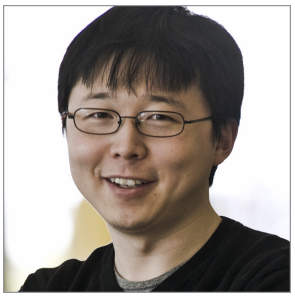

FENG ZHANG is a core institute member of the Broad Institute of MIT and Harvard, as well as an investigator at the McGovern Institute for Brain Research at MIT and an associate professor at MIT with a joint appointment in the Departments of Brain and Cognitive Sciences and Biological Engineering.

Feng Zhang, Ph.D.

Core Institute Member at the Broad Institute of MIT and Harvard

Associate Professor in the Department of Brain and Cognitive Sciences and Department of Biological Engineering at MIT

Investigator at the McGovern Institute for Brain Research at MIT

Zhang is a bioengineer focused on developing tools to better understand nervous system function and disease. His lab applies these novel tools to interrogate gene function and study neuropsychiatric diseases in animal and stem cell models. Since joining the Broad Institute and McGovern in January 2011, Zhang has pioneered the development of genome editing tools for use in eukaryotic cells – including human cells – from natural microbial CRISPR systems. These tools, which he has made widely available, are accelerating biomedical research around the world.

Zhang leverages CRISPR and other methodologies to study the role of genetic and epigenetic mechanisms underlying diseases, specifically focusing on disorders of the nervous system. He is especially interested in complex disorders, such as psychiatric and neurological diseases, that are caused by multiple genetic and environmental risk factors and which are difficult to model using conventional methods. Zhang's methods are also being used in the fields of immunology, clinical medicine, cancer biology, and other areas of research. Zhang's long-term goal is to develop novel therapeutic strategies for disease treatment.

Zhang's work on genome editing traces back to his seminal paper, published in January 2011, reporting the first systematic approach of an earlier system, using TALEs, to target specific genes in mammalian cells.

Soon after joining the Broad Institute and MIT, in early 2011 Zhang turned his attention to the CRISPR-Cas system – which researchers in Canada had just demonstrated as being able to create double-stranded breaks in target DNA at precise positions – as a potential tool for improved genome editing. On October 5, 2012, Zhang submitted a breakthrough paper that reported the first successful programmable genome editing of mammalian cells using CRISPR-Cas9 (Cong et al., *Science* 2013). Cong et al. remains the most-cited paper in genome editing.

Since then, Zhang's technique for mammalian genome editing has had enormous impact on experimental science and holds great promise for therapeutic applications. His lab continues to refine and improve upon the

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CRISPR system and to develop novel genome-engineering technologies aimed at perturbing and editing the genome for disease research. In 2015, Zhang and colleagues successfully harnessed a second system, called CRISPR-Cpf1, which has the potential for even simpler and more precise genome engineering.

Zhang and the Broad Institute are committed to making CRISPR technology widely available to the entire scientific community. The Zhang lab has trained thousands of researchers in the use of CRISPR-Cas9 genome editing technology through direct education and by sharing approximately 25,000 CRISPR-Cas9 components with academic laboratories in the U.S. and around the world to accelerate research that will benefit human health.

Although Zhang is well-known for his pioneering work on CRISPR, he is also widely recognized for developing another breakthrough technology called optogenetics (Zhang et al., *Nature Protocols* 2010) with Karl Deisseroth at Stanford University and Edward Boyden, now of MIT. *Nature Methods* named optogenetics its 2010 Method of the Year.

Zhang demonstrated the utility of optogenetics, in which neuronal activity can be controlled with light, by studying neural circuits in the brain. Zhang later served as a Junior Fellow at Harvard's Society of Fellows and collaborated with Paola Arlotta and George Church on using synthetic biology to study the patterns of gene activity during brain development, a topic with implications for neurological and psychiatric problems.

