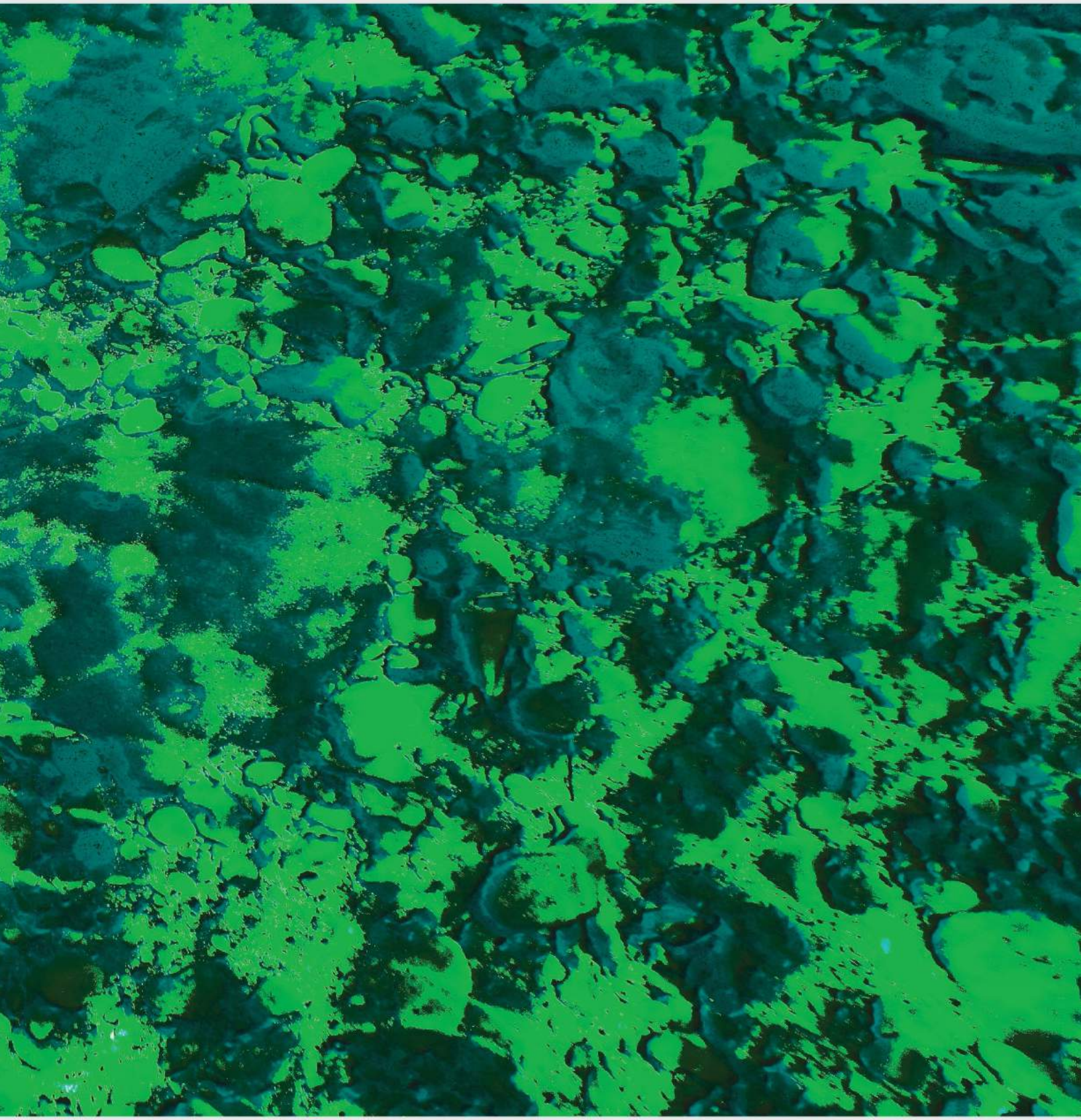
THE SYNAPSE

INTERCOLLEGIATE SCIENCE MAGAZINE ISSUE 21 | SEPTEMBER 2019



THE SYNAPSE

INTERCOLLEGIATE SCIENCE MAGAZINE

Writers

Meaghan Accarino
Rachael Branscomb
Monica Dix
Victoria Fisher
Anthony Gao
Anna Harrison
Julia Peterson
Neil Ruthen
Max Robinson
Elizabeth Toigo
Cecilia Wallace
Nathalie Weiss

Artists

Maria Altier
Julia Denlinger
Maeve Gualtieri-Reed
Claire Hoy
Jake Jorgl
Cecilia Larson
Steven Mentzer
Zoe Miller
Roger Ort
Aisling Smith
Rachael Williams
Yue Yu

Layout Editors

Victoria Fisher
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Emma Larson
Kathy Li
Megan McLaughlin
Steven Mentzer
Nico Vickers
Yue Yu
Joanna Zienkiewicz

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Rebecca Fenselau
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Anthony Gao
Loubna El Meddah El Idrissi
Emma Keppler
Emma Larson
Rajitha Narreddy
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Sarah Wilson
Joy Zhong

Copy Editors

Emery Angelo
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Dan Felley
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Alina Panek
Arin Pavlov
Caroline Pierotti
Elizabeth Rigby
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Emma Stillman

Photographer

Diep Nguyen

Cover Art

Barlow Wagner

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ENC Victoria Fisher (OC) & Emma Larson (OC)
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Emma Larson (OC '21)

With the intention of capturing a broad audience, contributors at The Synapse characterize scientific progression of the past, present, and future. As the Editor-in-Chief, Emma Larson (OC '21) works alongside writers, editors, and artists to make this magazine possible. We always welcome new and consistent contributors and appreicate our loyal readers. Thank you for supporting The Synapse!

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Featured Contributer

Diep Nyugen (OC '19)

Originally from Hanoi, Vietnam, Diep Nguyen graduated from Oberlin last spring with a major in biology. She took photographs of The Synapse team for two years. Not only an honors student but also a co-chair of the Vietnamese Student Association during her time at Oberlin, Diep is an incoming Ph.D candidate for the Computational and Systems Biology program at MIT. She loves The Synapse because it makes science fun and accessible, and allows for out-of-the-box creativity and collaboration. In her spare time, Diep likes going on walks, documenting interesting things with her camera, and playing guitar. She enjoys reading fiction books which are simultaneously an escape from this reality and a window into an alternate reality.

Featured Contributer

Rachael Branscomb (OC '19)

Rachael Branscomb graduated from Oberlin College with a major in biology and a minor in chemistry in the spring of 2019. She hails from Denver, but now works as a medical scribe for an emergency department in Brooklyn. Not only serving as Editor in Chief for a year but also contributing consistently as an editor and writer, Rachael started collaborating with The Synapse in 2016. The Synapse board will miss Rachael but she is excited to see what innovations it will implement as the magazine expands to other institutions. These days she especially enjoys exploring New York, visiting different parks, and playing with dogs outside of her work. Her favorite series is The Kingkiller Chronicles and given the chance, Rachael wouldn't hesitate to join the Arcanum and acquire some magical skills.

Contents

Fires in the Night Sky

All About Aurorae

Written by Rachael Branscomb
Illustrated by Yue Yu

On a frigid winter evening in Iceland you might be fortunate enough to witness curtains of brilliantly-colored lights gently rippling in a star-studded night sky. This phenomenon, commonly referred to as the northern lights or aurora borealis, has existed since the creation of our planet.

A variety of different cultures have long described aurorae with a certain magical mysticism. The Finnish word for the northern lights is revontulet, directly translating to "fox fires," referencing a fable in which a magical fox swept its tail across the snow, flicking flakes up into the night sky. Other Finnish myths describe aurorae as the spray from whale spouts. Algonquin Indian folklore of southeastern Canada attributes the northern lights to Nanabozho, the Creator of the Earth who continues to light great fires in the heavens, reminding the world of his everlasting love. Much farther to the south, the Maori people of New Zealand described the southern lights, or aurora australis, as the campfires of their ancestors.

But what causes these captivating night lights? Interestingly, the story of aurorae begins at the sun, far beyond our planet's atmosphere. The sun constantly produces the solar wind, a current of charged particles that flows throughout space. Fluctuating temperatures on the sun's surface cause solar storms that alter the strength of solar wind and lead to bursts of plasma called coronal mass ejections. Aurorae occur when the Earth happens to intersect the path of the solar wind as it travels through space.

The sun's solar wind is only part of the story; aurorae also depend on a unique characteristic of our planet. Just like a bar magnet, the Earth has a positive and a negative end. The Earth's magnetic field flows out of one pole, looping out in all directions, and comes back in at the other pole. This creates a large, squashed, beach-ball shape known as the magnetosphere. The magnetosphere is integral

> Aurorae occur when the Earth happens to intersect the path of the solar wind as it travels through space.

to maintaining a hospitable environment for life on Earth because it blocks incoming radioactive material carried by the solar wind. During a coronal mass ejection, the magnetosphere blocks most of the solar wind from entering our atmosphere. The force of the solar wind pushes the magnetosphere into a teardrop shape, where the side facing away from the sun develops a long tail of magnetic charge that extends even beyond our moon's orbit! In this way, the magnetosphere acts as a sort of shield, protecting the planet from the harmful radiation of solar storms. However, some ions from the solar wind manage to pass through the magnetosphere, following the Earth's magnetic field lines down into the planet's upper atmosphere at the poles. There the ions collide with atoms in our atmosphere, releasing the brilliant lights seen in aurorae.

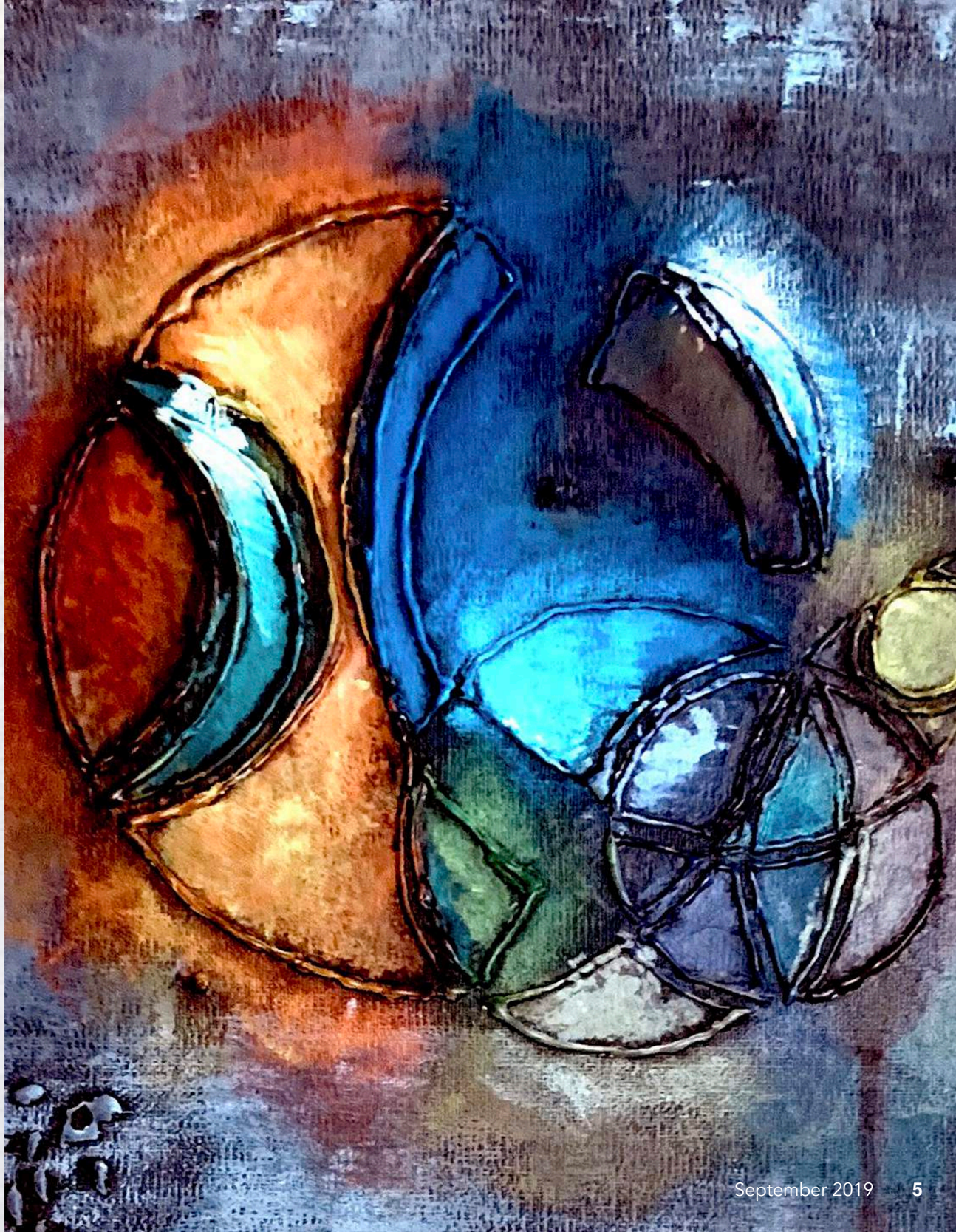
The color given off by such a collision can be explained by variations in altitude and colliding elements. Atoms are comprised of a central nucleus around which a diffuse cloud of negatively-charged electrons orbit. When the charged particles from the sun strike atoms

