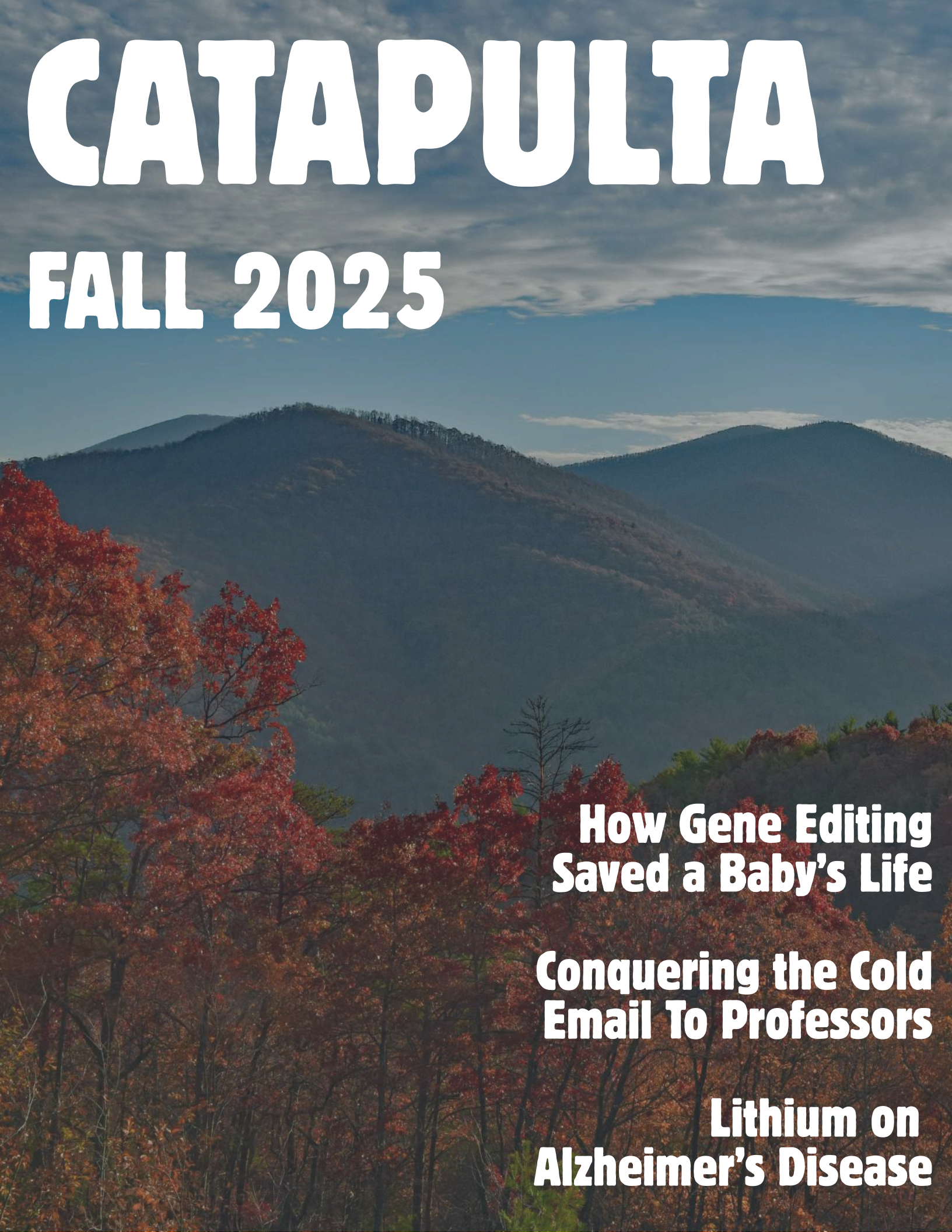
CATAPULTA

FALL 2025



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WELCOME TO CATAPULTA'S 2025 FALL ISSUE!

