greenland's ice sheet did not melt as much as computer models have predicted during a period 125,000 years ago, when Earth's sea level rose dramatically.

This raises the question of where the water actually came from.

The research is described in tomorrow's issue of the journal *Science.*

Core Findings

At a site in the center of southern Greenland, researchers drilled through the thick ice until they reached the bottom layer of frozen mud, similar to the permafrost now found

across areas such as Siberia.

Using a variety of techniques, the researchers were able to put an approximate date on the frozen mud: between 450,000 and 800,000 years.

"I'm not super-surprised that DNA could last that long" in Greenland, Willerslev said. "It's pretty much ideal conditions for DNA preservation."

Today it's a frigid -4°F (-20°C) at the bottom of the ice sheet at this spot, about 300 miles (500 kilometers) north of the southern tip of the island.

Researchers have known that Greenland had some trees in the past, since they've found fossilized trees more than two million years old on the north coast of the island.

But it has been difficult to tell what the island's full ecosystem was actually like.

"Ten percent of Earth's surface is covered with ice," Willerslev said. "We have little information about the ecosystems that were there before [these places froze over]."

By comparing the DNA they found to that of today's plants and insects, the team identified the flora and fauna that used to live in this part of Greenland.

They found a variety of trees, including spruce and pine, similar to those found in northern boreal forests across Canada and northern Eurasia.

They also found signs of a number of creatures such as beetles, spiders, and butterflies.

All this suggests that before the area froze over, it had an open forest that supported a diverse ecosystem.

Mystery Melt

While these DNA discoveries help answer questions about Greenland's past environment, they may have created a new mystery related to global warming.

Based on computer models, some researchers had thought that most of southern Greenland was ice-free about 125,000 years ago, during the last interglacial period—a gap in time between ice ages when Earth was warmer.

During this period, sea levels rose about 16 to 19 feet (5 to 6 meters).

Many researchers thought that much of this water came from the melting of the southern part of Greenland's ice sheet during this time.

Several studies have found that Greenland's ice sheet is melting today, so scientists are

working to predict how much the melt might contribute to sea level rise if the world continues warming.

But "then we started to date [the ice] and we found that it wasn't that young," Willerslev said.

If the island had been free of ice during the last interglacial, it would likely have supported plants and animals.

In that case, DNA from those creatures should have been found instead of the DNA from the much older forest.

The new discoveries suggest that southern Greenland has been ice-covered for at least four times longer than previously thought.

"We have firm data to state that there was ice in central and northern Greenland," said Valerie Masson-Delmotte of the Laboratory of Climate and Environmental Sciences in Saclay, France.

"The main surprise [of the new study] is the persistency of ice in southern Greenland," said Masson-Delmotte, who was not involved in the new study.

And if the region was still frozen during the last interglacial period, lead author Willerslev said, then some of that water "must have come from some melting of ice somewhere else."

How We Get Addicted

Thursday, Jul. 05, 2007 By MICHAEL D. LEMONICK

I was driving up the Massachusetts Turnpike one evening last February when I knocked over a bottle of water. I grabbed for it, swerved inadvertently--and a few seconds later

found myself blinking into the flashlight beam of a state trooper. "How much have you had to drink tonight, sir?" he demanded. Before I could help myself, I blurted out an answer that was surely a new one to him. "I haven't had a drink," I said indignantly, "since 1981."

It was both perfectly true and very pertinent to the trip I was making. By the time I reached my late 20s, I'd poured down as much alcohol as normal people consume in a lifetime and plenty of drugs--mostly pot--as well. I was, by any reasonable measure, an active alcoholic. Fortunately, with a lot of help, I was able to stop. And now I was on my way to McLean Hospital in Belmont, Mass., to have my brain scanned in a functional magnetic-resonance imager (fMRI). The idea was to see what the inside of my head looked like after more than a quarter-century on the wagon.

Back when I stopped drinking, such an experiment would have been unimaginable. At the time, the medical establishment had come to accept the idea that alcoholism was a disease rather than a moral failing; the American Medical Association (AMA) had said so in 1950. But while it had all the hallmarks of other diseases, including specific symptoms and a predictable course, leading to disability or even death, alcoholism was different. Its physical basis was a complete mystery--and since nobody forced alcoholics to drink, it was still seen, no matter what the AMA said, as somehow voluntary. Treatment consisted mostly of talk therapy, maybe some vitamins and usually a strong recommendation to join Alcoholics Anonymous. Although it's a totally nonprofessional organization, founded in 1935 by an ex-drunk and an active drinker, AA has managed to get millions of people off the bottle, using group support and a program of accumulated folk wisdom.