Zhang is a recipient of many awards including the Perl/UNC Prize in Neuroscience (2012, shared with Deisseroth and Boyden), the NIH Director's Pioneer Award (2012), the National Science Foundation's Alan T. Waterman Award (2014), the Jacob Heskel Gabbay Award in Biotechnology and Medicine (2014, shared with Jennifer Doudna and Emmanuelle Charpentier), and the Society for Neuroscience Young Investigator Award (2014, shared with Diana Bautista). He has also received technology innovation awards from the McKnight, New York Stem Cell, and Damon Runyon foundations. In 2013 Popular Science named him one of its "Brilliant Ten." He was named one of the "Top 20 Researchers in 2014" by *Nature Biotechnology*, which considers the total impact of a scientist's body of published work.

News coverage has credited Zhang with turning science fiction into "science fact," and he has been called the "Midas of Methods" for his breakthrough work on genome engineering using CRISPR-Cas9 and TALEs.

Zhang is a founder of Editas Medicine, a genome editing company founded by world leaders in the fields of genome editing, protein engineering, and molecular and structural biology, with specific expertise in CRISPR and TALE technologies.

Zhang grew up in Iowa after moving there with his parents from China at age 11. In high school he spent afternoons working at the Human Gene Therapy Research Institute in Des Moines as part of an academic program. Zhang received his A.B. in chemistry and physics from Harvard College and his Ph.D. in chemistry from Stanford University.

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365 days: *Nature's* 10

Ten people who mattered this year.

18 December 2013

FENG ZHANG: DNA's master editor

Borrowing from bacteria, a biologist helps to create a powerful tool for customizing DNA.

By Daniel Cressey

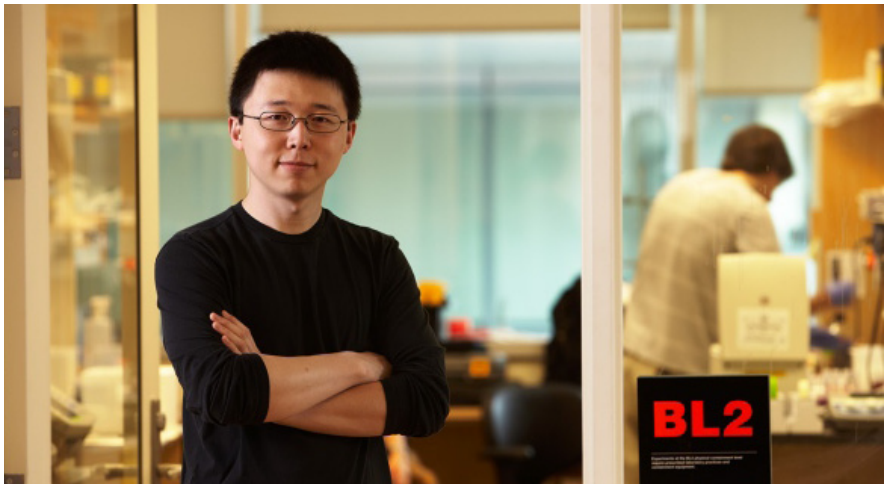
With a nip here and a tuck there, a DNA-cutting mechanism that bacteria use to protect themselves from viruses became one of the hottest topics in biomedical research in 2013. And a young neuroscientist with a penchant for developing tools helped to make it happen.

Thirty-two-year-old Feng Zhang of the Massachusetts Institute of Technology in Cambridge is among those leading the charge in using a system called CRISPR/Cas to edit genomes cheaply, easily and precisely. In January, his group showed that the system works in eukaryotic cells — ones with membrane-bound nuclei, including those of all animals and plants. This confirmed its potential for tweaking the genomes of mice, rats and even primates to aid research, improve human-disease modelling and develop treatments (L. Cong et al. *Science* 339, 819–823; 2013).

But as hot as the story has been this year, "the CRISPR craze is likely just starting", says Rodolphe Barrangou, a microbiologist at North Carolina State University in Raleigh.

CRISPRs (clustered regularly interspaced palindromic repeats) are DNA sequences that many bacteria and archaea use to defend themselves. They encode RNAs that can specifically recognize a target sequence in a viral genome. The RNAs work in complex with a CRISPR-associated protein, or Cas, which snips the DNA of the invader.