AS THE SUN SETS EARLIER AND THE LEAVES SLOWLY BEGIN TURNING SHADES OF RED, ORANGE, AND YELLOW, THE COLD WINDS WHISTLE IN THE TRANSITIONAL SEASON OF FALL. THE NEW SCHOOL YEAR USHERS IN A PERIOD OF RECONNECTING WITH FRIENDS, DEALING WITH FOREBODING DEADLINES, AND PULLING NUMEROUS ALL-NIGHTERS TO COMPLETE THAT ONE ENGLISH ESSAY. WHILE OUR BLS COMMUNITY MAY BE UNDER A LOT OF STRESS, WE HOPE THAT THIS NEW MAGAZINE WILL PROVIDE THAT MUCH DESERVED BREAK WHILE LEARNING ABOUT SCIENTIFIC BREAKTHROUGHS!

THE NEW BOARD HAS BEEN WORKING HARD TO BRING YOU THIS NEW ISSUE. FROM "THE FUTURE OF GENE THERAPY [IN VIVO]" (PG. 9) TO "THE MATTER OF LITHIUM ON ALZHEIMER'S DISEASE" (PG. 14) TO "THE EVER-CHANGING EIFFEL TOWER" (PG. 7), CATAPULTA STRIVES TO DELIVER THE NEWEST, CUTTING-EDGE SCIENCE IN A FUN AND INFORMATIVE WAY. MAKE SURE TO ALSO CHECK OUT OUR NEW SECTION (PG. 10) WHERE WE SHARE ESSENTIAL ADVICE ON HOW TO COLD EMAIL YOUR WAY INTO MEANINGFUL SCIENTIFIC OPPORTUNITIES. HAPPY READING!

SINCERELY,

Angelina and Evan

THIS ISSUE OF CATAPULTA HAS BEEN MADE POSSIBLE BY THE BOSTON LATIN SCHOOL ASSOCIATION AND OUR FACULTY SPONSORS:

MR. GALEGO
MRS. BATEMAN

FALL ISSUE STAFF

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RED-40

Scanning the snack aisles of a grocery store, your eyes may dart to the vast selection of intensely colorful packaging: chips, cereals, gummies — the harsh overhead lights of the supermarket illuminate the blazing red glare of your favorite prepackaged foods, urging you to pick them up. These treats are reliable — their red coloring indicates the vibrant flavor you love, so you know exactly what you're getting each time you purchase these favorites. Or do you?

The petroleum-based dye Allura Red AC (Red-40) is infamous for its presence in products like drinks, snacks, clothing, and makeup. Red-40 can last for an extended period of time, which appeals to mass producers. Many consumers, however, disregard the often severe implications Red-40 can have on digestive health and neurological well-being.

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A study, published by the National Library of Medicine in 2016, estimated the average exposure of food dyes across the United States and found that 100% of Americans over the age of two consume Red-40 across a two-week period. The trial, recording data on the consumption of food over two weeks, was meant to measure the chronic exposure that Americans experience to the color additive.

When Red-40 is consumed, the body views it as a foreign substance, which can trigger an immune response in the digestive tract. With the consumption of Red-40 being so high in the United States, many Americans' digestive system can become chronically inflamed.

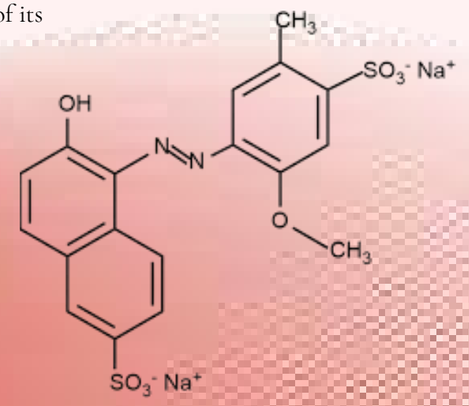
A recent 2023 finding2 saw a correlation between early-onset colon cancer and the rise in Red-40 consumption. Such persistent inflammation due to repetitive exposure to Red-40 can lead to colorectal carcinogenesis, when cells in the colon and rectum undergo genetic changes and eventually become cancerous.

Further, Red-40 consumption, paired with a high-fat diet, reduces the variation of microorganisms, or alpha diversity, and the changes in the composition of the microorganisms, or beta diversity, in the gut. The gut's microbiome is crucial for digestion, and changes to alpha and beta diversity of its microorganisms can increase the risk for colon cancer.

