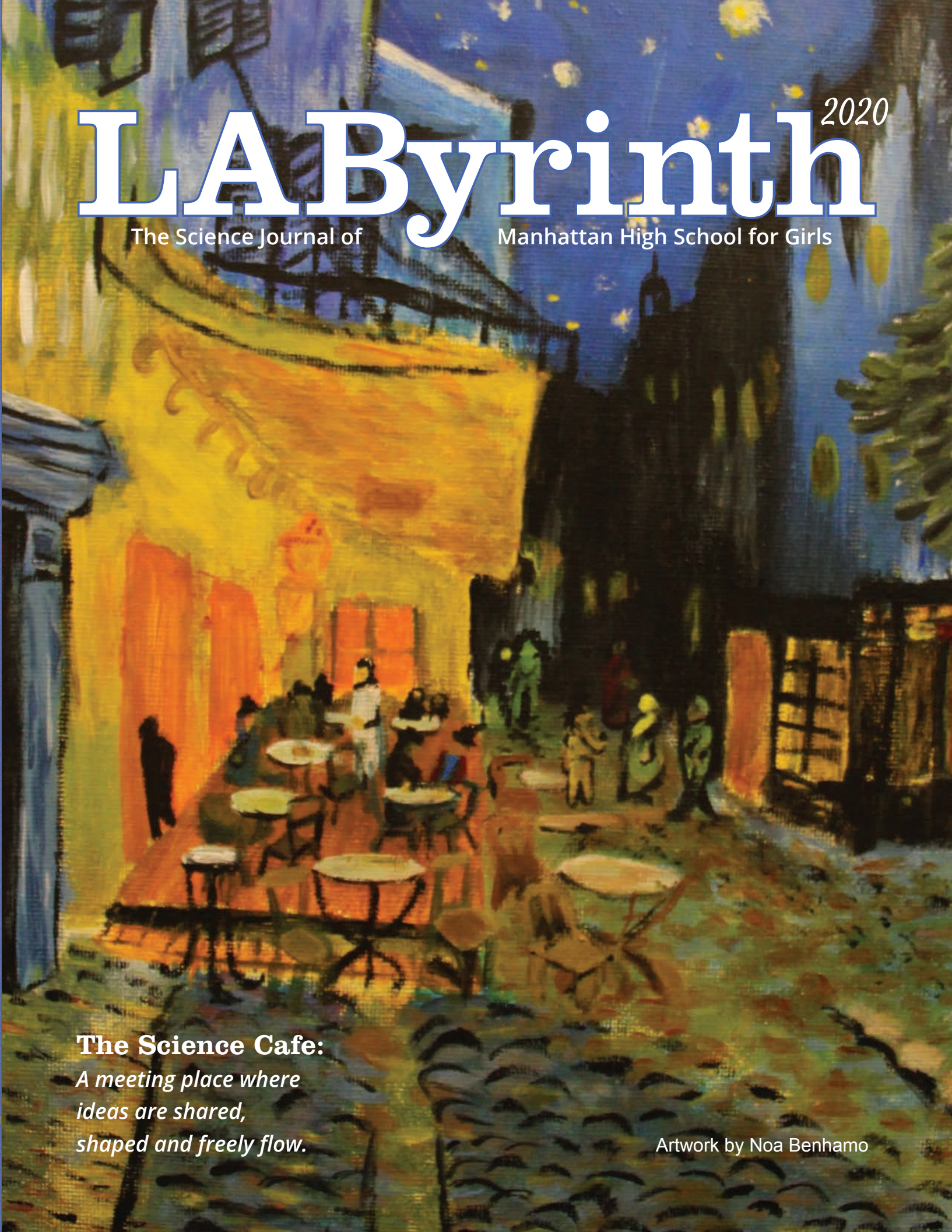


LAByrinth

2020

The Science Journal of Manhattan High School for Girls

The Science Cafe:
A meeting place where ideas are shared, shaped and freely flow.

Artwork by Noa Benhamo

LABYRINTH 2020

The Science Journal of Manhattan High School for Girls

LAByrinth 2020

The Science Journal of Manhattan High School for Girls

We are pleased to grant this year's

HARRY KAPLAN SCHOLARSHIP AWARD
FOR EXCELLENCE IN SCIENCE WRITING

to

SARAH STRAUSS

for her essay

"A MOMENT IN THE SUN"

and to

CHANA LIPSCHUTZ

for her essay

"THE PLASTIC BRAIN"

We gratefully acknowledge Dr. Tuckel and family for their generous sponsorship.

LABYRINTH 2020: FOREWORD

Mrs. Brenda From, Chair, Department of Science

What a remarkably historic year! COVID and Zoom learning have not dampened our spirit; on the contrary, we have admirably soldiered on with steadfast resilience, good cheer, and an infectious can do spirit.