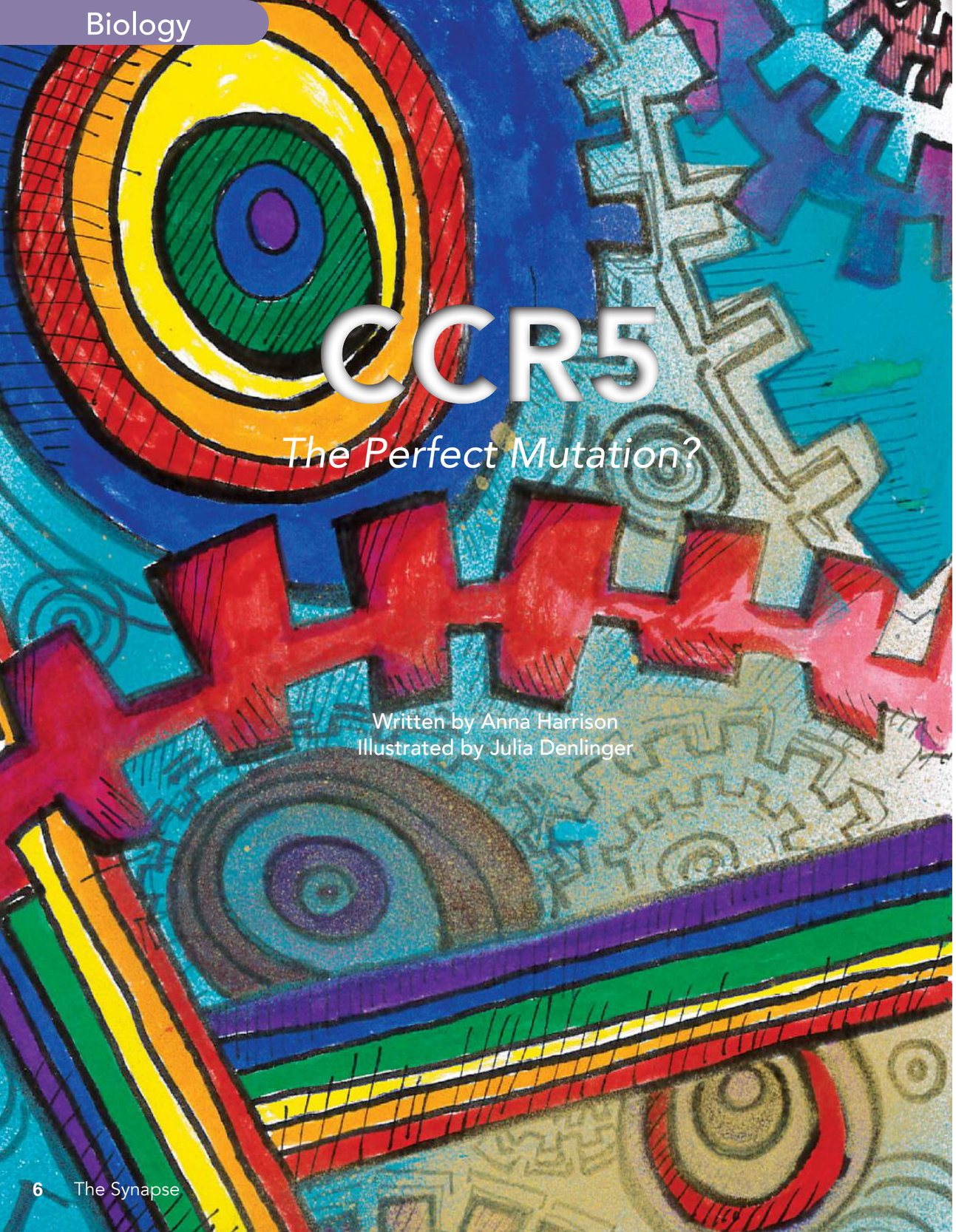
> The radiant colors of aurorae depend on both the identity of the atom and the altitude at which the collision takes place.

in the atmosphere, the atoms' electrons are excited to higher energy levels, moving them away from the nucleus. When the electrons fall back down to lower energy levels, they release energy in the form of photons and give off light. The radiant colors of aurorae depend on both the identity of the atom and the altitude at which the collision takes place. Ions colliding with oxygen atoms at higher altitudes give off red light whereas lower altitude collisions yield green and yellow hues. Nitrogen atom collisions appear red or blue. Hydrogen and helium atoms can produce purple, though this color falls within a part of the electromagnetic spectrum that our eyes can't detect.

Though aurorae generally appear as rippling sheets at the northern and southern poles, they also form spirals and arcs oriented along the Earth's magnetic field and occasionally appear at lower latitudes. Midlatitude aurorae may be seen when magnetic storms on the sun increase the activity of the solar winds and coronal mass ejections. Such magnetic storms typically occur near the spring and autumnal equinoxes and have generated aurorae seen as close to the equator as Mexico!

Furthermore, aurorae are generated through a similar mechanism to the neon lights that decorate the building fronts of any typical downtown street. Neon lights are made up of glass tubes filled with gases like helium, neon, or argon. Electricity is run down the length of the tube, colliding with the gas atoms and releasing colored photons of light. The next time a bright neon sign catches your eye, recall the dazzling natural displays of the aurora borealis and the aurora australis. • • •





CCR5

The Perfect Mutation?

Written by Anna Harrison
Illustrated by Julia Denlinger

Scientists have had the ability to edit genes for years, yet in 2012 a new development made gene editing much easier, cheaper, and more precise. Since then this tool, known as CRISPR-Cas9, has been integral to many advancements in fields across all disciplines of science. Of particular interest are the applications of this tool to the genes of germline cells.

Last year, Chinese geneticist He Jiankui was the first to implant CRISPR-Cas9 edited human twin embryos, which were carried to full term and apparently healthy upon birth in 2018. Since the father of the twins was infected with HIV, Jiankui attempted to reduce the twins' chances of contracting the virus during pregnancy by targeting the well known CCR5 delta 32 mutation. This mutant variant, which is associated with resistance to the bubonic plague, prevents most strains of HIV from infecting host T-cells—major players of the immune system—and is naturally prevalent in the human population. The resulting phenotype is resistance to contracting HIV.

But what exactly is this mutation? The CCR5 gene codes for a membrane protein that is present on many cells in the immune system. It is thought to play a role in guiding our immune cells to their foreign targets in order to initiate immune responses. Like most immune mechanisms, CCR5 activity can have positive and negative effects, depending on the context. HIV has exploited the membrane protein to anchor down onto T-cells, allowing the virus to inject its virion or infectious genome. Due to the structural changes in the mutant variant, CCR5 delta 32, it cannot be expressed on the T-cell membrane. These differences prevent most strains of HIV from injecting its noxious genome and proliferating infection.

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Since this mutation resulted in resistance to the plague, it is naturally prevalent in the population, making it relatively easy to study. Various health outcomes have been the topic of interest related to CCR5 delta 32. The shocking part of these outcomes is that most of the effects are positive. CCR5 delta 32 has been shown to prevent or slow the progression of several chronic inflammatory diseases, such as West Nile virus and Hepatitis B. The mutation also reduces the risk and recovery time of stroke, traumatic brain injury, dementia, and Alzheimer's disease.

The mechanism by which the mutation improves neuroinflammatory conditions is not entirely understood. The absence of active CCR5 dampens the host's immune response to neuroinflammatory diseases, thus reducing symptoms and damage, and allowing the brain to heal itself. In healthy people, the CCR5 gene is thought to promote learning and memory by regulating the excitability of neurons. The dampening of this excitatory effect is thought to limit damage in the context of stroke or traumatic brain injury. Conflicting studies have shown that individuals with the CCR5 delta 32 variant have enhanced memory and reduced risk for dementia.

While individuals with the CCR5 delta 32 variant may have a slightly greater susceptibility to certain viral infections, they tend to live normal, healthy lives. It is possible that the body responds to the absence of CCR5 gene expression by expressing other genes that allow "mutant" individuals to lead healthy lives. This is why isolating the CCR5 mutation could be dangerous: The body may not have the same negative feedback responses. The greatest concern, however, is for the off-target effects that gene manipulation may have. Additionally, certain strains of HIV remain able to infect mutant T-cells that do not express CCR5 through a different T-cell membrane protein, CXCR4. The broad distribution of CCR5 delta 32 T-cells through gene therapy may simply select for the strain of HIV that does not require CCR5 to get into the T-cell, just as certain antiretroviral therapies have selected for certain strains.

In theory, it is feasible that in order to eliminate the possibility of HIV infecting human T-cells on a population level, a combination gene-therapy would be necessary to eliminate risks of resistant strains developing. However, the odds of off-stream effects developing in such cells skyrocket. Most scientists would agree that we are still many years away from editing the genome of an embryo that will be carried to full term, and that He Jiankui's use of CRISPR on an implanted embryo was irresponsible at best.

In fact, the geneticist did not create the well studied and widely present CCR5 delta 32 mutation in the genetically modified twins. It is believed that this "delta 32" mutation is characterized by a deletion of 32 base pairs in the DNA sequence that normally codes for the membrane protein. Jiankui performed different deleterious mutations in each twin: a deletion of two base pairs and a deletion of fourteen. While it is possible that the mutations which he created will result in the same phenotype as the well-known delta 32 mutation, this manipulation comes across as poor science. Results of his ethically concerning "experiment" will be confounded because the mutations that he introduced aren't well studied in the population or the lab.

Ultimately, the negative effects of having an inactive CCR5 gene are not well understood. Neither are the negative effects of mutating this gene. By extension, the effects of two relatively new mutations that were introduced in these two twins are unpredictable. Dr. Jiankui may have set back the progress in this incredibly important field dramatically by performing this procedure without proper testing or approval. Restrictions have tightened globally since Jiankui's announcement. A global moratorium was written to ban the use of human germline modification around the world, and 30 countries have already implemented laws prohibiting such use.

Whether or not this mutation is the "best," it may very well be the one we know the most about, having the most diverse and possibly therapeutic effects. Yet as we reflect on the incredible work that has been done to reveal the roles of CCR5, this is but one of a million stories that our genes have to tell about the potential that they hold. • • •

Phage Therapy

The Abandoned Treatment Making a Comeback

Written by Anthony Gao
Illustrated by Maria Altier

Since the discovery of penicillin, antibiotics have been used as a treatment for a variety of bacterial infections to save countless lives. However, the frequent use of antibiotics has prompted bacteria to develop resistance, leading to the rise of "superbugs" that are unaffected by current medications. Pharmaceutical companies have fended off the problem for the past few years by cranking out new chemical remedies, but there are only so many compounds that they can develop to combat each unique pathogen. In response to these "superbugs," the number of new antibiotics that have been approved over the past 30 years has dramatically declined. Realistically, it is time to explore new approaches to stopping bacterial infections—and what better way than by harnessing the power of bacteria's natural enemy, the bacteriophage?

A bacteriophage, or "phage," is a type of virus that is tailored to seek out and hijack a specific strain of bacterial host. Phage therapy involves the ingestion of bacteriophages, usually taken orally, although they can also be applied to an infected wound or used during a surgical procedure. An injection is not often used, as the immune system naturally attacks viruses entering through the bloodstream. Usually the phages are selected after a swab sample is taken from a patient, although generic cocktails of phages targeting common infections are available in several Eastern European countries. It may seem dangerous to pump live viruses into a human body, but bacteriophages are only harmful to their target bacteria and have not shown any adverse effects in human patients. Outside of medicine, phage therapy has applications in veterinary science and agriculture, where it is used to eliminate food borne illnesses such as salmonella.

While phage therapy may be unfamiliar to many people, it is not a new idea. Scientists have experimented with bacteriophages in medical applications since Felix d'Herelle discovered them

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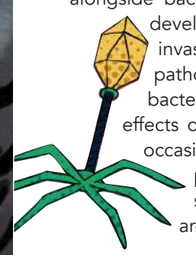
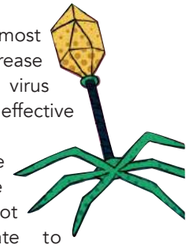
in the mid-1910s. After reports in the 1920s claimed it had the potential to cure dysentery, phage therapy became a hot topic in the medical community, and its use quickly spread throughout the developed world. However, phage therapy was often a matter of chance, as scientists at the time knew nothing about the mechanisms of bacteriophages beyond that they could kill bacteria. The practice was poorly understood and unregulated, and it was soon shut down after further testing found inconclusive results and showed that previous publications utilized substandard protocols and tampered data. Following the discovery of penicillin in the 1940s, all consideration of phage therapy was extinguished, and scientists all but abandoned the practice.

Despite its disappearance in much of Western society, phage therapy lived on in parts of Eastern Europe as a result of the Cold War. After cutting off scientific contact with the Western world, the Soviet Union fell behind in antibiotics research, instead opting to develop phage therapy with some success. The technique lost popularity after the Soviet Union dissolved, but phages are still widely marketed and used around Eastern Europe today, especially in Georgia where they are legally recognized. In recent years, following the rise of "superbugs," Western society has revived the study of bacteriophages as a medical remedy, and many companies are beginning to focus on the promising technique. For example, in 2006, the American company Intralytix obtained approval from the FDA to add phages to prepared food, although the FDA has yet to approve of phage therapy as a medical treatment.

So how exactly is phage therapy any better than modern antibiotic development? For one, bacteriophages can evolve alongside bacteria, making it more difficult for the bacteria to develop resistance. In addition, phages are not toxic or invasive, and won't destroy anything besides the target pathogen, while antibiotics will often damage the helpful bacteria in our guts. There are few documented side effects of bacteriophage treatment in humans beyond the occasional immune response, making it a relatively safe procedure. Phages can be effective even in a single small dose, thanks to their ability to replicate, and are also capable of damaging gram-negative bacteria that have double cell membranes, something that most antibiotics cannot do. Most importantly, with the increase in new technology and knowledge relating to virus function, the treatment has grown to be much more effective than it was 90 years ago.