Beyond the digestive system, food dyes impact behavior. A review in 20223 examining clinical trials on the correlation between consumption of food dyes like Red-40 and changes in adolescent behavior found that 64% of the trials saw a positive correlation.

This change in behavior can present itself through hyperactivity and lack of focus. Because the nervous system doesn't fully develop until an individual reaches their mid-20s, children are more susceptible and sensitive to the dangers of Red-40 consumption.

With Red-40 being so widely-available, it's nearly impossible for consumers to avoid Red-40. Researchers, however, do agree that consuming Red-40 within the recommended daily intake of 7 mg/kg is safe. In any case, it's important for Americans, especially adolescents, to be aware of its prevalence as well as its potential wellness impacts.

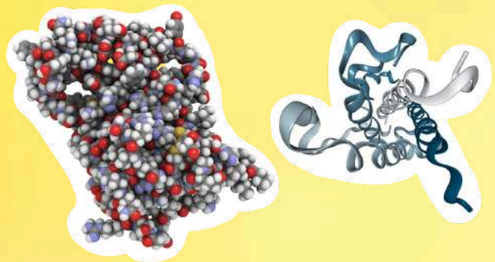


BEYOND GENETICS: HOW SLEEP, DIET AND EXERCISE AFFECTS YOUR HEIGHT

Scientists from Tufts University have determined that 60-80 percent of human height is determined by genetics. There are, however, many factors outside of your parents that play a role too. Namely, how much protein you consume, how much HGH (human growth hormones) you secrete, and how much you sleep. There are countless other factors too since height is a polygenic trait—a characteristic influenced by two or more genes.

HGH (human growth hormone) is a peptide hormone made from amino acids. Amino acids are compounds that are found in foods like meat, fish, and eggs. These are proteins, which are made from amino acids. HGH has various functions, such as promoting gluconeogenesis, a process that breaks down proteins to produce glucose and height and causes the release of another hormone called IGF-1. HGH eventually acts on the epiphysis, which is the part of the bone that is elongated, leading to growth.

HGH release is closely related to sleep hygiene. According to J. R. Davidson and other researchers at the Toronto Western Division of the Toronto Hospital in Canada, sleep deprivation will result in HGH release being nullified on the night of sleep following deprivation. HGH does not actually increase epiphyseal growth directly: it is part of a feedback loop with the aforementioned hormone IGF-1 (Insulin-like Growth Factor 1). When HGH is released, it acts on the liver to release IGF-1, which more directly stimulates epiphyseal plates by promoting the cells of the bones called chondracytes to enlarge in a process called endochondral ossification, which advances bone formation. In fact, Professor at Masaryk University P. Grasburger's 2016 paper shows that higher protein intake positively correlates with an increase in height since the hormones responsible are made from amino acids, derived from proteins.



Pictured: Chemical Structure of Human Growth Hormone
Left: Space-filling, all atom representation
Right: Ribbon representation of protein

Another key factor contributing to height growth is exercise. Exercise increases HGH not only in the short term but also increases the amount of HGH released over a 24 hour period, especially if the exercise is intensive. It is, however, important to keep in mind the complexity of the matter, as frequent running can lead to blunted HGH release over time in response.

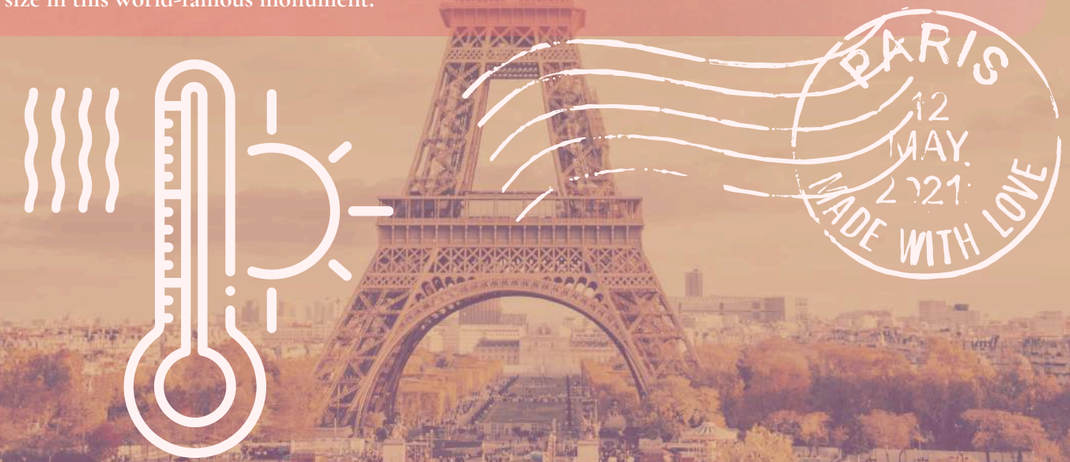
It must be emphasized that the interactions between hormones, cells, genetics, and the environment is an extremely intricate process that is constantly being studied with new findings. Youn Jee and her team from the National Institute of Child Health and Human Development describe this phenomenon perfectly, calling height a "black box", highlighting the complexity of human nature.