In 2012, Jennifer Doudna of the University of California, Berkeley, Emmanuelle Charpentier, now at the Helmholtz Centre for Infection Research in Braunschweig, Germany, and colleagues showed that they could reprogram a CRISPR system to cut apart potentially any specific DNA target



Kent Dayton

(M. Jinek et al. *Science* 337, 816–821; 2012). By controlling how the break is repaired, they can edit a gene — adding, switching or removing parts to change the protein it encodes or disable it altogether.

CRISPR is similar to two earlier genome-editing methods: the zinc-finger nuclease (ZFN) and transcription activator-like effector nuclease (TALEN) systems. But both of those locate target sequences using proteins that are often difficult and costly to produce. CRISPRs use RNA, making them easier to design. Zhang says he feels limited only "by what I can imagine is possible".

Although Charpentier and Doudna are generally credited with kick-starting the growth of CRISPR editing, Zhang demonstrated its vast potential by showing that it works in eukaryotes, a finding independently confirmed by George Church at Harvard Medical School in Boston, Massachusetts (P. Mali et al. *Science* 339, 823–826; 2013). Zhang says that he had a head start on many of the teams who jumped in: he had been investigating the technique before it was widely reported, and because his lab had previously fine-tuned ZFNs and TALENs to edit DNA, it had procedures in place for perfecting CRISPRs.

Zhang now says that he feels challenged to be creative with other applications. One particularly ambitious project on his slate is to build a library of CRISPRs that can delete any sequence in an organism's entire genome in 100–200 base-pair increments. This could make it easier to investigate the function of non-coding DNA.

But he is most interested in using CRISPR to treat neuropsychiatric conditions such as Huntington's disease and schizophrenia by repairing genes in human tissues. To pursue therapeutic use of the technology, he and other CRISPR pioneers last month launched a company called Editas Medicine, based in Cambridge, that is backed by US$43 million in venture-capital funding. CRISPR "allows us to start to make corrections in the genome", says Zhang. "Because it's easy to program, it will open up the door to addressing mutations that affect few people but are very devastating."

POPULAR SCIENCE THE FUTURE NOW

How Feng Zhang Modified A Cell's Genome On The Fly

His techniques could be used to study the genetics of autism and schizophrenia.

By Veronique Greenwood and Valerie Ross Posted 10.23.2013 at 9:00 am

Feng Zhang *Joel Kimmel*

Each year, Popular Science *seeks out the brightest young scientists and engineers and names them the Brilliant Ten. Like the 110 honorees before them, the members of this year's class are dramatically reshaping their fields--and the future. Some are tackling pragmatic questions, like how to secure the Internet, while others are attacking more abstract ones, like determining the weather on distant exoplanets. The common thread between them is brilliance, of course, but also impact. If the Brilliant Ten are the faces of things to come, the world will be a safer, smarter, and brighter place.--The Editors*

Feng Zhang

Massachusetts Institute of Technology and Broad Institute

Achievement

Modifying a cell's genome on the fly

When Feng Zhang was in graduate school, he discovered that the tools for splicing new genes into living cells were costly, time-consuming, and proprietary. Unhappy with that reality, he did what any enterprising open-source enthusiast would do—he made his own tools and shared them with other scientists. They dramatically sped up the study of genetics and disease.

The techniques Zhang helped develop, called TALE and CRISPR, create transgenic or otherwise genetically modified organisms with unprecedented efficiency. TALE is a molecule that gloms onto a section of DNA and affects whether a nearby gene is turned on or off. CRISPR is based on a microbial enzyme that snips the DNA to introduce new genetic material. Using these methods, Zhang can make a transgenic mouse in three weeks (normal methods require more than six months to achieve that feat). Almost 2,000 labs have requested information about CRISPR alone since it was first cited in a publication in January 2013. "These technologies are so fundamental, it's best to keep them as open as possible," Zhang says. "If someone had protected the HTML language for making Web pages, then we wouldn't have the World Wide Web."

Zhang plans to use the techniques to study the genetics of autism and schizophrenia. He has already begun to insert genes linked to each disorder one by one into animal models to observe their effects. Now that he has the tools, he says, the rest of his work can begin.