Although it has potential, phage therapy is certainly not a miracle cure. Despite recent developments in the technology, it has not yet been used with a high enough success rate to meet modern standards. A single type of phage will often not be sufficient to wipe out the variety of microbes responsible for an infection, necessitating a cocktail of several different phages and antibiotics. This takes time and resources to develop, making the treatment expensive. Furthermore, phage therapy can cause an adverse reaction in some patients. It may trigger an immune response, as the human body's nonspecific immune system is wired to attack anything unfamiliar. Additionally, viruses kill bacteria by rupturing their cell wall and membrane, which may cause the bacteria's toxins to spill out and damage nearby body cells. Not all phages can be used for treatment, considering viruses' two different cycles of reproduction: the lytic cycle in which viruses rapidly replicate themselves before bursting out of the bacteria, and the lysogenic cycle in which viruses allow the bacteria to remain alive so they can steadily produce more offspring. Phage therapy can only be done with viruses that exclusively use the lytic cycle, as only lytic viruses will kill their host bacteria as intended, while lysogenic viruses benefit from ensuring that their host survives. However, these problems are solvable: Scientists have recently gained the ability to modify phages such that they bypass an immune response or lyse a bacterium while containing its toxic residue.



Currently, the progress of phage therapy is trapped behind financial and regulatory hurdles. Regulatory agencies will not approve of the technique without a large-scale clinical trial, and investors will not provide money to research the technique

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due to high perceived-risk. Many private companies have worked on developments in phage therapy, but none have been able to treat infections with the technique consistently. Despite advances in molecular biology, much of the research on phage therapy involves guesswork, and significant time and effort are needed before scientists can use it efficiently. However, phage therapy has shown some success recently as a last-resort treatment, saving the lives of several patients with infections that were unresponsive to other methods. Overall, as more bacteria develop resistance to pharmaceutical treatments, the demand for new forms of medicine will grow. Phage therapy offers a promising solution. • • •

Phones: The Hand We Don't Wash

How to Protect Yourself Against Common Diseases

Written by Nathalie Weiss
Illustrated by Maeve Gualtieri-Reed

Do you think it's unsanitary not to wash your hands after using the restroom? If you answered "yes," the majority of Americans would agree with you. A survey found that 95% of Americans claim they wash their hands after using the bathroom. However, 75% of Americans also admitted to using their phones in the bathroom. Our phones have become analogous to a third hand. They are a third hand that we don't wash after using the bathroom, if ever. The biggest contributor of germs to phones are hands. A survey carried out by Deloitte, one of the "Big Four" accounting organizations, found that the average American checks their phone 47 times per day. That's 47 chances a microorganism has to jump onto your screen! This makes one wonder: What kinds of germs do our phones actually carry? Are they harmful?

Microbiologists at the University of Arizona found that smartphones carry more bacteria on average than toilet seats. This is not surprising, as our thighs carry fewer germs than our hands. Thighs do do not touch as many objects in a day as hands do.

One of the bacteria most commonly found on our phones is E. coli, a type of fecal coliform that is found in human and animal feces as well as in the soil. Before you get nervous, we're not talking about the type of E. coli that causes foodborne illness: E. coli O157: H7. It is much less common to see this type of E. coli on your screen than one of the more benign strains. One such benign strain is E. coli HS, which actually helps digestion in humans.

Staphylococcus aureus is another type of bacteria commonly found on our cell phones. Some kinds of Staphylococcus aureus can cause staph skin infections. In a 2009 study, it was found that 52% of 200 health care workers' cell phones were found to have S. aureus on them. It was also revealed that 38% of those phones were exposed to the methicillin-resistant strains of the bacteria, which can cause painful skin boils and are transferred by skin contact.

Another bacteria found on cell phones that are less harmful than S. aureus is Coagulase Negative Staphylococci (CoNS.) This bacteria is antibiotic resistant and resides in human skin and the vaginal tract. It has the capability of causing bloodstream infections. A 2011 study in Ghana found that 15% of the cell phones randomly selected from 100 students tested positive for CoNS. You might think twice before putting your phone to your face!

Aside from harming your skin, the germs on your phone can also affect your respiratory system. In a Turkish healthcare workers study, 10% of the phones tested positive for mold. Exposure to mold can result in shortness of breath, nasal congestion, fevers, and, sometimes even lung infections. The same study also found yeast on 1.5% of the phones tested. Yeast is a bacteria that can live almost anywhere on the human body and it can cause itching and vaginal discharge.

Another respiratory infection that we can catch from our cell phones is strep throat. The bacteria responsible for this infection are Streptococcus pyogenes, which can also cause other illnesses like pneumonia, meningitis, and sepsis. Luckily, if you do happen to catch strep throat, it can be treated with antibiotics.

Two very common illnesses, the common cold, and the flu can also be caused by exposure to bacteria on your phone. The common cold may be caused by coronaviruses or rhinoviruses while the flu is caused by Haemophilus influenzae bacteria. The flu is more serious. Its symptoms include mid-to-high fevers, sore throat, coughing, fatigue, headaches, body aches, and congestion. To keep yourself healthy during flu season, get a shot and sanitize your phone!

Now that we've covered some of the common bacteria found on your cell phone, you are probably wondering how you can keep yourself safe against these bacteria. To safely clean your phone, you may need a microfiber cloth, isopropyl rubbing alcohol, water, cotton swabs, and cleaning gloves, according to Apple, Google, and Motorola. When cleaning a waterproof phone, you can make your own cleaning solution with rubbing alcohol and distilled water and put it into a spray bottle. It is then recommended that you spray the outside of the device and wipe it down with a cotton swab while wearing gloves. For non-waterproof phones, you can use a Lysol wipe, which the company advertises as safe to use on electronics. Disinfectants for your phone include UV lights, which can kill up to 99.9% of the germs on your phone. UV lights are sold by PhoneSoap. Dr. Charles Gerba, a microbiology professor at the University of Arizona, recommends sanitizing your phone daily. By following these tips, you can protect yourself against common diseases as well as keep your phone looking brand new. • • •

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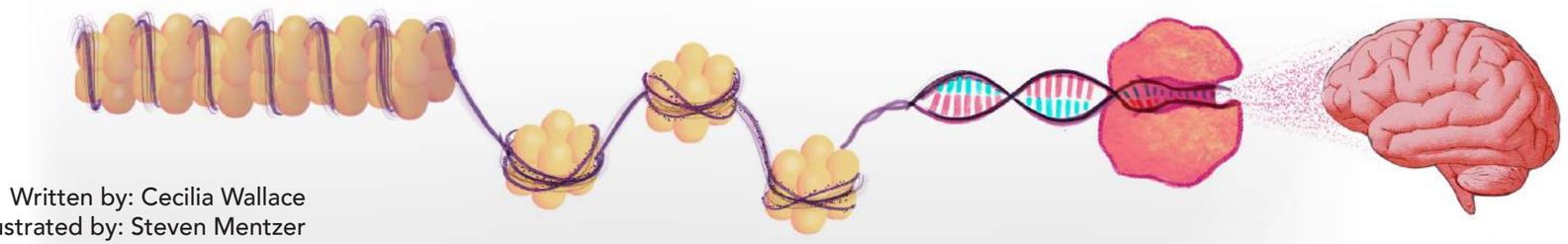
It's Not Your Genes, It's How You Use Them

An Intro to Epigenetics



Written by: Cecilia Wallace
Illustrated by: Steven Mentzer

Our genetics: the tangle of DNA that we inherit from mom and dad, the arsenal of genes encoded within the DNA that forms us, the celebrated blueprint that makes us who we are. With the rise of cheap, personalized genetic testing by companies like 23andMe, people know more about their genetic material than ever before. "They analyzed my DNA, and told me I probably have black hair!" "You do have black hair..." "So there you go." It is a comforting thought that we are built from such a definite, orderly set of genetic instructions.

Not to disturb this peace, but have you ever wondered how our liver cells and brain cells have exactly the same genetics—the same set of DNA—and do completely different things? Our intestinal cells have built a happy life for themselves cranking out fat-digesting bile, while our brain cells have embarked on a more academic lifestyle, passing our thoughts back and forth through precise electrical signals. Our liver cells can't make the equipment needed to join our brain cells' conversation, and our brain cells couldn't digest fat to save their lives. But why is that? Don't they have the exact same genetic material?

In the 1940s, Conrad Waddington wondered about the same things, and concluded that there must be some conductor beyond genetics that directs each cell to use all the DNA that it has

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been given. He called this mysterious orchestrator "epigenetics." "Epi" is a Greek prefix that means "above," "over," or "upon," so "epigenetics" refers to all that is above/over/upon simple genetics. Our genetics is the big wad of DNA that we received from mom and dad. Our epigenetics is how we use that DNA. Liver and brain cells have the same genetics. They just use it very differently. Waddington couldn't have known, but epigenetics controls far more than just cell differentiation. It also plays a role in things as innocent as lactose intolerance and coffee addiction, as significant as learning and memory, and as somber as cancer and diabetes.

Epigenetics is the new science on the block. For decades we've been educating about genetics, integrating it into medicine, and incorporating it into societal conversations. Scientists only started to really understand epigenetic pulleys and levers in the 1990s. And we are just starting to work through its societal implications.

For example, epigenetics renders us more deeply sensitive to the environment than we previously understood. This lends extra urgency to issues of environmental justice—what conditions people live and work in, who has to live and work in them, and what that implies about pollution and healthcare. Even more famously, landmark studies since 2010 suggest that trauma may be passed down over generations epigenetically, even though it can't alter DNA sequences at the genetic level. This has serious implications for huge groups of people, like the descendants of Holocaust survivors and victims of European colonialism.

Epigenetics is dazzling mechanistically, and significant socially. To understand how epigenetics works, we have to briefly review what happens in regular old genetics. In genetics, the main character is the gene. There are about 20,000 genes written into our DNA. A gene hopes that one day the protein it codes for will actually be built and go on to make a name for itself out in the body. For that to happen, first a scribe-like enzyme called RNA polymerase must read the gene and copy it into a molecular transcript. Then, the resulting transcript must make its way to a sort of protein-making factory called a ribosome. The ribosome uses the transcript to construct the correct protein. Once the protein is built, it goes off to do its thing—maybe break down fats, maybe detect neurotransmitters. At this point, the gene has satisfied its destiny and made itself known in the world; the gene has been expressed.

Epigenetics meddles with gene expression in many ingenious ways. There's no one main character—there are more mechanisms than we could possibly cover, though there are some main players that provide a taste of what epigenetics can do. We'll explore three: histone modification, DNA methylation, and RNA interference.

Through histone modification, epigenetics can determine whether or not a gene is accessible to be read in the first place. See, our cells have contrasting needs with all their genetic material. They each have about six feet of DNA that they need to pack into an incredibly tiny space, so they need to compact it tightly. But they also need to transcribe genes to make proteins, so they need to loosen the DNA enough to let the scribe enzyme, RNA polymerase, get at the necessary genes. Histones offer this instrumental flexibility. Histones are little protein spools that help wrap up our DNA. Our DNA loops twice around each histone before moving to the next, so a full chromosome looks something like a strand of histone spools connected by a DNA string. One crucial epigenetic tool can tag one of these histone spools and make it wrap tighter to the other spools. Any gene wrapped around such tagged, scrunched histones becomes impossible to reach. An opposite tool can make histones loosen up, rendering any gene wrapped around those spools easily accessible to RNA polymerase. A brain cell doesn't need to use its genes that make bile, so it tags the histones wrapping those genes to tighten up. A liver cell, on the other hand, needs its bile genes, so it tags the histones to loosen.

Even if a gene is accessible, looped loosely around its histone spools, epigenetics can prevent it from being transcribed into a message. DNA methylation is an epigenetic tool that stamps small molecular roadblocks, called methyl groups, onto a gene to block RNA polymerase from binding where it needs to bind. Arsenic is one of the most common environmental toxins, often inhaled in occupational settings or ingested in ground water. Research has found that it methylates important cancer-suppressing genes. Once those genes are methylated, RNA polymerase can't bind to the DNA, so the genes can't be made into a transcript and will never be built into crucial tumor-fighting proteins.