THE EVER CHANGING EIFFEL TOWER

We all know the Eiffel Tower. It's the iconic Parisian landmark, the centerpiece of tourist activities. But did you know that its height actually changes? According to La Tour Eiffel, the structure was originally 300 meters tall (984 feet), and, with TV and radio antennas, it's only gotten bigger. But aside from the antennas, the tower itself is also changing in size. Thanks to seasonal temperature fluctuations, the metal structure expands and contracts with the weather. The Eiffel Tower is made of puddled iron, a version of wrought iron, which is an iron precursor to steel that contains small amounts of carbon. The process of creating puddled iron has four key steps: first, melting, in which pig iron—an iron made directly from smelting raw iron with high carbon-dioxide levels—is heated up in a furnace; second, stirring, where a person known as a "puddler" constantly stirs the melted pig iron; third, oxidization, which involves oxidizing impurities and removing them from the metal; last, shingling, where hammers roll the doughy metal mass into shape.

The puddled iron of the Eiffel Tower weighs a total of 7300 tons. Because of thermal expansion, all 7300 tons of puddled iron expand when the temperature increases—higher temperatures mean higher kinetic energy of the iron atoms, leading to the expansion of the metal. The annual temperatures in Paris are sometimes unpredictable, but according to Paris Discovery Guide, summer temperatures generally stay around 70 to 80 degrees Fahrenheit. The warm heat causes thermal expansion in the puddled iron, and the Tower can sometimes grow about 15 centimeters taller. In the winter, with colder temperatures of 20 to 30 degrees Fahrenheit, the Eiffel Tower shrinks about 15 centimeters down to its original height.

Thanks to the properties of puddled iron, everyone can observe this phenomenon of ever-changing size in this world-famous monument.



THE FUTURE OF GENE THERAPY

Currently, the majority of genetic diseases have no available treatment options. Thus, scientists are using a revolutionary technology, gene therapy, to develop a cure for them. Genes come from our parents and decide everything, from how we look to how likely we are to develop diseases. They consist of four chemicals called bases, which contain instructions for making proteins, the building blocks of the human body. When a gene mutation causes a protein to be built incorrectly, gene therapy can restore the protein.

In gene therapy, disease-causing genes can be replaced with healthy or inactivated ones. Alternatively, new genes can be introduced into the body to treat the disease. Gene therapy includes gene editing and base editing. The first is used to precisely add, remove, or alter specific parts of DNA. During gene editing, scientists cut the specific spot of the DNA that is malfunctioning, allowing the cell to naturally repair itself, replacing the cut DNA. Disrupting unwanted parts or inserting new parts of DNA leads to permanent genetic changes, fighting against genetic disease. Base editing, on the other hand, targets single-point mutations, which are genetic alterations in a single DNA base, by creating precise changes in the genetic code to inactivate disease-causing genes or activate other genes to help treat the disease. In base editing, double-stranded DNA isn't cut, but, instead, an enzyme is inserted into a DNA sequence to change the base on one strand. Then, cellular repair proteins recognize the change in the base and convert it into a permanent mutation, resulting in the curing of diseases caused by those mutations.