While AA is astonishingly effective for some people, it doesn't work for everyone; studies suggest it succeeds about 20% of the time, and other forms of treatment, including various types of behavioral therapy, do no better. The rate is much the same with drug addiction, which experts see as the same disorder triggered by a different chemical. "The sad part is that if you look at where addiction treatment was 10 years ago, it hasn't gotten much better," says Dr. Martin Paulus, a professor of psychiatry at the University of California at San Diego. "You have a better chance to do well after many types of cancer than you have of recovering from methamphetamine dependence."

That could all be about to change. During those same 10 years, researchers have made extraordinary progress in understanding the physical basis of addiction. They know now, for example, that the 20% success rate can shoot up to 40% if treatment is ongoing (very much the AA model, which is most effective when members continue to attend meetings long after their last drink). Armed with an array of increasingly sophisticated technology, including fMRIs and PET scans, investigators have begun to figure out exactly what goes wrong in the brain of an addict--which neurotransmitting chemicals

are out of balance and what regions of the brain are affected. They are developing a more detailed understanding of how deeply and completely addiction can affect the brain, by hijacking memory-making processes and by exploiting emotions. Using that knowledge, they've begun to design new drugs that are showing promise in cutting off the craving that drives an addict irresistibly toward relapse--the greatest risk facing even the most dedicated abstainer.

"Addictions," says Joseph Frascella, director of the division of clinical neuroscience at the National Institute on Drug Abuse (NIDA), "are repetitive behaviors in the face of negative consequences, the desire to continue something you know is bad for you." Addiction is such a harmful behavior, in fact, that evolution should have long ago weeded it out of the population: if it's hard to drive safely under the influence, imagine trying to run from a saber-toothed tiger or catch a squirrel for lunch. And yet, says Dr. Nora Volkow, director of NIDA and a pioneer in the use of imaging to understand addiction, "the use of drugs has been recorded since the beginning of civilization. Humans in my view will always want to experiment with things to make them feel good."

That's because drugs of abuse co-opt the very brain functions that allowed our distant ancestors to survive in a hostile world. Our minds are programmed to pay extra attention to what neurologists call salience--that is, special relevance. Threats, for example, are highly salient, which is why we instinctively try to get away from them. But so are food and sex because they help the individual and the species survive. Drugs of abuse capitalize on this ready-made programming. When exposed to drugs, our memory systems, reward circuits, decision-making skills and conditioning kick in--salience in overdrive--to create an all consuming pattern of uncontrollable craving. "Some people have a genetic predisposition to addiction," says Volkow. "But because it involves these basic brain functions, everyone will become an addict if sufficiently exposed to drugs or alcohol."

That can go for nonchemical addictions as well. Behaviors, from gambling to shopping to sex, may start out as habits but slide into addictions. Sometimes there might be a behavior-specific root of the problem. Volkow's research group, for example, has shown that pathologically obese people who are compulsive eaters exhibit hyperactivity in the areas of the brain that process food stimuli--including the mouth, lips and tongue. For them, activating these regions is like opening the floodgates to the pleasure center. Almost anything deeply enjoyable can turn into an addiction, though.

Of course, not everyone becomes an addict. That's because we have other, more analytical regions that can evaluate consequences and override mere pleasure seeking. Brain imaging is showing exactly how that happens. Paulus, for example, looked at methamphetamine addicts enrolled in a VA hospital's intensive four-week rehabilitation

program. Those who were more likely to relapse in the first year after completing the program were also less able to complete tasks involving cognitive skills and less able to adjust to new rules quickly. This suggested that those patients might also be less adept at using analytical areas of the brain while performing decision-making tasks. Sure enough, brain scans showed that there were reduced levels of activation in the prefrontal cortex, where rational thought can override impulsive behavior. It's impossible to say if the drugs might have damaged these abilities in the relapsers--an effect rather than a cause of the chemical abuse--but the fact that the cognitive deficit existed in only some of the meth users suggests that there was something innate that was unique to them. To his surprise, Paulus found that 80% to 90% of the time, he could accurately predict who would relapse within a year simply by examining the scans.

Another area of focus for researchers involves the brain's reward system, powered largely by the neurotransmitter dopamine. Investigators are looking specifically at the family of dopamine receptors that populate nerve cells and bind to the compound. The hope is that if you can dampen the effect of the brain chemical that carries the pleasurable signal, you can loosen the drug's hold.

One particular group of dopamine receptors, for example, called D3, seems to multiply in the presence of cocaine, methamphetamine and nicotine, making it possible for more of the drug to enter and activate nerve cells. "Receptor density is thought to be an amplifier," says Frank Vocci, director of pharmacotherapies at NIDA. "[Chemically] blocking D3 interrupts an awful lot of the drugs' effects. It is probably the hottest target in modulating the reward system."