Finally, even if a gene is accessible around its histones, remains totally unmethylated, and is transcribed successfully, there is an epigenetic tool that can blow the transcript out of the water and prevent it from becoming a protein. In RNA interference, a mirror image version of a gene transcript—a sort of molecular alter ego—hunts the transcript down as it makes its tentative way to a ribosome protein factory. The transcript's alter

> Epigenetics explains that we're more sensitive to environmental exposure than previously thought. Some materials in our environment like arsenic and nickel can't mess with our sturdy DNA (though others can), but they can take over epigenetic tools and alter the way we express our genes

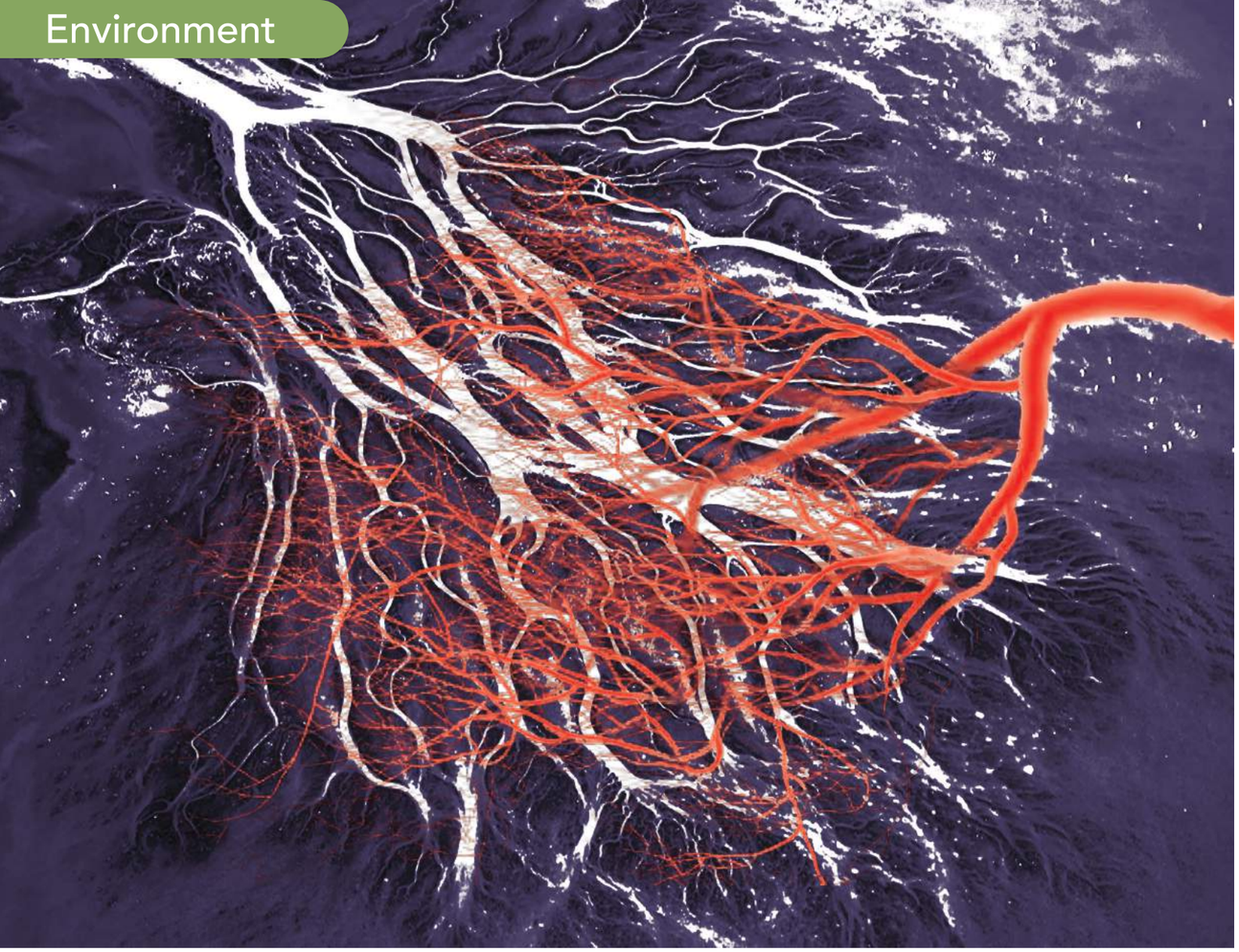
ego complements it perfectly, so they bind together and the alter ego drags the original to a sort of cellular shredder.

These three mechanisms represent a tiny sample of all the epigenetic tools at play in our bodies. They can be used intentionally by our bodies, hijacked by environmental exposure, or induced by experiences. When epigenetics helps our liver cells express different genes from our brain cells, all is well in the world. But other sources of epigenetic change—like environmental exposure and stressful experiences—have more complicated implications.

Epigenetics explains that we're more sensitive to environmental exposure than previously thought. Some materials in our environment like arsenic and nickel can't mess with our sturdy DNA (though others can), but they can take over epigenetic tools and alter the way we express our genes. This adds extra weight to environmental justice and occupational health rights. Ohio is home to many fracking wastewater injection wells; toxic fluid left over from fracking is injected into special wells that have a history of leaking into groundwater. Furthermore, toxic environmental exposure often follows racial lines, as systemic racism in politics directs dumping and industry towards marginalized communities. Negative epigenetic effects hit these communities hardest.

In addition to environmental health problems, epigenetics presents the issue of inherited trauma. A parent can't pass down their life experiences to a child genetically—they can only pass on their set DNA sequence. A parent who lifts weights for years can't pass down any genes for extra muscle. Similarly, trauma and prolonged-stress don't mutate our genetics. But they do change our epigenetics—after a long period of starvation, for example, we turn up genes that slow metabolism and can lead to future health problems if food becomes more available. Previously, scientists didn't believe epigenetics got passed down to children. It seemed like we erased any histone modifications, DNA methylation, and RNA interference when we built eggs and sperm. But recent studies have begun to suggest that in fact, we can pass down epigenetic changes. Famously, researchers examined people who were babies during the Dutch hunger winter of 1944, along with their children. The parents had health problems that suggested starvation-induced epigenetic changes. And shockingly, so did their children. This means that the trauma our ancestors experienced could be passed down to us epigenetically. While the mechanism for this inheritance is still uncertain, it adds further gravity to the effects of trauma and injustice.

Epigenetics encompasses a wild box of fascinating phenomena, which have serious implications for society. Inherited trauma is just one such phenomena, but we may uncover many more with future research. Epigenetics is certainly a field worth following. • • •



Weeks of Inundation, Years of Devastation

Processing Catastrophic Midwestern Flooding in Spring 2019

Written by Monica Dix
Illustrated by Jake Jorgl

With a short news cycle and a building number of extreme weather events due to climate change, it would have been easy to let the catastrophic flooding of this past March slip under your radar. Taking nearly a week to manifest in full force, the extent of the flooding left media, government agencies, and scientists blindsided. Concentrated on the Missouri River, tributaries in Iowa, Nebraska, and Missouri saw the worst of the flooding, but the effects extended to South Dakota and Wisconsin. Reports after the worst of the flooding show at least $2.9 billion in property damage and three deaths.

Nebraska and Iowa faced some of the most dramatic impacts. In Iowa, the governor signed an emergency disaster proclamation on March 14, and asked the president to declare a disaster in 67 counties. Nearly 100 miles of Interstate 29 were closed between Missouri and Iowa, parts of the road under more than 15 feet of water. There were 30 levee failures, mostly in the Council Bluffs area, that left 1,200 homes destroyed or extensively damaged, with 23,540 having minor damage. The damage in Nebraska reached an estimated $1.4 billion, with 2,000 homes and 341 businesses damaged or destroyed. Infrastructure failures in the state exacerbated flooding: For example, the Spencer Dam collapsed on the Missouri River and created unregulated flooding that collapsed three bridges downstream. This contributed to record flooding on the Missouri, Platte, and Elkhorn Rivers; in some sites historical records were broken by as much as five feet. States of emergency were also declared by governors in Missouri and Wisconsin.

How did this happen? On a short-term scale, weather conditions created what meteorologists have characterized as "the perfect storm." The region had lots of preexisting ground moisture from flooding in September and October of the past year, which left the ground frozen and densely saturated through the winter. In early 2019, temperatures in the Midwest held below freezing with record snowfall, then when the bomb cyclone hit in March,

> The rainfall intensity triggered widespread snowmelt, and ground absorption so limited that it has been compared to cement, conspiring to cause record amounts of runoff into local streams and rivers.

temperatures rose to 60 degrees after just three days in some states, coupled with 1.5 inches of rain. The rainfall intensity triggered widespread snowmelt and ground absorption so limited that it has been compared to cement, causing record amounts of runoff into local streams and rivers. Additionally, the force of the water broke up the existing thick ice cover on rivers, dislodging ice chunks into projectiles that flowed down the stream and knocked out bridges as they went.

On a long-term scale, this event was a culmination of weather, water, climate, and aging infrastructure. While the weather and water can be understood as part of the storm itself, a changing climate which promises more variability and intensity in regional weather patterns is a relatively unpredictable component of the system. Infrastructure is the main factor that we can actually anticipate. While it is easy to hope that future communities will not build on these high-risk floodplains of major rivers, there is a lot of modern development along the banks of these rivers which depends on vital infrastructure like levees and dams for protection. In 2011 one Iowa town with a population of 1,200 even recorded a flashmob in an attempt to raise funds for an increase in height of their levee. In part due to an inability to raise the needed money, the recent flooding left that town completely underwater, speculated never to recover. With limited funding and hundreds of miles to protect in these states, resources are spread thin enough that catastrophic floods jeopardize livelihoods of those in the region.

Is this a one-off? The National Oceanic and Atmospheric Administration's 2019 flood watch indicates that flooding of severe magnitude will continue to persist. Ed Clark, director of NOAA's Water Center said of the flooding forecast, "The extensive flooding we've seen in the past two weeks will continue through May and become more dire and may be exacerbated in the coming weeks as the water flows downstream. This is shaping up to be a potentially unprecedented flood season, with more than 200 million people at risk for flooding in their communities."

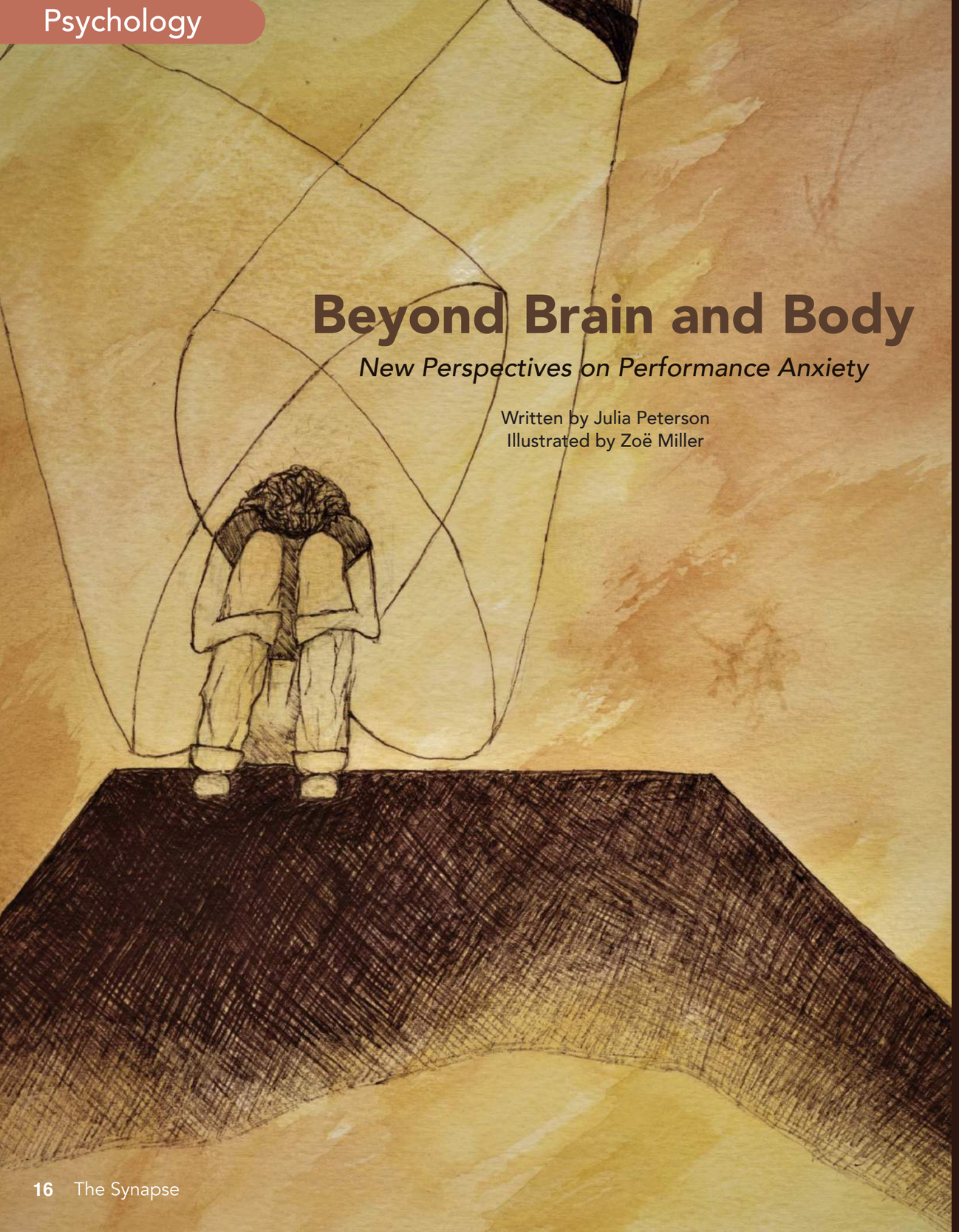
The economic impacts of this flood show the potential, financial devastation of further flooding. Out of the estimated $1.4 billion lost in Nebraska and $1.6 billion lost in Iowa, $840 million

> These are felt not just on a financial scale, but as we continue to stress our planet's carrying capacity, we will more directly feel the this impact as food shortages.

were in crop and cattle losses and $240 million were in agriculture, respectively. Farmland is supposedly plantable into the late season, with fields potentially silted and unusable for years, while livestock losses are even more difficult to recover from. These are felt not just on a financial scale, but as we continue to stress our planet's carrying capacity, we will more directly feel an impact with food shortages. From this event alone, over 200 miles of Iowa levees need repair, at a cost of $525 million to federal and nonfederal stakeholders. In Nebraska, cleanup efforts are predicted to stretch on for the rest of the year, with estimates taken from historic flooding in 2011, when water took as long as 3.5 months to recede. While no contamination warnings have arisen, as the water recedes it likely carries pollutants. A number of propane tanks seen floating downstream in Nebraska represent the current and continued risk to water quality from flooded and contaminated sites.

Looking towards the future, the mission is to become proactive rather than reactive. States can invest in better flood monitoring, as deployed in Iowa after 2008 flooding, including bridge sensors for water levels to provide real-time inundation maps. With similar issues in the lower Mississippi delta, solutions such as extensive modeling of affected regions to monitor the way that the river will adapt to different precipitation-loads, or gated and channelized openings in the levees (the bill for this project estimated at $1.4 billion) are seen as additional options. The constant reassembly of regional flood-protection infrastructure after events like this provides an economic incentive to invest in these measures that mitigate flood risk. However, some stakeholders also argue that money should be invested into better land management with long-term outcomes which reduce devastation as events continue to increase in magnitude. Yet, no plans have been made to recover from such disaster.