In gene therapy, genetically engineered vectors are used to deliver genetic material into the body. Viruses that are modified to not be infectious, meaning that they cannot replicate, are used as vectors because of their natural ability to infect target cells. This, however, creates risks. The body's immune system might see the vectors as intruders. It may attack them, therefore, causing adverse effects ranging from swelling to organ failure. Additionally, viruses can affect more than one type of cell, so the virus vectors may affect other cells unintentionally (off-target effects). Furthermore, the vectors can introduce errors in the genes, possibly leading to cancer. Moreover, even though the viruses are modified to no longer be infectious, once they enter the body, they may be able to once again cause diseases.

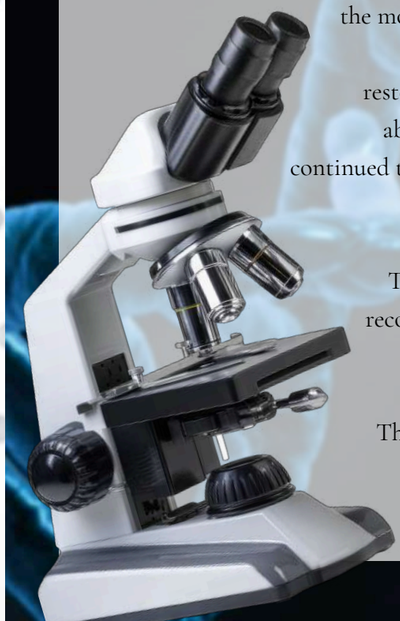
Ultimately, gene therapy is an advanced technology that could be the future of curing currently untreatable diseases. While it hasn't yet been fully developed, many scientists are working to improve it and are hopeful in its potential to change the lives of the many who suffer from these diseases.

How Gene Editing Saved a Baby's Life

Earlier this year, scientists reached an amazing milestone. They used Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) gene editing to save the life of a baby.

This past February, a team at the Children's Hospital of Philadelphia (CHOP) and Penn Medicine treated a 10-month-old infant, nicknamed KJ, who had carbamoyl phosphate synthetase 1 (CPS1) deficiency. When a person lacks CPS1, their body cannot break down proteins properly. This leads to a buildup of excess amino acids and toxic ammonia in the bloodstream and brain. Without treatment, this can result in brain damage or even death. Using recent research developments from UPenn along with help from scientists at the Innovative Genomics Institute (IGI), the team at CHOP and Penn Medicine designed a personalized in vivo (inserted directly into the body) CRISPR therapy that repaired the faulty gene inside KJ's liver cells.

This treatment was historic because it marks the first time that doctors used in vivo gene editing to save a human life. KJ had spent nearly his entire first year hospitalized on a strict protein-restricted diet. Usually, a case like this would call for a liver transplant. KJ, however, was too young to undergo such a major procedure, forcing the doctors to look for a different solution. So, the research team, led by Dr. Rebecca Ahrens-Nicklas of CHOP and Dr. Kiran Musunuru of Penn Medicine, developed an N-of-1 therapy — a treatment designed for one specific patient. The treatment was delivered through an IV infusion and involved sending an mRNA package coding for a modified CRISPR/Cas9 base editor (a gene-editing tool that changes one DNA base to another) directly to his liver, where the defective gene was active. There, the mRNA instructed the liver cells to produce the modified protein. A custom-designed guide RNA was used to direct the base editor to the location of the mutation in KJ's DNA. It corrected the faulty DNA base, restoring the function of the CPS1 gene. Within weeks of KJ's first treatment, he was able to consume more protein in his diet without accumulating toxic ammonia. KJ continued to safely receive treatments over the next few months, helping him better process the protein and return his ammonia levels back to normal.



The potential benefits of this breakthrough go far beyond just one individual. KJ's recovery shows us what might be possible with patient-specific CRISPR treatments. Scientists believe a similar approach could help people with other rare ailments, too, such as muscular dystrophy, cystic fibrosis, or even some types of cancer. Though KJ is just one baby, the possibilities of CRISPR technology could very well help millions.

CONQUERING THE COLD EMAIL

Especially in our increasingly interconnected digital world, cold emailing has become an essential part of networking, initiating conversations, building connections, and uncovering untapped opportunities. Cold emailing, in a high-school science context, means sending emails to professors or scientists whom you have not talked to before for insight on a topic, for research opportunities, or even mentorship. Here are the key steps and strategies for crafting a compelling cold email.