Five Questions for Feng Zhang

By Veronica Meade-Kelly, Broad Communications

September 17, 2013

Core faculty member Feng Zhang, who joined the Broad in 2011, has quickly earned a reputation as one of the brightest young scientists working today. His research on optogenetics and genome engineering earned him a spot in this year's "Brilliant 10," *Popular Science* magazine's annual list of the most promising scientific innovators. He was also recognized last month by *MIT Technology Review*, which named him as one of their "35 Innovators Under 35" for 2013.

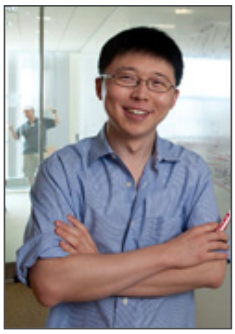


Photo by Len Rubenstein

In this edition of "Five Questions," Zhang, who is also an assistant professor at MIT and a principal investigator at the McGovern Institute for Brain Research at MIT, talks about his research, the excitement it has generated, and where he hopes his work may lead him.

Q1: Your lab is remarkably prolific. What is the primary focus of your research?

FZ: We're looking at how the cells' molecular machinery works and, to do that, we need new tools that allow us to have precise control over the biological activities happening inside the cell. My lab mainly focuses on developing tools that can help us understand how the genome works and how it influences the activity of brain cells to help organisms carry out complex functions. We are also looking at what goes wrong in these systems to cause disease.

Q2: Can you describe the different lines of research that your lab pursues, and how they relate to each other?

FZ: Understanding how the brain works is one of my main interests. Two different lines of research – optogenetics and genome engineering – are helping my lab do that.

Optogenetics allows us to use light to control specific neurons, and thus control when and how information is sent from one brain cell to another. It's really unique in the sense that you can specifically control the activity of a subset of cells in the brain without affecting nearby cells. This allows you to send information to the brain and see how it gets processed, so you can start to understand signaling in the brain.

Genome engineering looks at the brain from a different perspective. Using genome engineering or genome editing, we can change the actual DNA letters in the genome to understand which genes are involved in what function, and which genetic mutations are involved in a specific disease.

Q3: You were the senior author of a paper that came out just last week in *Cell*. Can you tell us a little bit about it?

FZ: The *Cell* paper builds on previous work that we did developing a new genome engineering system called **CRISPR**. One of the applications of CRISPR is to make very precise changes in the genome so that you can ask questions like, "What does this specific difference in the DNA sequence do to the biology of the cell?" We developed CRISPR over the past two and half years, but recently we came to realize that it would sometimes make imprecise, off-target modifications in the genome, and that's a problem if our whole premise is that we are making precise changes to the genetic code without changing anything else in the cell. The paper in *Cell* describes a new approach that we found that makes editing much more precise; we show that we can overcome the off-target activity that we saw before.

Q4: What is the ultimate goal of your research?

FZ: It's twofold: many of the diagnoses in medicine today are based on observation of symptoms. We don't necessarily know what the cause of the disease or symptom is, so we just treat the symptom. One of my long-term goals is to make disease diagnosis more scientific by using genome editing to identify and understand the roles that genetic mutations play in disease. A second, more distant goal is to use genome editing to correct the genetic mutations that lead to disease. To do that in a therapeutic context we will have to be very precise; we don't want to mutate other genes that are not involved in the disease. The work we did for the *Cell* paper takes us one step closer to reaching that therapeutic potential.

Q5: Your research has gotten a lot of attention over the last couple of years. What is it about your work that fascinates people?

FZ: I think people are excited for a few reasons. First, this technology allows many researchers to do what they've wanted to do for a long time: to manipulate the genome precisely. In the past, that could only be done in yeast, and to some extent in mice. Making genetic changes – however you want, in any organism you want – was not possible until about a year ago. I think that's why people are excited – it opens up the door to studying more interesting biology in a wider variety of organisms. The second reason that I think people are excited is that this technology makes it really easy to work on things. Before, it would take a year or two to make a transgenic mouse; now they can do it in a few weeks. That ease of access is rapidly opening up new avenues for research.

Paper cited:

Ran, FA et al., "Double Nicking by RNA-Guided CRISPR Cas9 for Enhanced Genome Editing Specificity." *Cell* DOI: 10.1016/j.cell.2013.08.021.