Overarching land-management reform along major rivers is a big step, so a place to begin is creating awareness of media biases for coverage of environmental disasters. Writers like former American Meteorological Society President J. Marshall Shepherd have discussed the presence of urban and coastal biases toward extreme climate and flooding events. For example, it might surprise us that six of the eight states experiencing the most major floods are inland given the intensity of reporting around hurricane-driven flooding. Since 2008 this is the 14th flooding disaster in Nebraska, which ranks fifth in the nation for flooding. Compared to the effects of hurricanes, the impacts of river-based flooding are much more widespread, especially in the Midwest, where towns are isolated with far fewer financial resources including limited access to flood insurance. Therefore, awareness around the impact of these events is not only important for understanding our changing climate, but also protecting communities across the nation where people have resorted to dancing in the street as the last hope of survival for their town. • • •



Beyond Brain and Body

New Perspectives on Performance Anxiety

Written by Julia Peterson
Illustrated by Zoë Miller

From the stage to sports to public speaking, performance anxiety is a common experience and has become more widely discussed in recent years. Top-level performers, including Olympic skier Mikaela Shiffrin, world-renowned opera singer Amanda Majeski, and award-winning actor Hugh Grant, have been interviewed about their struggles with nervousness before events.

When we think about performance anxiety, there is a tendency to think about it as an internal trait: Some people have performance anxiety while others do not, and people who do have performance anxiety have to find ways to cope with it so that it won't interfere with their life and work. However, external elements on performance anxiety, such as the systemic biases that people face, the type of activity that they do, or the space that they use, should not be ignored. A great deal of the research around performance anxiety focuses on potential physical and psychological causes and remedies. Alongside this area of focus, some researchers have broadened the scope of the field to examine factors beyond an individual's brain and body that affect their performance anxiety. These factors may also prove fruitful in enhancing our understanding of this experience.

Many performers experience anxiety, even those at the highest levels of their discipline. According to the International Conference of Symphony and Opera Musicians, 24 percent of professional musicians experience stage fright, and other studies suggest that this number may be much higher. However, not all performance situations are created equal. Researchers, Nicholson, Cody, and Beck, found that professional musicians are significantly more likely to experience performance anxiety when they are giving a solo performance as opposed to performing in a group. This study provides evidence for the common belief that people are more anxious with an audience's undivided attention,

> People are more anxious when they believe that they have the audience's undivided attention and less anxious when that attention is split.

and less anxious when that attention is split between performers. Another study examining the relationship between audience and performers—in this case, actors—found that the actual layout of the theater had a significant impact on performance anxiety; actors in front of a packed audience in a small space felt more anxiety, while actors in front of a more dispersed audience were less anxious.

There is also evidence to suggest that working in different genres affects performance anxiety. A study of young musicians between 7 and 20 discovered that classical musicians and performers of popular music tend to develop performance anxiety at different ages. Young classical musicians exhibited high levels of performance anxiety while older classical musicians reported lower levels, and older popular musicians reported significantly more performance anxiety than their younger counterparts. The study also found that women across all genres of music were more likely to experience performance anxiety than men. The anxiety levels for musicians of different ages could be an effect of the different cultures around the different types of music, but there are other possible explanations. Perhaps something about being a classical musician in particular prompts more women to leave the genre before their late teenage years. As a result, if male classical musicians' performance anxiety did not increase as they aged, then the average performance anxiety in older classical musicians would appear to be lower, even if the effect had nothing to do with age.

While some causes of performance anxiety may be hardwired in each individual—with some people being naturally more susceptible to experiencing performance anxiety than others, some situations or experiences can cause performance anxiety in people who may not experience it otherwise. For example, another group of researchers, Mizala, Martinez, and

> 24 percent of professional musicians experience stage fright, and other studies suggest that this number may be much higher.

Martinez, found that when girls in primary school have a female teacher who has performance anxiety related to math, those students become more likely to agree with gendered stereotypes about math, to experience higher levels of performance anxiety when doing math, and to perform worse on math tests than their male counterparts. This research suggests that if systemic bias against their demographic already exists in an area and their teacher displays performance anxiety in that same field, then students who resemble this teacher will have increased performance anxiety related to a subject.

Another way that researchers have been looking at performance anxiety is in terms of rituals such as a student bringing their "lucky pen" with them to take a test or a tennis player bouncing the ball a specific number of times before they serve. There is no logical reason for these rituals to have any impact—positive or negative—on performance. A lucky pen doesn't change how much you know and bouncing a ball four or six times won't change the trajectory of your serve. However, a 2018 study found that these rituals actually do decrease anxiety and improve performance even more effectively than other forms of distraction or attempts to calm down. Performing a simple ritual reduced not only reported feelings of anxiety but also measurable, physical symptoms of anxiety, provided that the ritual had a symbolic meaning for the person doing it. That is to say, they found that writing a test with a "special pen" won't make the test-taker any less anxious, but if the test-taker believes that they have a "lucky pen," then they are likely to feel less anxious than they otherwise would have.

Many other effective treatments for performance anxiety have already been found and continue to be researched, from medication to training in simulated situations. Through continued examination of the many factors that increase or mitigate performance anxiety—from the presence of other performers to audience layout, from early educational experience to comforting rituals—researchers will undoubtedly show that performance anxiety is more holistic than just being "all in your head." • • •

Harnessing the Bigger Picture

Treating Dyslexia by Improving Neuroplasticity

Written by Neil Ruthen
Illustrated by Cecilia Larson

The 21st century is an age of miracles for people with disabilities. Amputees can now purchase thought-controlled prosthetic limbs, individuals with color blindness can differentiate between a full spectrum of colors using special glasses, and organizations around the world are making public spaces more accessible for people with disabilities. Though out of the limelight, research related to learning disabilities has had an equally significant impact on individuals with dyslexia. By constructing the mechanism behind reading difficulties, neuroscientists have been able to collaborate with schools and technologists to create treatment programs for dyslexics.

Dyslexia is a learning disability defined by difficulty parsing words into phonemes (speech sounds) and deconstructing those phonemes into their component letters. The phonological impairment theory is the most widely accepted proposal for the neural mechanism underlying reading issues in dyslexics. Ultimately, this theory suggests that dyslexia is caused by a kink in the phonological processing system. By examining brain scans, scientists have been able to trace the neural pathway for the phonological processing system. First, letters must be identified in the visual cortex located in the occipital lobe. Then, the visual word form area must communicate with phonemic representation areas in the temporal lobe to connect letters to phonemes and phonemes to words. Finally, higher-order processing must occur in the frontal cortex for reading comprehension. All of the above steps of the phonological processing system must be performed efficiently for fluent reading. Therefore, reading takes a whole neural network: the coordination of visual processing, verbal working memory, and executive functioning. Persons with reading-based disabilities have a mechanistic problem in their neural network that prevents fluent reading.

Not all dyslexics have the same mechanistic issue, however: they can be divided into three groups based on where their kink lies in the neural network responsible for reading. Those with visual processing problems have slower reaction times, while those with verbal working memory problems usually have trouble holding verbal information in their heads. Those with executive functioning problems have trouble with compiling information sent from multiple regions of the brain. Researchers under Dr. John Gabrieli at MIT have found that the underlying problem for deficiencies in visual reaction time, working memory, and executive functioning relates to reduced plasticity. Plasticity is a measure of the brain's ability to learn new things. In the case of dyslexia, people have a harder time acclimatizing to repeated inputs—a process known as neural adaptation.

> Despite the difficulties associated with Dyslexia, dyslexics have an adaptive ability that neurotypical people have trained out of their brains.

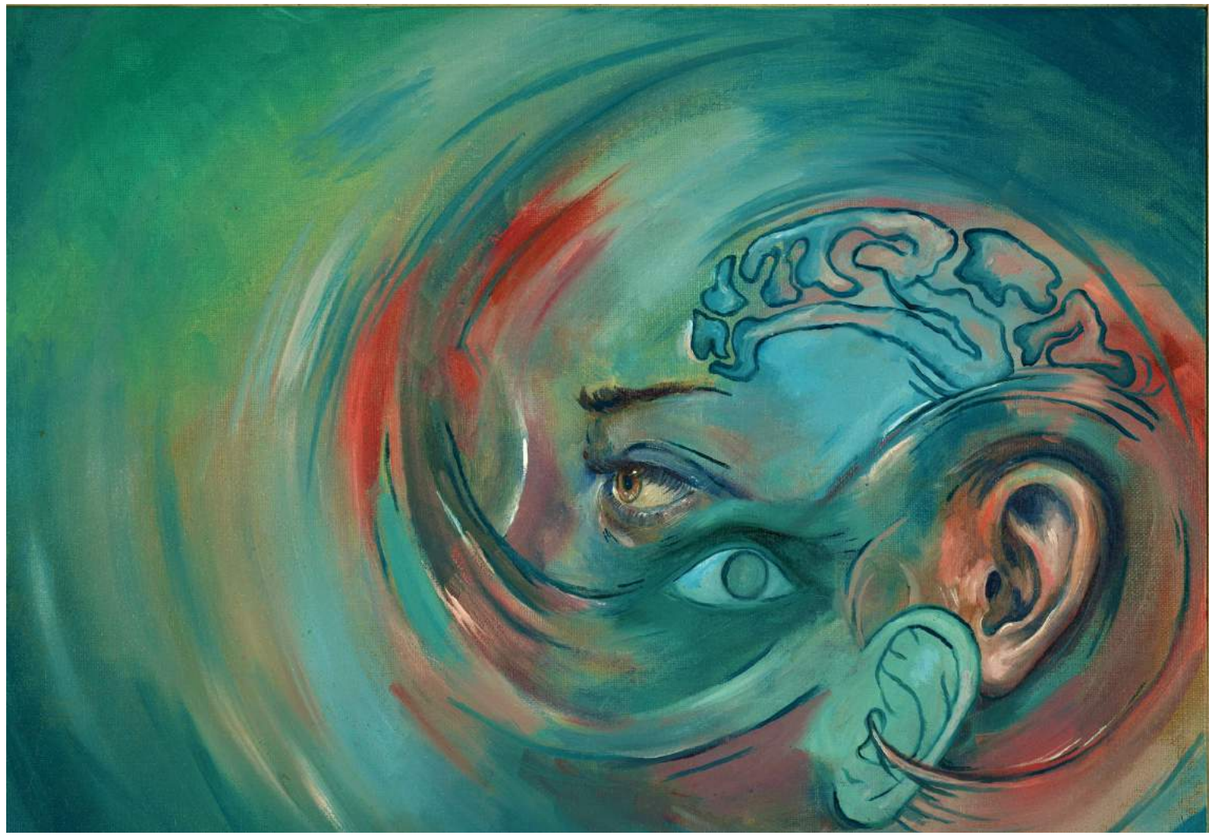
"You learn something upon the initial presentation that makes you better able to do it the second time, and the ease is marked by reduced neural activity," Gabrieli says. "Because you've done something before, it's easier to do it again."

In contrast to the way in which neurotypical people learn, dyslexics must relearn letters and words many times after they are first encountered. This phenomenon is caused by diminished, gradual neural-adaptive abilities in dyslexics. Neural adaptation is dependent on the myelination around a neuron's axon which, like insulation around a copper wire, allows the brain's electrical signals to flow more efficiently. Thus, even once the information is learned, a dyslexic will be slower at recalling that information or have a harder time keeping the information in their head.

To combat these difficulties, students who are diagnosed with dyslexia are exposed to the Orton-Gillingham teaching approach for struggling readers. Based on the phonological processing theory, this approach focuses solely on helping dyslexic students break down and build up words. A significant limitation to this approach is that it does not focus on reading comprehension, a skill with which many dyslexics struggle, so reading remains a constant struggle for dyslexic students even after developing Individualized Education Plans with a school psychologist. For students with learning disabilities, diagnosis is akin to a permanent sentence of adapting to their disability rather than a suggestion to seek further treatment. Problems such as slow development of vocabulary, poor motor coordination, and difficulty remembering letters or numbers only become more difficult for dyslexics to tackle as school expectations for reading fluency and comprehension increase. They often fall behind their peers within the confines of public schools without intervention.

Despite these difficulties, dyslexics have a quite adaptive ability that neurotypical, literate people have trained out of their brains. In a study of human cognition conducted by the University of Wisconsin-Eau Claire, a team led by Catya von Károlyi discovered that dyslexics are faster at spotting impossible figures and parsing optical illusions than the average human. In addition to this discovery, an investigation conducted by a laboratory at the Harvard-Smithsonian Center for Astrophysics suggests that dyslexics have an acute sensitivity to visual anomalies. Tasks which require attentiveness to all the details of a visual or auditory field are more easily performed by dyslexics. They have looser top-down filters—neural frameworks that match the sensory stimuli we perceive to our expectations—such that the percepts of a dyslexic are closer to reality than the percept of an average person, though such percepts are not as efficient. In the age of information technology, such visual acuity might be useful for parsing through data and lines of code. A meta-analysis of research on the advantages of dyslexia suggests that the very mechanism which prevents dyslexics from habituating to the patterns of written language is the mechanism which enables them to see a bigger picture.

The truth is, however, that dyslexics may not have to be dyslexic for much longer. Recent studies show that a tailored and effective treatment of dyslexia should move away from a narrow reading based approach and address the true problem: reduced plasticity in specific areas of the neural network for reading. These studies indicate that video games have the potential to counteract reduced plasticity in the brain. Instead of overtraining specific reading skills, video games exercise the neural pathways involved in reading and other activities for faster processing and better information retention. Researchers at the Carroll School have shown that video games which focus on improving reaction time, working memory, or fast executive-functioning improve dyslexic students reading fluency indirectly. Games such as timed sudoku and even virtual whack-a-mole have a marked effect on the processing speeds of the gamers. Over the course of several years, students at schools using computer-based brain training have risen into normal reading fluency percentiles. As technology continues to be integrated into the classrooms of public schools, video games for improving neuroplasticity might be available to students under the supervision of school psychologists. With the rise of online brain training, treatment for dyslexia might one day be a URL away. • • •



Did you Hear That?