1. Research your Receiver

Before sending the email, take the time to research the recipient. Understand their interests, background, projects, and publications; this knowledge will allow you to customize your email so that it aligns with their interests, resulting in a captivating message.

2. An Eye-catching Subject Line

The first thing that appears in your recipient's inbox will be the title of your email; it is the initial impression. Keep it brief, but engaging. It needs to attract their attention, leading them to open your email.

3. Add Personality

Most emails sent to them will be generic and lack the personalization necessary to prove that you have done your research. To have an outstanding email, this is where you add the information you have dug up about them in step one: say their name or state some knowledge you learned about from their research papers.

4. Keep it Short

Make sure that your email is clear, concise, and straight to the point; those researchers are busy people. Include a clear goal, whether it is introducing yourself, proposing a collaboration, or presenting a solution to their problem.

5. Contribute

If possible, add specifically what you can help out with in their lab. Ultimately, most researchers will be unwilling to take on a trainee if they are just going to be a burden. Can you utilize your skills to help do elementary tasks in their lab space? Even if you are unable to directly contribute to their research at the moment, you can still highlight that you are willing to put in the effort to learn new skills to be able to help out. Self-learning new material beforehand is also not a bad idea.

6. Highlight your accomplishments

Although not necessary, you can include relevant accomplishments, honors, and experiences you have received in the past to build credibility. Do not worry if you do not have any, as most researchers do not take this into heavy consideration, unless they are specifically about research (for example, if you received honors in olympiads or conducted a pure math project when you are applying for a wet biology lab, do not even bother adding those). They are looking for potential and a good academic mindset.

7. Follow Ups

Many times, you will not receive a response as your recipients are most likely extremely busy, but do not be disheartened. They potentially are swamped with work or have not had the chance to read your emails, so try and send a follow-up after a couple of days. This will prove your motivation and investment in this opportunity.

8. Test and Change

Try different approaches, subject lines, and email bodies. Analyze the results to see what works best. Over time, you will refine your cold emailing strategies and achieve better outcomes.

9. Again, do not be surprised by the lack of responses

Again, do not fret if most of the researchers do not respond. They either did not see your email or used silence as a polite rejection. This is normal. Oftentimes, people can send up to hundreds of cold emails before getting a response or acceptance, but this is just the process. However, on the bright side, it only takes one good offer, and you have succeeded.

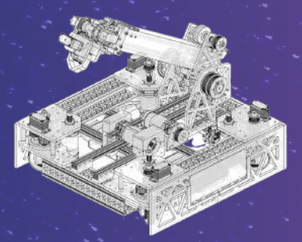
Cold emailing stands as a vital asset in your skills, as it unlocks possibilities that you have only dreamt about. If you ever need help in writing these emails, do not be afraid to reach out to the Catapulta email, as many on our officer board have done this process in the past. Your next scientific opportunity could be one email away.

Club Feature! Isotech 23461/BLS Robotics

We are Boston Latin School's newly founded robotics team, Isotech 23461. We compete in the FIRST Tech Challenge (FTC), where high school students design, build, and program robots from the ground up. Founded in 2023, it aimed to fill the gap of engineering opportunities at BLS when the FIRST Robotics Competition team the 125 NUTRONs relocated from Northeastern University (a 10 minute walk from BLS) to Revere High School (a 45+ min drive). The NUTRONs are the 2001 FRC World Champions, 2023 World Finalists, and multiple time NE District Champions. Before 2018, BLS students were able to play a pivotal role developing the NUTRONs robot, but their new location makes it hard for any BLS student to commit time. BLS Robotics strives to bring those missing engineering opportunities back to BLS through the smaller FTC format of competitive robotics. At Isotech, instead of the traditional adult/college student mentor, students serve as mentors of each other.

Events:

- **CAD Workshops**: Teaching CAD skills using Onshape to BLS students
- **Community Exhibits**: Showcasing Isotech and FIRST robotics program to the public in the Greater Boston area at events such as Boston Dragon Boat Festival, MassRobotics Robot Block Party, and BLS Friends of Chinese Culture Events.
- **Summer Camps:** Isotech students serve as camp counselors at NUTRONs 125's summer STEM camps.