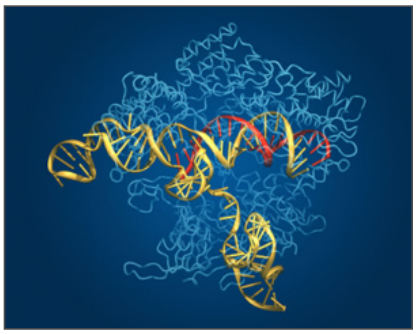
BROAD INSTITUTE

Broad, MIT researchers reveal structure of key CRISPR complex

February 13, 2014

By Veronica Meade-Kelly, Broad Communications

Researchers from the Broad Institute and MIT have teamed up with colleagues from the University of Tokyo to form the first high definition picture of the Cas9 complex – a key part of the CRISPR-Cas system used by scientists as a genome-editing tool to silence genes and probe the biology of cells. Their findings, which are reported this week in *Cell*, are expected to help researchers refine and further engineer the tool to accelerate genomic research and bring the technology closer to use in the treatment of human genetic disease.



This image of the Cas9 complex depicts the Cas9 protein (in light blue), along with its guide RNA (yellow), and target DNA (red). Image courtesy of Bang Wong, from source material provided by Feng Zhang

First discovered in bacteria in 1987, CRISPRs (Clustered Regularly Interspaced Short Palindromic Repeats) have recently been harnessed as so-called genome editing tools. These tools allow researchers to home in on "typos" within the three-billion-letter sequence of the human genome, and cut out and even alter the problematic sequence. The Cas9 complex, which includes the CRISPR "cleaving" enzyme Cas9 and an RNA "guide" that leads the enzyme to its DNA target, is key to this process.

"We've come to view the Cas9 complex as the ultimate guided missile that we can use to target precise sites in the genome," said co-senior author Feng Zhang, a core member of the Broad Institute, an investigator at the McGovern Institute for Brain Research, and an assistant professor at MIT. "This study provides a schematic of the entire system – it shows the missile (the Cas9 protein), the programming instructions (the guide RNA) that send it to the right location, and the target DNA. It also reveals the secret of how these pieces function together to make the whole system work."

To deconstruct this system, Zhang approached the paper's co-senior author Osamu Nureki at the University of Tokyo. Together, they assembled a team to work out the complicated structure.

"Cas9-based genome editing technologies are proving to be revolutionary in a wide range of life sciences, enabling many new experimental techniques, so my colleagues and I were excited to work with Feng's lab on this important research," said first author Hiroshi Nishimasu, an assistant professor of biophysics and biochemistry who works in Nureki's lab at the University of Tokyo.

The two teams worked closely to reveal the structural details of the Cas9 complex and to test their functional significance. Their efforts revealed a division of labor within the Cas9 complex. The researchers determined that the Cas9 protein consists of two lobes: one lobe is involved in the recognition of the RNA and DNA elements, while the other lobe is responsible for cleaving the target DNA, causing what is known as a "double strand break" that disables the targeted gene. The team also found that key structures on Cas9 interface with the guide RNA, allowing Cas9 to organize itself around the RNA and the target DNA as it prepares to cut the strands.

Identifying the key features of the Cas9 complex should enable researchers to improve the genome-editing tool to better suit their needs.

"Up until now, it has been very difficult to rationally engineer Cas9. Now that we have this structural information, we can take a principled approach to engineering the protein to make it more effective," said Zhang, who is also a co-founder of Editas Medicine, a company that was started last year to develop Cas9 and other genome editing technologies into a novel class of human therapeutics.

Currently, Cas9 is used in experiments to silence genes in mammalian cells – sometimes at multiple sites across the genome – and large libraries of RNA sequences have been created to guide Cas9 to genes of interest. However, the system can only target specific types of sites. Some studies have also shown that the RNA could lead Cas9 "off-target," potentially causing unexpected problems within the cellular machinery.

The researchers plan to use this new, detailed picture of the Cas9 complex to address these concerns.