An Exploration of Auditory Hallucinations in Schizophrenia Disorder

Written by Victoria Fisher
Illustrated by Aisling Smith

Have you ever heard your name when no one uttered a word? Or perhaps thought you heard a friend's voice in a crowded area when they weren't there? While these occasional auditory hallucinations are typical for many individuals, they become a constant and debilitating nuisance for those with schizophrenia.

For neurological disorders, "positive"symptoms induce certain behaviors, thoughts, or feelings, while "negative" symptoms reduce other behaviors, thoughts, and feelings. Schizophrenia is a neurological disorder characterized by negative symptoms such as loss of emotional capacity or mobility, and positive symptoms such as disorganized speech, delusions, and hallucinations—a defining trait of schizophrenia, as approximately 60-70 percent of those diagnosed will experience them. Auditory hallucinations are most frequently experienced, though visual, tactile, and even gustatory and olfactory hallucinations also occur. These hallucinations can have significant and detrimental effects on people's lives, because they are unable to properly understand the world around them. As with all disorders, in order to alleviate the burden of hallucinations, we must first understand what causes them.

There have been several proposed theories for the development of hallucinations. One theory is that those with schizophrenia rely on their expectations over what is actually occuring. When we perceive sensory stimuli, our perceptions incorporate a myriad of factors which are separated into two categories of processing: bottom-up and top-down. Bottom-up processing refers to components about the sensory stimuli themselves, such as their intensity, spatial relationship, and temporal relationship. Top-down processing refers to the modulation of our perception due to our own characteristics or experiences. Examples of this would be influences from our memory or emotional state, and the attention we give to stimuli. The influence of expectation in hallucinations would be reflective of individuals ignoring the bottom-up or objective features of stimuli and instead allowing their expectations to take precedence.

This theory surrounding the cause of hallucinations in schizophrenic individuals is supported by recent research. For instance, Albert Powers, a psychiatrist at the Connecticut Mental Health Center, and his clinical team published a study in 2017 that looked at how strong the influence of previous associations was on schizophrenic individuals' perceptions. In the study, participants with and without schizophrenia were trained to associate a flash and a beep together after exposure to both the stimuli repeatedly. The researchers then adjusted the environment such that only the flash was presented and asked participants if they heard a beep. While, initially, both sets of individuals heard the beep, over time those without psychosis were significantly less likely to report hearing the beep than those with schizophrenia. This discrepancy indicates that prior associations play a bigger role in the perceptions of schizophrenic individuals than in neurotypical individuals.

Another recent study further corroborates the involvement of abnormalities during top-down processing that cause hallucinations. In 2016, Jean-Paul Noel, Ryan Stevenson, and Mark Wallace lead a study focussed on the underlying mechanisms of sensory perception in individuals with schizophrenia. Neurotypical and schizophrenic individuals were exposed to temporally disparate simple-speech stimuli (i.e., a video in which the voice came before the mouth began moving) and were asked to assess if the voice and the mouth were synchronized. This type of procedure permits the analysis of the role of different modulations to sensory processing. Noel and colleagues found that schizophrenic participants were less able to accurately assess temporal disparities between the mouth and the voice than neurotypical individuals. This difference in performance could be attributed to alterations in prior likelihood to assume synchronicity (which for simplicity is shortened to "prior"). The prior acts as a proxy for the influence of higher-order processing in the top-down modulation of sensory perception. Thus, these findings further support the role that deviations in top-down processing, such as expectations, play in the sensory perception and hallucinations of schizophrenic individuals.

As with any neurological disorder, efforts have also been made to connect the associated symptoms with specific abnormalities in brain regions. While we may have evidence that expectations and top-down modulations play a role in hallucinations, there is more uncertainty about the parts of the brain that contribute to this atypical nature. This uncertainty is difficult to resolve, given the highly variable nature of different brain regions from one individual to another, as well as the intrusive and expensive nature of examining the brain. Nonetheless, a few brain regions are implicated in the hallucinogenic aspects of schizophrenia.

A recent review published by Patricia Boksa, who works in the Department of Psychiatry at McGill University, found that the brain area most frequently associated with hallucinations is the superior temporal gyrus—which includes the primary and secondary auditory cortices of the brain. These areas are respectively responsible for processing the frequency or pitch of auditory waves and localizing or analyzing complex sounds. Many studies have found that there is decreased gray matter in this gyrus of schizophrenic patients who have auditory hallucinations. Meaning, they have fewer neuronal cell bodies to process the sensory information that they experience. While these are relatively low-level areas of sensory processing, they still receive inputs from various higher-order areas of sensory perception.

There are also several other areas in the brain that are implicated in the development of hallucinations of schizophrenic individuals. Recently a large study found a reduction in the size of the dorsolateral prefrontal cortex—which is associated with higher-order processes, such as decision making, action planning, and behavior. This would indicate not only a decrease in the cell bodies of this area but also a decrease in connectivity to other regions of the brain. Finally, actively hallucinating schizophrenic individuals have increased activation in regions of the brain that are associated with speech and language perception. Overall, we see modulations and deviations in areas associated with a diverse range of functions and levels in the processing of auditory stimuli.

It would be a natural conclusion that medication used to decrease hallucinations targets the aforementioned parts of the brain; this is not the case. Typically, a type of medications called second-generation antipsychotics is used to treat disorganized speech, delusions, and hallucinations in patients with schizophrenia. These medications specifically target the pathway for dopamine between the nucleus accumbens and the ventral tegmental area. Dopamine is a neurotransmitter associated with movement, reward, and learning. The ventral tegmental area has neurons which extend into the nucleus accumbens. These neurons will typically release dopamine, which binds to many receptors. The binding of dopamine to receptor D2 is particularly relevant to schizophrenia, as antipsychotics are D2 receptor antagonists in that they inhibit the ability of dopamine to bind to this receptor. The use of antipsychotics significantly reduces hallucinations, indicating that these brain areas also play a pivotal role in integration despite the lack of research directly associating these areas with hallucination. However, second-generation antipsychotics are also associated with severe effects on metabolic functioning by increasing a patient's risk for weight gain, diabetes, stroke, and heart disease. Ultimately more research is necessary to improve current medications.

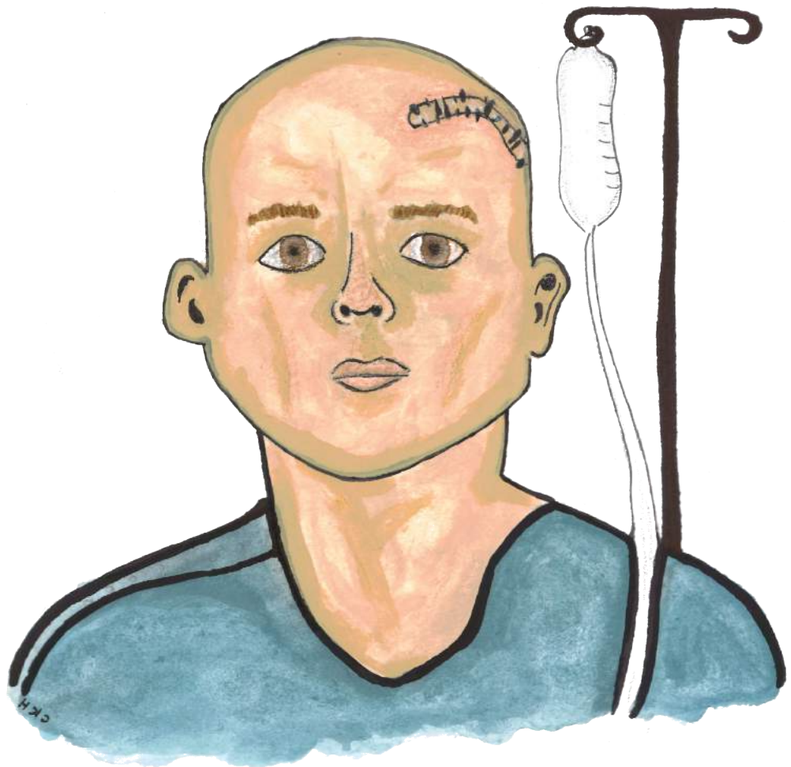
> While we may have evidence that expectations/top-down modulations play a role in hallucinations, what part of the brain is contributing to this atypical nature?

There is still much to learn about the functional cause of hallucinations in schizophrenic patients. While there is empirical support for certain theories and associated brain regions, each fails to provide a holistic picture of hallucinations. Given the complexity of the schizophrenia and the brain itself, it is not surprising that we have yet to determine the exact cause. That said, neuroimaging is improving as new technologies and techniques emerge with each passing day. And thus, with each passing day, we come closer and closer to understanding why hallucinations occur and what we can do to help those who experience them. • • •

Blast From the Past

The Effects of Battlefield Exposure on War Veterans

Written by Elizabeth Toigo
Illustrated by Claire Hoy



Many in this country have been affected by the unstoppable, horrific plague of war. Over 20.4 million men and women have returned from distant conflict, only to have their demons follow them home: nightmares that arise even during the day, flashbacks of battle from walking down an eerily quiet street, anxiety developed after being trapped in an environment with no sense of control. The appearances of unknown blasts during their military service can prove especially devastating to veterans upon return. But are these struggles considered mental health disorder, or injury, or both?

Battlefield-blast exposure due to improvised explosive devices is an extremely common cause of mild traumatic brain injury in war-torn areas, such as Iraq and Afghanistan. Following their return to the United States, war veterans commonly experience mental health problems. Approximately 31 percent of war veterans are diagnosed with post-traumatic stress disorder, 19 percent are diagnosed with traumatic brain injury, and 7 percent are diagnosed with both. Unfortunately, most scientific attention has focused on the moderate to severe end of the injury spectrum, not the mild TBIs which are the most common among returning veterans. Consequently, recent studies utilize rodent models in order to understand mild TBIs and potentially find a beneficial treatment. Researcher Perez-Garcia and his colleagues at the Veterans Affairs Medical Center in New York tackled this question by studying whether or not a drug called BCI-838 could reverse PTSD-related behaviors in rats exposed to low-level blasts mimicking explosions that cause mild TBIs in humans.

How exactly do you simulate such a scenario for research? Rats cannot be truly exposed to IEDs in a battlefield setting and later studied to see the effects, as there would be a large number of confounding variables. In order to recreate these effects under experimental conditions, the researchers gave the rats blast injuries with a shock tube. The rats' heads faced blast exposure without any body shielding, resulting in full exposure to the blast wave, once per day for three consecutive days.

The study also used four experimental groups: control, blast-exposed with no treatment, blast-exposed treated with a low dose of BCI-838, and blast-exposed treated with a high dose of BCI-838. The drug was administered orally for 60 days to mirror how a human would take a prescribed medication, starting two weeks after the last blast exposure. At the end of 60 days with drug administration, the rats went through a range of behavioral tests in order to analyze anxiety and other mental health symptoms that correspond with PTSD. During these behavioral tests, researchers monitored the rats' movements to either the opaque black side or to the illuminated side of a box, their movements in a circular maze with half enclosed by dark walls and half having no walls, their abilities to recognize new objects compared to familiar ones, their responses to an acoustic noise, and their development of fear to electrical shocks. Upon completion of testing, the rats' brains were processed in order to measure neurogenesis—the development of nervous tissue.

The study found that blast-exposed rats had fewer entries and traveled less distance on the light side of the box compared to the dark side, suggesting anxiety to novel and open spaces. However, treatment with the high dose of drugs reversed this effect, demonstrating the decrease in anxiety symptoms. Additionally, blast-exposed rats moved less, and spent less time in the open area, further exemplifying increased anxiety. Similarly, treatment with a high dose of the drug reversed this effect, supporting how the drug may be beneficial in combating anxiety symptoms. During fear conditioning, both high and low drug treated groups froze less when shocked. Blast-exposed rats also spent less total time exploring novel objects, but four weeks later, they explored the novel object no more than the familiar object during the novel object recognition test. Importantly, the results showed increased tissue growth in the brain following chronic BCI-838 administration in an animal model of blast-related TBI.

What do the results in this study truly tell us? How can we protect soldiers while they serve us and care for them after their service? These behaviors suggest that the main drug effect is not on fear memory but on how the fear response is maintained. However, treatment with both high and low doses reversed various effects, so the drug may be able to counteract memory impairment as well. Studies such as this one provide a model to study the chronic behavioral effects of blasts such as PTSD in former warfighters and implement a useful therapeutic model to ultimately improve life in this population. This research is critical, as it may lead to a treatment for veterans affected by mild TBIs.

Upon return to the States, veterans may experience chronic debilitating behavioral syndromes such as PTSD associated with blast mTBIs that persist long-term. The drug, BCI-838, can reverse multiple PTSD-related traits improving anxiety-related behaviors, fear responses, and long-term memory recognition in rodent models. This study highlights BCI-838, hippocampal neurogenesis and a specific pathway involved in antidepressant neurons (the Group II metabotropic glutamate receptor pathway) as potential leads in positive development of former warfighters suffering from PTSD symptoms. Thus, a new neural pathway emerges that may help unravel some of the questions we have surrounding PTSD in war veterans. Although this study found that the glutamatergic system is involved in this process, the pathway was not explored further. With these developments and continual efforts, war veterans may no longer have to stand alone. • • •

Dark Matter

An Invisible Universe

Written by Meaghan Accarino
Illustrated by Roger Ort

Stars, planets, black holes. These are the three main objects that we consistently see throughout the universe. The Milky Way contains about 250 billion stars—but that's just our galaxy. Astronomers estimate 200 billion to 2 trillion galaxies exist in our observable universe, including over 50,000 billion stars. Yet all of this material only makes up about five percent of the universe. Everything you see when you look at the night sky on a dark, clear night is only a fraction of what's out there. Dark energy and dark matter make up the other 95 percent of the universe. But what are dark energy and dark matter?