TO LAUNCH A COASTER

HOW COASTERS GET WICKED FAST WICKED QUICKLY

On March 15th, 1997, the roller coaster Superman: The Escape officially opened to the public at Six Flags Magic Mountain. Boasting a 415-foot (126 m) vertical spike, it was also the first ever roller coaster to break the 100 mile-per-hour barrier. A feat like this could not have been accomplished with only gravity, however. Just before this giant tower, there was a long straight track. This track contained electromagnetic fins that propelled the ride to its top speed in about 7 seconds. This type of launch is called a linear synchronous motor system (LSM). These launches work by using electromagnets powered by alternating currents that rotate the magnetic field. The magnetic field rotation is synchronized with the magnetic field on permanent magnets attached to the train, creating thrust and launching the roller coaster.

Despite its popularity, there are other ways to launch roller coasters. Before coaster manufacturer Intamin used LSMs on Superman and its sister coaster Tower of Terror at Dreamworld, Premier Rides used linear induction motor (LIM) magnets to launch their coasters. The first LIM coasters, Flight of Fear at Kings Island and Kings Dominion, also used electromagnets powered by AC power, a slightly different application. When the electromagnets on the track switch polarity, they create loops of electric current, or eddy currents, on fins made of aluminum (or another metal) on the train. The train, by design, lies slightly behind the field on the track, creating propulsion as the fields repel. This launch is rarely used nowadays, however, because of the energy cost and lower efficiency of eddy currents. Its use of cheap aluminum and simple operation, however, keep it alive on 27 coasters globally.

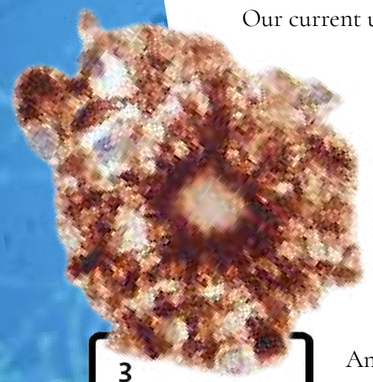
Magnetic launches are by far the most common modern-day launches, but there are many other launch systems. The first launch coasters, made by Arrow Dynamics (also known for making the first steel tube track, the basic shape of steel coasters) and Schwarzkopf, used primitive systems. Arrow used an electrical winch system, where a cable was wrapped around a push-car. When the launch was activated, the winch would wind the cable in, slowly pushing the push-car and train forwards. Schwarzkopf used a weight-drop system, where a large 40-ton weight would be dropped, pulling a cable connected to the train. This was used in coasters such as King Cobra at Kings Dominion, and MonteZOOMa's Revenge at Knott's Berry Farm.

Continuing Kings Dominion's streak of new launch systems, they opened HyperSonic XLC in 2001. This ride used a compressed air launch system. Air is pumped into a tank (usually built within the structure of a large "top hat"), raising the pressure inside of it. Inside an adjacent tank is a piston, with a cable connected to a push-car. When the pressure is released, the piston goes up, pulling the cable and the push car. This method is ridiculously powerful, creating some of the fastest launches of all time. At Fuji-Q Highland in Japan, S&S built the coaster Dodonpa (the name cannot be directly translated, but the word "dodon" comes from onomatopoeia for the sound an explosion makes). This coaster launched riders at a speed of 106.9 mph (172 km/h) in just 1.8 seconds. It, however, was actually shut down a couple of years ago due to reports of back injuries.

One final launch system is arguably the greatest. This launch system powered not only the fastest coaster of all time, but also the two tallest coasters. The hydraulic launch. The first one, Xcelerator, was built at an Anaheim theme park (Knott's Berry Farm, again). The launch uses very similar technology to the compressed air launch, but instead of air, it utilizes hydraulic fluid. The release of pressure pushes a winch and cable attached to a push-car. This technology launches both the tallest coaster in the world (Kingda Ka) and the fastest coaster (Formula Rossa).

For most of roller coaster history, rides were propelled by gravity. A chain would pull a train up a wooden structure, and the coaster would slowly roll down the hill. While this method still has its place in modern day coasters, newer magnetic launch systems are arguably the most influential innovation since steel coasters themselves were invented.