But just as there are two ways to stop a speeding car--by easing off the gas or hitting the brake pedal--there are two different possibilities for muting addiction. If dopamine receptors are the gas, the brain's own inhibitory systems act as the brakes. In addicts, this natural damping circuit, called GABA (gamma-aminobutyric acid), appears to be faulty. Without a proper chemical check on excitatory messages set off by drugs, the brain never appreciates that it's been satiated.

As it turns out, vigabatrin, an antiepilepsy treatment that is marketed in 60 countries (but not yet in the U.S.), is an effective GABA booster. In epileptics, vigabatrin suppresses overactivated motor neurons that cause muscles to contract and go into spasm. Hoping that enhancing GABA in the brains of addicts could help them control their drug cravings, two biotech companies in the U.S., Ovation Pharmaceuticals and Catalyst Pharmaceuticals, are studying the drug's effect on methamphetamine and cocaine use. So far, in animals, vigabatrin prevents the breakdown of GABA so that more of the inhibitory compound can be stored in whole form in nerve cells. That way, more of it could be released when those cells are activated by a hit from a drug. Says Vocci, optimistically: "If it works, it will probably work on all addictions."

Another fundamental target for addiction treatments is the stress network. Animal studies have long shown that stress can increase the desire for drugs. In rats trained to self-administer a substance, stressors such as a new environment, an unfamiliar cage mate or a change in daily routine push the animals to depend on the substance even more.

Among higher creatures like us, stress can also alter the way the brain thinks, particularly the way it contemplates the consequences of actions. Recall the last time you found yourself in a stressful situation--when you were scared, nervous or threatened. Your brain tuned out everything besides whatever it was that was frightening you--the familiar fight-or-flight mode. "The part of the prefrontal cortex that is involved in deliberative cognition is shut down by stress," says Vocci. "It's supposed to be, but it's even more inhibited in substance abusers." A less responsive prefrontal cortex sets up addicts to be more impulsive as well.

Hormones--of the male-female kind--may play a role in how people become addicted as well. Studies have shown, for instance, that women may be more vulnerable to cravings for nicotine during the latter part of the menstrual cycle, when the egg emerges from the follicle and the hormones progesterone and estrogen are released. "The reward systems of the brain have different sensitivities at different points in the cycle," notes Volkow. "There is way greater craving during the later phase."

That led researchers to wonder about other biological differences in the way men and women become addicted and, significantly, respond to treatments. Alcohol dependence is one very promising area. For years, researchers had documented the way female alcoholics tend to progress more rapidly to alcoholism than men. This telescoping effect, they now know, has a lot to do with the way women metabolize alcohol. Females are endowed with less alcohol dehydrogenase--the first enzyme in the stomach lining that starts to break down the ethanol in liquor--and less total body water than men. Together with estrogen, these factors have a net concentrating effect on the alcohol in the blood, giving women a more intense hit with each drink. The pleasure from that extreme high may be enough for some women to feel satisfied and therefore drink less. For others, the intense intoxication is so enjoyable that they try to duplicate the experience over and over.

But it's the brain, not the gut, that continues to get most of the attention, and one of the biggest reasons is technology. It was in 1985 that Volkow first began using PET scans to record trademark characteristics in the brains and nerve cells of chronic drug abusers, including blood flow, dopamine levels and glucose metabolism--a measure of how much energy is being used and where (and therefore a stand-in for figuring out which cells are at work). After the subjects had been abstinent a year, Volkow rescanned their brains and found that they had begun to return to their predrug state. Good news, certainly,

but only as far as it goes.

"The changes induced by addiction do not just involve one system," says Volkow. "There are some areas in which the changes persist even after two years." One area of delayed rebound involves learning. Somehow in methamphetamine abusers, the ability to learn some new things remained affected after 14 months of abstinence. "Does treatment push the brain back to normal," asks NIDA's Frascella, "or does it push it back in different ways?"

If the kind of damage that lingers in an addict's learning abilities also hangs on in behavioral areas, this could explain why rehabilitation programs that rely on cognitive therapy--teaching new ways to think about the need for a substance and the consequences of using it--may not always be effective, especially in the first weeks and months after getting clean. "Therapy is a learning process," notes Vocci. "We are trying to get [addicts] to change cognition and behavior at a time when they are least able to do so."