The answer is, we don't really know. Dark energy is a mostly unknown form of energy that repels matter, acts on a large cosmic scale, and is believed to cause the ongoing expansion of the universe. On the other hand, dark matter is a form of energy that attracts matter, acts on both a large and small scale, and is more directly implied by calculations from observable galactic activity (especially near black holes, another mystery of the universe). Dark energy is especially mysterious and difficult to study, but people are making significant headway in understanding dark matter.

Everything we know about the universe today is information we've gathered via the electromagnetic spectrum, or the range of waves that carry energy produced by electrically charged particles. For example, we observe black holes through radio waves and X-rays. However, dark matter doesn't interact with electromagnetic waves or light waves, which means that it is arduous for researchers to observe its activity. Any wavelength of light will pass right by dark matter without changing its path. As far as we know, dark matter only interacts through gravitational waves and energy, a completely different type of energy than electromagnetic energy. Logistically this means that scientists who study dark matter primarily use highly sensitive instruments to decipher absolutely miniscule changes in gravitational waves which are easily distorted in space. Though many scientists are trying, no one has actually succeeded in detecting dark matter yet.

> Because we had seemed to find dark matter everywhere, we could not be sure if it actually existed or if our laws of physics were deeply flawed.

So how do we know that dark matter is there? Scientists first discovered this mysterious material with so many unknown properties by studying the rotation of galaxies and the motion of stars within them. Researchers realized that the observed rotational speeds of galaxies were too fast for the galaxies to stay intact without some kind of internal, attractive force. The only explanation was that there must be some strange matter in galaxies that keeps them from spinning themselves apart.

In Albert Einstein's thought experiments about gravity, he discovered that masses bend the fabric of space-time. Imagine the Universe as a big, stretched out sheet. Stars and planets and black holes and any massive objects create a curvature in the sheet. This curvature is gravity. The nature of light is to travel in the straightest way across a curved path, meaning that as light comes across a massive object in space, it's going to travel in the direction which is the least amount of distance for it to traverse. This can happen in such a way that light will appear as a circle around a massive object or that a star can be seen at the side of one object even if the star is really directly behind the object. In this way, all matter, even dark matter interacts with light waves, if only through curving light's path with its gravitational pull—the only interaction of dark matter and electromagnetic energy that is known. While this property doesn't let us actually see dark matter, it does allow scientists to measure the amount of dark matter in a system. Tracking the curvature of light helps in measuring how much mass—visible or dark—is in a given galaxy.

Aside from its interaction with light, dark matter may also differ from normal matter in a more significant way: it's behavior with itself. Scientists hypothesize that dark matter is distributed almost evenly around the Universe, instead of behaving like normal matter, which aggregates into stars and planets. This speculation suggests dark matter to be diffused in individual particles. The most basic units of normal matter are protons, neutrons and electrons. A strong theory among astrophysicists is that dark matter is made of weakly interacting massive particles (WIMPs). These WIMPs could be ten to one hundred times the mass of a proton, but, because they are "weak," their interactions with normal matter are not easily detected. There may be alternate ways of discovering more about dark matter, if technological advancements can permit the detection of these "weak" interactions.

Another hypothesis is that dark matter is a different form of neutrinos, or particles that are produced from fusion in the sun. However, in an early experiment conducted by Raymond Davis and some of his colleagues, neutrinos were measured in only one-third of the predicted number. Many years later, scientists realized this occurred because neutrinos are able to switch between three different states, so the machines in the experiment only had a one in three chance of detecting neutrinos. Neutrinos are also special because they are not considered to make up regular matter. The proposed "sterile neutrino" would be a fourth state that only interacts with normal matter through gravity—just like dark matter—but this theory is a work in progress without much hard evidence.

Many scientists believe that dark matter is the scaffolding of the universe. Supporting this idea, most observed galaxies are at least 30 percent dark matter, but last year we discovered a galaxy that appears to contain almost no dark matter. This galaxy is named NGC 1052-DF2; it is very dim, about one-two-hundredth the mass of our Milky Way galaxy, and about 65 million light-years away. The reason this discovery was so bizarre is that a galaxy as isolated as NGC 1052-DF2 is expected to have an almost one hundred times greater amount of dark matter as normal matter. It also rocked the theory that all galaxies must have dark matter. In response to this discovery, scientists may need to reconsider how galaxies are defined.

The discovery of this galaxy is also exciting because, until now, dark matter was everywhere that scientists had looked in the Universe. Dark matter's ubiquitous presence created some uncertainty about the legitimacy dark matter. Because we had seemed to find dark matter everywhere, we could not be sure if it actually existed or if our laws of physics were deeply flawed. Since a galaxy completely void of dark matter has been discovered, there is more certainty of its existence. Now we have a prime example of a galaxy that exists and that could have formed without dark matter.

In spite of numerous theories, discoveries, and research efforts, dark matter remains one of the most puzzling aspects of the universe. Scientists have made incredible progress in unearthing the secrets of dark matter, but modern science promises to explore even more. • • •

Half-Rations

Written by Max Robinson
Illustrated by Steven Mentzer

Twice as salty! Unbelievable. How could half-rations possibly be twice as salty?

"Is the food always this terrible?" Isaac bleakly inquired. He leaned in, inspecting the contents of the tray. The scoop of grey-green mash had been bisected lengthwise and neatly tucked into the top left corner of his flat, grey lunch tray. It was parallel to a thin cut of oily, iridescent binomial. A viscous liquid that was not quite juice or blood sloshed around the tray.

"Sometimes food is good, sometimes it isn't," Biggs said, bringing his mess tray down onto the table. "But if there's one lesson I'd try to get into that thick skull of yours, it's that food is food."

"I'd hardly call this food," Isaac mumbled, moving a half of mash across his tray with a flat utensil.

"Give it another segment. You'll be thanking the stars themselves for anything The Chef puts in front of ya."

Another segment? How could he last another segment? After seven span off course and five at half-rations, whatever meager comforts he had aboard the Arcturus seemed to be slipping further away each day. It wasn't even that difficult of a life (Biggs would say, "No marque means no fightin'," whenever the topic came up), but the monotony of it all was like sandpaper on the mind.

"You know, I don't know how The Chef did it," Isaac mused over another mouthful of mash. "A week of trying to kill us through starvation, and all he's managed to kill is my appetite."

"Could be worse," Biggs said between sips of strong water. "We could be on quarter-rations."

"They could just put us on no-rations and save us the misery." Isaac took a long swig from his glass, which seemed just enough to help water down the metallic, saltier-than-usual aftertaste of his biomeal. "Do you think that's their plan, have us voluntarily starve ourselves out?"

"Damned if I know." Biggs quipped back "but if this is part of some revenge scheme a thousand years in th-"

"I-HOPE-YOU-FIND-THE-FOOD-TO-YOUR-LIKING-GENTLEMEN."

"Darn it," muttered Briggs under his breath.

"Mess Sergeant!" Isaac exclaimed, turning around to find the hulking mass of the Arcturus' head chef. Standing upright at just under seven kebit tall and two and a half thick, their dull, grey body rattled and hummed with archaic energy. Although common law prevented The Chef from manning any essential stations, having a "thinky" act as a cook was a popular method of employing such dangerous crew.

"IF-THE-FOOD-IS-NOT-TO-YOUR-LIKING-GENTLEMEN-I-ASSURE-YOU-THAT-THE-GALLEY-IS-OPEN-TO-ANY-SUGGESTIONS-YOU-MAY-HAVE-TO-IMPROVE-THE-QUALITY-OF-YOUR-MEAL."

"You know what…" Isaac attempted to say between bites, trying to calculate how quickly he could bolt out the door, "The food is great. Really great, and my only real complaint is that the captain seems adamant on us getting so little of it. Darn shame really, the captain is usually so understandi-"

The Chef brought one of his arms down on the table with a sharp crack.

"NONSENSE-GENTLEMEN-THERE-IS-ALWAYS-ROOM-FOR-IMPROVEMENTS-AND-AS-THE-FORMER-PERSONAL-CHEF-OF-THE-LATE-EMPEROR-DARIUS-I-AM-AN-EXPERT-IN-PREPARING-DISHES-CONTAINING-ALL-VARIETIES-OF-EXOTIC-INGREDIENTS." The Chef leaned in closer to the now adequately frightened Isaac. "PERHAPS-YOU-WOULD-LIKE-TO-TRY-ONE-OF-MY-SPECIALTIES ..."

The Chef paused for dramatic effect and positioned their "face" a few inches away from Isaac's.

"CREAM-OF-MIDSHIPMAN"

"Chef!" Barked a harsh voice from the threshold. "Why doncha leave the poor kid alone and go check on yer staff."\

"YES-CAPTAIN" The Chef buzzed, withdrawing from the confrontation. Captain Gregor entered the galley with his telltale step, thunk-chik, thunk-chik, thunk-chik. The various augmentations and prosthetics on his body made him look lopsided as if he was leaning just a bit to the left, but his ramrod posture and subtle grace dispelled any notions that the captain was not in total control of his bulky, cumbersome form.

"Midshipman, fancy meetin' you here at this hour."

"This is when I normally eat my meals, Captain."

"I know," Gregor said, turning to scan the room, perhaps literally. "Rigger, why doncha go check in on engineering. I asked Halsy for a report on the fuel supplies a half-span ago, and I still haven't 'eard back from her."

"Yes, Captain," Biggs mumbled, already halfway out the door before the order was finished. Isaac noticed that the usual commotion that inhab /Users/stevenmentzer/Downloads/Untitled_Artwork 2.png
ited the Arcturus had all but ceased. He and the captain were the only two souls left in the galley.

Gregor took a seat across from Isaac and began to study the young man sitting across from him.

"Tell me, midshipman, how are ya likin' the ship?"

"It's been good," Isaac said slowly, thinking through each word carefully.

"Seein the sights?"

"I found Heckam to be…" Isaac thought back to the dusty port they had picked up provisions almost two cycles ago. It had squat buildings and squat people, milling about a station that Isaac would generously call seedy.

"I didn't see ya at the Siren's Arms wit the rest 'o the crew," the captain interrupted.

"Oh- I…" Isaac stammered "I actually got a bit sidetracked. One of the longshoremen sold me a deck of Hesian playing cards for a few dracha, even taught me a few card games for a halfdra more."

"Mhmm…" The captain hummed, before abruptly calling out, "Chef! The good stuff!"

"RIGHT-HERE-SIR," The Chef whirred, appearing suddenly behind the captain. A glass of amber liquid was poured in front of the two privateers before Isaac and Gregor found themselves alone again.

"Is that the good stuff?" Isaac asked.

"It is, it is…" Gregor said while taking a long, slow, sip. "But sometimes, ya start to miss the bad stuff." Gregor paused and went back to studying the midshipman.

"Why didja want to join me crew?"

"I'm sorry sir?"

"You had a good life back home. Food, family. That's more than most here. What made ya decide ta give that all up?"

"I wanted to become something," Isaac said, shifting uncomfortably under the line of questioning.

"Yer a good man Isaac," Gregor paused to take another drink, this time from Isaac's glass. "But this ain't a line o' work that takes to kindly to good men," Gregor paused as if to let the words linger in Isaac's mind. "The void isn't a kind place to us Isaac. If ya stay in it too long, she keeps ya, trapped in the cold. And she took me arm. And both of me good legs!"

Gregor took another sip of Isaac's liquor, finishing the clear liquid in a single gulp.

"Is it worth it?" Isaac asked

"Oh, it's worth it. That's how she gets ya." Gregor began reaching around in the pockets of his long coat. "Tell me, Midshipman, what's your schedule lookin' tomorrow?"

"I take watch at 08:00, meal at 12:00, and R&R until second watch."

"Mhmm…" the captain hummed, somewhat disinterested in his own query, until he finally pulled out a gausspistol and placed it gently on the table in front of Isaac. "We make planetfall at 05:00, and I'm in need of an extra pair a hands," He motioned toward the firearm, "should ye be interested."

Isaac hesitated, before reaching for the handgun and holstering it on his belt. "And what exactly am I going to find on-planet?"

The captain laughed, a hearty, metallic laugh, and began making his way out the door. "If I knew midshipman, I wouldn't need those extra hands, now would I."

His footsteps grew more muffled, *thunk-chik, thunk-chik, thunk-chik, thunk-chik, thunk-chik*. And then silence. • • •

Chistopher Howard

Interview by Victoria Fisher
Photos Provided by Chris Howard

Christopher Howard is an assistant professor at Oberlin College as well as the faculty advisor for The Synapse board at Oberlin. He completed his undergraduate and graduate work at The Univisierty of Illinois. He then moved to La Jolla, California where he completed his post-doctoral fellowship at the Salk Institute. Since coming to Oberlin, Professor Howard has taught five different courses at Oberlin, including upper level Neursoceince Courses: Neurophysiology, Studies of Neural Functions, and the Addiction Capstone. Professor Howard's research at Oberlin focusses on dopamine signaling in the brain. We at The Synapse are excited to introduce you to this wonderful professor!

This interview has been edited for length and clarity.

When did you begin your interest in neuroscience?

Great question. So, I have two answers. The first is that I have had three grandparents in my life that were diagnosed with Alzheimer's Disease. So, that instilled in me a deep interest in how the brain works. My grandfather is of particular note, he was a musician. He was a composer and would play piano. Even deep stages into Alzheimer's disease he could flawlessly play the piano, which was phenomenal – it was incredible. It was clearly a conscious brain that was disrupted in Alzheimer's, and then there's this motor brain that totally intact even deep into this disease. So that's the first answer, right? That's what first gave me the interest. The second thing was, I sort of ended up in the field on accident.There was a fellowship, and I applied for it, and I got it.