The Matter of Lithium on Alzheimer's Disease



Alzheimer's disease (AD) is a progressive neurological disorder commonly present in the elderly. Our current understanding of the disorder suggests that buildup of amyloid-beta (Aβ) plaques — protein fragments that build up between nerve cells in the brain — impairs cognitive functions, leading to symptoms like worsening memory and language deterioration. Recently, a study published by the scientific journal Nature by Aron et al. examined the impact of Lithium (Li) levels in brain tissues from AD patients and in the brain of an AD mouse model, and found that a loss of Li homeostasis — the regulation of Lithium — in the brain represented an early-onset incident towards the pathogenesis (development) of AD.

Analytical chemistry tools to measure the elemental composition of cells — namely, inductively coupled plasma mass-spectrometry (ICP-MS) and laser absorption plasma mass-spectrometry (LA)-ICP-MS — aided Aron et al in their research. They discovered that Aβ plaques from human brain tissues were highly abundant with Li-ions compared with plaque-free brain tissues. This finding was further supported by an animal model, where Li was sequestered by transgenic mice. When mice were fed with a Li-deficient diet, an acceleration of cognitive decline and an increase in Aβ deposition in the brain were observed. On the contrary, treatment with a Li salt, or lithium orotate, decelerated AD pathological alteration and memory loss in both AD and wild-type (normal) aging mice. In the article, Aron et al. concluded that endogenous Li in the brain may play an important physiological role as a neuron protective agent, and the imbalance of Li homeostasis represents an early event in the development of AD.

Our common wisdom recognizes the Li-ion as a key ingredient in the battery and energy industry, but the findings by Aron et al. open up a new avenue of research, which could potentially lead to breakthroughs in the development of diagnostic techniques when monitoring the early onset of AD in aging populations and novel medical treatments for AD patients. With only two antibody medicines, Decanemab and Donanemab, yet approved by the US Food and Drug Administration as treatments for slowing down human AD progression, many questions remain: what is the next cure for AD? A Li salt may be the answer!

The Lost Sámi Languages of Finland

Sámi, also known as Saami, is a language originating from the northern Scandinavian peninsula, a region in northern Europe which includes Sweden, Norway, and Finland, among other countries. It consists of a total of 9 surviving dialects out of 13. Many of the surviving dialects, however, are still at risk of extinction: a language is extinct once all of its native speakers have died, and most of the dialects have fewer than 1000 native speakers, with one dialect, Ter Sámi, consisting of only 2 surviving native speakers. Sámi has roots in the Uralic language family, which consists of languages such as Hungarian, Finnish, and Estonian. The Uralic languages were born out of the original Proto-Uralic language first spoken in Western Siberia, which then spread closer to the Ural Mountains. Dialects of Proto-Uralic languages then spread across Scandinavia and Eastern Europe, eventually reaching northern Scandinavia. This would later become the basis for the modern Finnish and Sámi languages.

It also did not help that Sámi was a hard language to learn – most of the dialects were complex in nature, with some having as many as 14 grammatical cases.

Research has been done to try to recover aspects of the Sámi language, which has been largely successful, yet it has not been able to fully replicate the nuance of the language itself. As it is right now, the lost Sámi languages will stay lost, at least for the next 50 years. The four extinct dialects are among the many languages that have been lost to time.

Akkala Sámi, Kemi Sámi, Kainuu Sámi, and Gävle Sámi were all once dialects of Sámi spoken in Scandinavia. Out of those four, however, the scientific world only has knowledge of three. Even out of those three, only Akkala Sámi has any surviving passive speakers — people who can understand the language when it is spoken. Most of these declines were due to the forced assimilation of many of the native Sámi living in Finland, leading to a loss of language and culture. It also did not help that Sámi was a hard language to learn — most of the dialects were complex in nature, with some having as many as 14 grammatical cases. Research has been done to try to recover aspects of the Sámi language, which has been largely successful, yet it has not been able to fully replicate the nuance of the language itself. As it is right now, the lost Sámi languages will stay lost, at least for the next 50 years.

PUZZLE

1. Gene editing technology that saved baby KJ's life

2. Toxic substance that builds up in CPS1 deficiency

3. Element linked to Alzheimer's disease prevention

4. Protein deposits found in Alzheimer's patients

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