"Understanding this structure may help us engineer around the current limitations of the Cas9 complex," said study author F. Ann Ran, a graduate student in Zhang's lab. "In the future, it could allow us to design versions of these editing tools that are more specific to our research needs. We may even be able to alter the type of nucleic acid sequences that Cas9 can target."

Such technological improvements will be needed if the CRISPR-Cas system is to evolve into a therapeutic tool for the treatment of genetic disease.

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The study was supported by the National Institute of Mental Health (NIMH); an NIH Director's Pioneer Award; the Japan Science and Technology Agency; the Japan Society for the Promotion of Science; the Keck, McKnight, Poitras, Merkin, Vallee, Damon Runyon, Searle Scholars, Klingenstein, and Simons Foundations; as well as Bob Metcalfe and Jane Pauley.

Other researchers who worked on the study include Patrick D. Hsu, Silvana Konermann, Soraya Shehata, Naoshi Dohmae, and Ryuichiro Ishitani.

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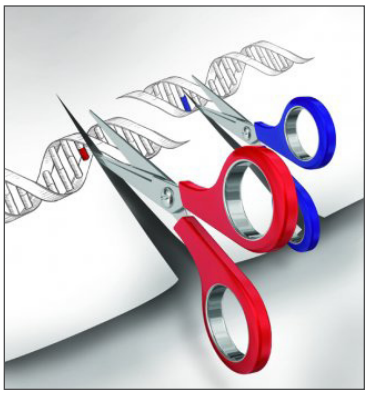
BROAD INSTITUTE

CRISPR system scales up in human cells

By Haley Bridger, Broad Communications

December 12, 2013

For decades, researchers have sought a biological toolset capable of precisely and systematically turning off genes throughout the genomes of human cells. The CRISPR-Cas9 system – a recently discovered system with bacterial origins – has the potential to overcome many of the limitations of currently available gene-silencing techniques. Earlier this year, several research groups showed that it was possible to use CRISPR-Cas9 to turn off genes in mammalian cells.



The CRISPR-Cas9 system causes a precise double strand break in DNA, which leads to a gene being turned off. Researchers have scaled up to turn off genes accurately and efficiently at a genomic scale instead of just one or a few genes at a time.

Composite image by Lauren Solomon, Broad Communications. Scissors © iStockphoto.com/ewg3D. Double helix © Can Stock Photo Inc./lhfgraphics.

But in order to investigate and better understand the genetics of health and disease, scientists need a toolset that can reliably turn off each of the genes in the genome on a large scale.

In companion papers published together this week in *Science*, researchers from the Broad Institute, Whitehead Institute, McGovern Institute for Brain Research, and elsewhere have scaled up, demonstrating the capabilities of the CRISPR-Cas9 system in large-scale studies of several types of human cells, turning off genes accurately and efficiently at a genomic scale instead of just one or a few genes at a time. The two papers – which offer up libraries of tens of thousands of complexes tailored to match precise locations throughout the genome – lay the groundwork for a range of future studies, including investigations into neural development, cancer, and many other human diseases.

"We can now use this technology on a genome-wide scale, giving us the ability to interrogate any gene we want," said Feng Zhang, a core member of the Broad Institute, an investigator at the McGovern Institute, and an assistant professor at MIT. Zhang is also the senior author of one of the *Science* papers. "Additionally, we demonstrate the utility of CRISPR for making biological discoveries, identifying new genes likely involved in how cancer cells become resistant to treatment. This is likely the first biological discovery made using CRISPR."

Unlike other gene-silencing tools, the CRISPR-Cas9 system targets the genome's source material: while RNA interference (RNAi) must target many copies of messenger RNA in order to tamp down a gene's expression, CRISPR-Cas9 permanently turns off genes at the DNA level. The CRISPR-Cas9 system includes an enzyme that makes a cut in DNA, and is paired with a single guide RNA (sgRNA), which researchers construct to home in on a specific site in the genome. The DNA cut – known as a double strand break – closely mimics the kinds of mutations that occur naturally, for instance after chronic sun exposure. But unlike the UV rays that can result in genetic alterations, the CRISPR-Cas9 system causes a mutation at a precise location in the genome.

When cellular machinery repairs the DNA break, it removes a small snip of DNA, rendering a gene non-functional. In this way, researchers can precisely turn off specific genes in the genome.