Was this where you developed your interest in dopamine research?

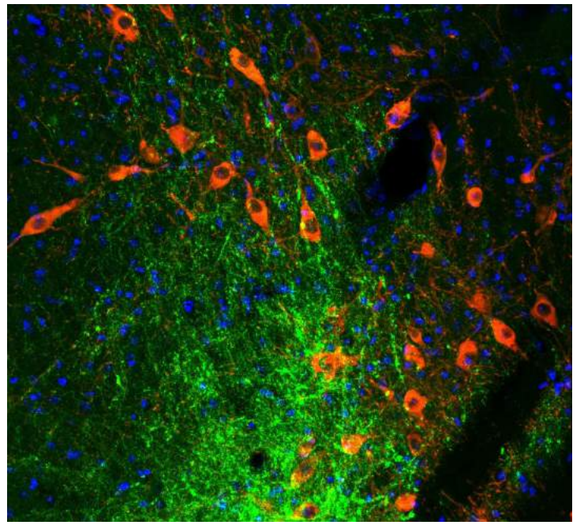
I began in my future graduate advisor's lab. So, I started in that lab. I liked the work they were doing, and I was kind of okay at it. So, he offered to give me a position. I figured, if I'm going be here, might as well get a Ph.D. and see what that is all about. And that's kind of what happened. [My lab at Oberlin] is still interested in dopamine, but we come from a physiological background than a behavioral background focus.

Could you discuss what you're currently researching?

We have currently three main projects in the lab. We're interested in how dopamine supports the formation of habits. We have two types of behaviors. There are goal-directed behaviors, for instance I pull a lever and I win some money. If I stop winning money, I no longer pull the lever. Or you have habitual behaviors, where you pull the lever and, regardless of the out come, you're going to continue to pull it. Habits have a bad connotation and clearly there I brought in a gambling analogy. Habit circuits underlie a lot of disease states including addiction, OCD – obsessive compulsive disorder, Tourette syndrome, and other non-drug related addictions.

So, we're interested in how dopamine facilitates or supports habit formation. What's known is that you need dopamine to form habits. If you take drugs of abuse, it exacerbates habit formation. We are now using optogenetics, which is the light activation of dopamine terminals, to see if we can facilitate habits to develop faster than they would typically form. So that's project one.

Project two, within the same area of brain where dopamine neurons project to, the striatum, there are these patchy areas of different receptors and different markers. The patches have been known about for 50 years. They are incredibly cool looking structures (*see photo below*). Most brain structures are bilaterally symmetrical, and they're the same more or less between individuals. Patches don't follow that arrangement. So, these patches are different between the left and right brain hemispheres and radically different between



Above is an image taken in Professor Howard's research lab at Oberlin College. This is an image of the substantia nigra in the midbrain of a mouse. The green indicates the dopamine receptor patches which project from the striatum to the substantia nigra. The orange are the dopamine neurons that are interacting with the dopamine receptors.

individuals, which I think is super cool. Despite the fact that patches are so salient and prominent, the role for patches has not been understood for about 50 years. They've been known about for 50 years, but no one knows what they really do. So, the field is ripe for investigation. We are now using the same sort of habit strategy to determine if we kill patches, can animals form habits? If we activate patches, do animals form habits faster or slower? So, that's what we're focused on.

The third project involves dopamine neural transmission. This is me returning to my roots as a graduate student – it's all I ever did. We evoke dopamine responses and then we can apply drugs – many different classes of drugs – to determine how that modulates dopamine neural transmission. So it's really pharmacology in its purest form. But we are specifically looking at melatonin, a neural hormone, to see if that changes dopamine signaling. And we're moving into – and I never thought I'd do this – we're moving into endocannabinoids too. So, I just got a small grant to study Cannabidiol (CDB), which is all the rage in the world right now.

Could you talk about the endocannabinoids and CBD?

This is probably common knowledge to most of the readers of *The Synapse*, but the industry is exploding right now with both recreational marijuana and this drug CBD or cannabidiol, has been added to a number of consumer products. Carl's Jr. now has a CBD burger. What is not known is what CBD actually does to the brain. We know that it binds to CB2 receptors, which are expressed in glial cells. They might also be expressed in some other neurological systems. It's likely that it is modifying brain activities. So, is it a good idea to distribute a drug that's not well known across the entire American population? Probably not.

You know what? Now that I've start talking about it, an interesting thing that I've found is that Carl's Jr., a fast food restaurant, can put CBD in its burger, but CBD is technically a Class 1 restricted substance. You know it doesn't get people high. So, me, as a neuroscience researcher, I have extremely restricted access to this chemical. There's some loophole, which I can't figure out. I am not allowed to order CBD from the NMH unless I apply for a schedule 1 license. Other schedule 1 licenses include THC, PCP. Methamphetamine is a schedule 2 substance – it's less tightly regulated than CBD. Explain that to me!

What impact does your research have on drug addiction?

Drug addiction is a multifaceted disorder. So there are sociological factors, for example. That's the only thing we don't talk about in my senior seminar course - although it is important. Habit is one of those factors. When people are returned to one of the cues for taking drugs they are more likely to fall back into those deeply ingrained habituated behaviors. So if you associate a drug with a place and you go back to that place, the craving for the drug will often present itself. How much habit plays a role in that is somewhat debated in the field, but it definitely is a factor. So, understand the neural mechanism of habit is a piece of the addiction story.

Turning the subject to your role here at Oberlin, how is research and teaching at Oberlin different than at other institutions.

I think research is the primary thing I can talk about here. When I was an undergraduate, I was at a school with graduate students and post-docs. 80-95 percent of the research that is done there is done by graduate students and post-docs. Undergraduate students can help, they can come along to see how things work, but they're not the main drivers of research. Obviously, that's not the case here at Oberlin. I would say 90 percent of the work that gets done in my lab gets done by students. So I think we interact in a really tight-knit way, that students may not get at a larger university. The opportunity is here for great research.

Do you think that relationship is also in the non-research aspects of the academics at Oberlin?

Yes. What I try to do, particularly in my teaching lab, is to give students to explore research techniques on their own terms. Students do their own projects in my teaching lab. In addition to that, I think we all expect a lot from the students because they are really getting these good experience here.

Do you have any advice for students who want to become involved in the physiological aspects of neuroscience?

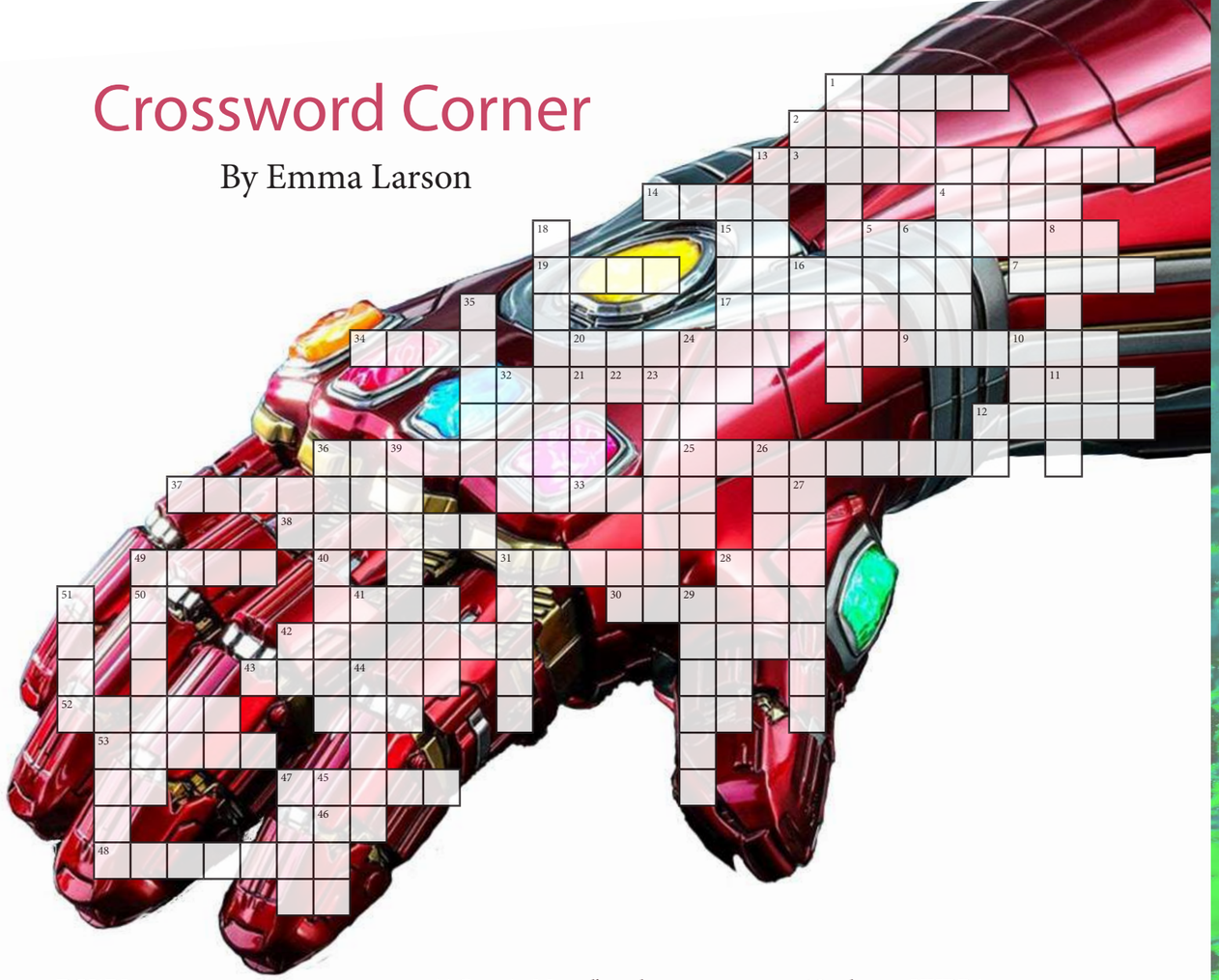
Well, I think my advice would be general. Always work hard. Don't get over-involved. That's one of the things that both faculty and students fall into at Oberlin. It's great to have lots of opportunities, but it's better to excel in the opportunities you have. That's something I tell all of my advisees too, but I'll reiterate here. Another thing is, get into research. Get into research anyways you can - whether it's on campus, off-campus, or just during summer. Those kinds of experiences will inform you if this is the career you want or the career you don't want - which is sometimes better to find out.

Have their been recent discoveries in your field that have been particularly cool?

Yeah! Last week someone brought a pig brain back to life. Did you hear about that? There's a paper from last week. They took a brain that was dead for four hours and profused it with life-giving who-knows-what substance. They recorded neural activity in it. Now I'm not going to speak to the meaning of it, but I found that particularly interesting. Other big findings in the field? I think are really cool are the technical advances. There's an interesting debate about whether it's techniques that drive hypotheses or ideas that drive hypotheses. In the field, a lot of time the techniques advance first, and the hypotheses follow accordingly. There are a lot of great sensors that are being developed that we are hoping to use in my lab eventually. Thinking about other big findings - it seems like there's a big finding every week. Science is happening so rapidly. It's hard to keep up!

Crossword Corner

By Emma Larson



DOWN

1 teleportation cube
5 Mystic Arts master and librarian
8 savoir of the universe
10 taken from Thor by his evil sister
13 Vistacorp Robinhood
15 the purple infinity stone's aspect
18 Iron Man finally tries this
21 the stone guarded by Red Skull
23 also known as Legolas
24 location of the Soul Stone
26 founded by Peggy after WWII
27 project to study the Tesseract
28 the Winter Soldier
29 the brains, not the brawn
31 teen superhero recruited by Stark
32 Sokovian responsible for Civil War
35 type of radiation that made the hulk
39 Asgard's doomsday
40 Star-Lord
44 Captain Marvel's real name
46 first to know Spider-Man's identity
47 physicist and Thor's ex
50 reptilian aliens from Skrullos
51 Captain Marvel's nickname
53 blue aliens from Hala

ACROSS

1 Thanos's home planet
2 Thor's evil sister
3 people of asgard
4 Director Fury
6 Stark's right-hand man
7 he has sensitive nipples
9 Spider-Man's main neighborhood
11 Peter's cool aunt
12 there's proof that Tony Stark has this
14 Thor's father
16 I am...
17 hidden world on Earth
19 salute to HYDRA
20 Earth's first superhero
22 god of thunder
25 Jarvis's adress to Pepper
30 Gamora's cyborg sister
31 Captain America's beloved
33 god of mischief
34 HYDRA scientist under Schmidt
36 ruler of the Dark Dimension
37 king of Wakanda
38 queen of Asgard
41 Banner's love interest
42 allows travel to the Nine Realms
43 the next Captain America
45 tried to start rebellions on Sakaar
48 scientist that gave serum to Steve
49 the Abomination, fought the Hulk
52 Wakanda's tech genius
53 arms dealer that sold vibranium

Meet *The Synapse*...

...at Oberlin

Pictured from left to right, back to front: Emma Larson, Rebecca Fenselau, Steven Mentzer, Miranda Marnik-Said, Victoria Fisher, Evelyn Morrison, Rachael Branscomb, Yue Yu

...at Denison

Pictured from left to right: Casey Pearce, Kileigh Ford, Elizabeth Toigo, Delaney McRitchie

/syn . apse/ noun : the point at which a nervous impulse passes from one neuron to another.
The Synapse is an undergraduate science magazine that serves as a relay point for science-related information with a threefold objective. First, we aim to stimulate interest in the sciences by exposing students to its global relevance and contributions. Second, we work to bridge the gap between the scientific and artistic disciplines by offering students a medium through which to share their passions, creativity, and ideas. Third, we strive to facilitate collaboration between undergraduate institutions across the country, especially within the natural science departments.