One important discovery: evidence is building to support the 90-day rehabilitation model, which was stumbled upon by AA (new members are advised to attend a meeting a day for the first 90 days) and is the duration of a typical stint in a drug-treatment program. It turns out that this is just about how long it takes for the brain to reset itself and shake off the immediate influence of a drug. Researchers at Yale University have documented what they call the sleeper effect--a gradual re-engaging of proper decision making and analytical functions in the brain's prefrontal cortex--after an addict has abstained for at least 90 days.

This work has led to research on cognitive enhancers, or compounds that may amplify connections in the prefrontal cortex to speed up the natural reversal. Such enhancement would give the higher regions of the brain a fighting chance against the amygdala, a more basal region that plays a role in priming the dopamine-reward system when certain cues suggest imminent pleasure--anything from the sight of white powder that looks like cocaine to spending time with friends you used to drink with. It's that conditioned reflex--identical to the one that caused Ivan Pavlov's famed dog to salivate at the ringing of a bell after it learned to associate the sound with food--that unleashes a craving. And it's that phenomenon that was the purpose of my brain scans at McLean, one of the world's premier centers for addiction research.

In my heyday, I would often drink even when I knew it was a terrible idea--and the urge was hardest to resist when I was with my drinking buddies, hearing the clink of glasses and bottles, seeing others imbibe and smelling the aroma of wine or beer. The researchers at McLean have invented a machine that wafts such odors directly into the nostrils of a subject undergoing an fMRI scan in order to see how the brain reacts. The reward circuitry in the brain of a newly recovering alcoholic should light up like a

Christmas tree when stimulated by one of these alluring smells.

I chose dark beer, my absolute favorite, from their impressive stock. But I haven't gotten high for more than a quarter-century; it was an open question whether I would react that way. So after an interview with a staff psychiatrist to make sure I would be able to handle it if I experienced a craving, I was fitted with a tube that carried beer aroma from a vaporizer into my nose. I was then slid into the machine to inhale that still familiar odor while the fMRI did its work.

Even if the smells triggered a strong desire to drink, I had long since learned ways to talk myself out of it--or find someone to help me do so. Like the 90-day drying-out period that turns out to parallel the brain's recovery cycle, such a strategy is in line with other new theories of addiction. Scientists say extinguishing urges is not a matter of getting the feelings to fade but of helping the addict learn a new form of conditioning, one that allows the brain's cognitive power to shout down the amygdala and other lower regions. "What has to happen for that cue to extinguish is not for the amygdala to become weaker but for the frontal cortex to become stronger," says Vocci.

While such relearning has not been studied formally in humans, Vocci believes it will work, on the basis of studies involving, of all things, phobias. It turns out that phobias and drugs exploit the same struggle between high and low circuits in the brain. People placed in a virtual-reality glass elevator and treated with the antibiotic D-cycloserine were better able to overcome their fear of heights than those without benefit of the drug. Says Vocci: "I never thought we would have drugs that affect cognition in such a specific way."

Such surprises have even allowed experts to speculate whether addiction can ever be cured. That notion goes firmly against current beliefs. A rehabilitated addict is always in recovery because cured suggests that resuming drinking or smoking or shooting up is a safe possibility--whose downside could be devastating. But there are hints that a cure might not in principle be impossible. A recent study showed that tobacco smokers who suffered a stroke that damaged the insula (a region of the brain involved in emotional, gut-instinct perceptions) no longer felt a desire for nicotine.

That's exciting, but because the insula is so critical to other brain functions--perceiving danger, anticipating threats--damaging this area isn't something you would ever want to do intentionally. With so many of the brain's systems entangled with one another, it could prove impossible to adjust just one without throwing the others into imbalance. Nevertheless, says Volkow, "addiction is a medical condition. We have to recognize that medications can reverse the pathology of the disease. We have to force ourselves to think about a cure because if we don't, it will never happen." Still, she is quick to admit that just contemplating new ideas doesn't make them so. The brain functions that addiction commandeers may simply be so complex that sufferers, as 12-step recovery programs

have emphasized for decades, never lose their vulnerability to their drug of choice, no matter how healthy their brains might eventually look.

I'm probably a case in point. My brain barely lit up in response to the smell of beer inside the fMRI at McLean. "This is actually valuable information for you as an individual," said Scott Lukas, director of the hospital's behavioral psychopharmacology research laboratory and a professor at Harvard Medical School who ran the tests. "It means that your brain's sensitivity to beer cues has long passed."

That's in keeping with my real-world experience; if someone has a beer at dinner, I don't feel a compulsion to leap across the table and grab it or even to order one for myself. Does that mean I'm cured? Maybe. But it may also mean simply that it would take a much stronger trigger for me to fall prey to addiction again--like, for example, downing a glass of beer. But the last thing I intend to do is put it to the test. I've seen too many others try it--with horrifying results.