To turn off genes on a grand scale, both sets of investigators developed libraries of more than 65,000 sgRNAs. The library from Zhang's group is designed to target almost every protein-coding gene in the genome.

Both research teams used CRISPR-Cas9 to study the development of resistance to drugs used to treat cancer. The research team from the Whitehead and the Broad investigated genes whose loss conferred resistance to etoposide, a drug used to treat many forms of cancer including lung cancer and testicular cancer. The Broad and McGovern researchers used CRISPR-Cas9 to find genes involved in resistance to a drug commonly used to treat melanoma. In both sets of experiments, the research teams uncovered several previously unknown genes tied to resistance as well as many established genes.

In addition to these experiments on resistance, the researchers also conducted several other studies, using the CRISPR-Cas9 system in human pluripotent cells and cells grown in different culture conditions. The CRISPR-Cas9 system proved effective across these cell types, further demonstrating its versatility.

"With this work, it is now possible to conduct systematic genetic screens in mammalian cells," said David Sabatini, a member of the Whitehead, professor of biology at MIT, investigator of the Howard Hughes Medical Institute, senior associate member at the Broad, and a member of the Koch Institute. Sabatini is also a co-senior author of one of the *Science* papers. "This will greatly aid efforts to understand the function of both protein-coding genes as well as non-coding genetic elements."

The research teams compared the results of using the CRISPR-Cas9 system to turn off genes to the effectiveness of RNAi to turn down the signal of genes. In general, RNAi only partially reduces a gene's signal – knocking it down – while CRISPR-Cas9 turns off the gene's signal completely – knocking it out. RNAi is also prone to off-target effects – disrupting unintended gene targets – while CRISPR-Cas9 showed more consistent results.

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David Root, director of the Broad's Genetic Perturbation Platform (formerly known as the RNAi Platform), and his platform colleagues assisted in the development of the GeCKO (genome-scale CRISPR knockout) screening technique reported in the Broad-McGovern study. In addition to RNAi screening, the platform now offers a range of genetic perturbation technologies to the Broad community. That list now includes the CRISPR-Cas9 system.

"The CRISPR-Cas9 screening method distinguishes itself from RNAi by producing knockouts instead of knockdowns and it will be cleaner for many phenotypes to see the complete knockout," said Root who is a co-author of the Broad-McGovern paper. "The agreement among the distinct reagents targeting the same gene looks a lot higher for CRISPR-Cas9 compared to RNAi, which gives you a lot more confidence about gene specificity for these results."

One of the unique advantages to the CRISPR-Cas9 system that researchers intend to explore in the future is that the system offers access to the world beyond genes: non-coding regions of the genome that may influence when and where proteins are produced in a manner that does not depend on their production of RNA. Such regions were out of reach for RNAi, but with CRISPR-Cas9, these elements may now be exploitable in mammalian cells.

"These papers together demonstrate the extraordinary power and versatility of the CRISPR-Cas9 system as a tool for genome-wide discovery of the mechanisms underlying mammalian biology," said Eric Lander, director of the Broad Institute and co-senior author of one of the *Science* papers. "And we are just at the beginning: we're still uncovering the capabilities of this system and its many applications."

Other researchers who contributed to the Broad-Whitehead study include first author Tim Wang and Jenny J. Wei. Other researchers who contributed to the Broad-McGovern study include co-first authors Ophir Shalem and Neville Sanjana, Ella Hartenian, Xi Shi, David Scott, Tarjei Mikkelson, Benjamin Ebert, Dirk Heckl, and John Doench.

Funding for the former study was provided by the National Institutes of Health (NIH), National Human Genome Research Institute, the Broad Institute, and an award from the US National Science Foundation. The latter was supported by an NIH Director's Pioneer Award, the NIH, the Keck Foundation, McKnight Foundation, Merkin Foundation, Vallee Foundation, Damon Runyon Foundation, Searle Scholars Foundation, Klingenstein Foundation, Simon Foundation, Klarman Family Foundation, Simons Center for the Social Brain at MIT, German Cancer Center, Bob Metcalfe, and Jane Pauley.

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