What you hold in your hands is the product of several months' collaborative effort by the science department together with the Graphic Arts, Technology, and ELA departments. Twelve articles were culled from submission from 10th grade Chemistry (Shani Brody, Adina Hoffman, Rikki Klein, Tziporah Pinczower) and Advanced Electives: Genetics (Michal Englander, Sarah Strauss), Neuroscience (Chana Lipschutz, Leah Harris, Sima Leah Mandelbaum), Forensics (Ariella Shloush, Musia Kirschenbaum) and the Jerusalem Science Contest (Dassi Mayerfeld). It was a difficult choice. The topics are as diverse as the interests exhibited by the students, but the one feature they all share is a commitment to communicating science with authenticity, clarity and passion.

Thank you to my colleagues, the teachers of the science department who directly guided the students in this project: Ms. Judy Alper-Tenth grade chemistry and Neuroscience; Dr. Abigail Haka-Forensics; Mrs. Sara Tendler-Genetics. Special *Hakarat HaTov* to Mrs. Dena Szpilzinger, who with her characteristic skill and wizardry at all things related to technology and desktop publishing, stepped in at the 11th hour to produce this beautiful publication. As always, kudos to the intrepid team leaders of our school, Mrs. Yanofsky and Mrs. Friedman-Stefansky for their unwavering support of excellence in education in all disciplines.

A word about the cover. It is Noa Benhamo's rendition of Van Gough's Café Terrace at Night, produced under the creative guidance of Mrs. Nehorai. No, dear reader, I have not gone off my rocker due to caffeine withdrawal from my daily dose of double espresso as Mrs. Friedman-Stefansky had originally feared. (For more on that, read the article by Tzipora Pinczower, "Addiction Fiction.") I looked at it and it whispered in my ear, "What a perfect metaphor for this entire LAByrinth project." Good ideas no longer come from lone geniuses working in a lab; rather more often they come from the foment of ***interactions*** between creative minds. Just think of what the coffeehouse did for the Age of Enlightenment. Discussions originating from coffee houses prompted both the American and French Revolutions.

"If you look at the long view, the good ideas that underlie most of the great changes in our society—that have driven progress—more often than not actually have roots in the open kind of information commons of the university or the British coffeehouse. … In those environments, ideas are free to connect with each other and build on top of each other. That remixing is really where great ideas happen."

Steven Johnson, Where Good Ideas Come From

ACKNOWLEDGEMENTS

Mrs. Estee Friedman-Stefansky, Principal, General Studies

Covid-19 has hijacked us with an avalanche of questions and speculations and has brought science conversations into our homes in a new unprecedented way.

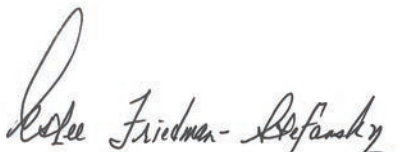
We have always known that science holds more questions than answers. Since the beginning of time, that black hole has lured many restless minds and from its murkiness so much meaningful meaning has surfaced. We now experienced that hole firsthand as individuals and as a nation. Science matters, and for us here at MHS, nurturing the life of the questioning mind matters.

Our science teachers condition our students to see the stature of a question. Our students learn how to craft a quality question by accessing their existing knowledge in multiple disciplines, by studying the answers that scientists have supplied in the academic literature, and by probing the question that persists personally for them.

This journal holds a collection of our students' questions about natural phenomena spanning from marine biology to genetic diseases and manufactured foods. I am so grateful to our teachers for inspiring our girls to see that science needs them — both for their questions and beH for their answers. I am proud of our faculty who create an environment that not only respects students' questions but also encourages them.

Congratulations to Mrs. Brenda From for another year of exceptional growth in our Science department and to the dedication of our terrific teachers: Dr. Abi Haka, Ms. Judy Alper and Mrs. Sara Tendler. Our science teachers continue to contribute to our school's ethos of achievement.

While The One Above has all the Answers, we offer our gratitude to Him for blessing our girls with healthy minds that can wonder, synthesize, evaluate and infer. May they continue to question and may their questions contribute to the betterment of healthcare for all of humanity.



FRANKENFOODS

Shani Brody, 10th Grade

The knee jerk response of most people to the term "genetic modification," is one of ghastly horror — keep them out of the pantry at all cost. Yet, we live in a society where genetically modified products are found lurking in almost every aisle of our supermarkets.

Before we pass judgement on this issue, it is necessary to understand what genetic modification is and how it affects the food that we eat.

Genetic Modification is a unique set of gene technology that is able to alter the DNA in living organisms, such as plants or animals (4). Genetically engineered foods have had their DNA adjusted using genes from other plants or animals. Another name for organisms which have undergone this process is GMOs, genetically modified organisms (2). Although genetic modification is now a conventional practice and introduces many benefits to both the consumer and producer, its use is very controversial. There are many advantages to genetic modification, as well as risks. The research of this paper will extend to the history, benefits, risks and the science behind the process of genetic modification.

The buildup to the discovery and production of commercial genetically modified crops spanned more than half a century. In 1928, Fredrick Griffith's experiment laid the groundwork for the development of commercial genetically modified crops. Griffith was studying two specific strains of Streptococcus pneumoniae. The two strains varied in both their appearance and their virulence, the ability to cause disease. The S strain was highly virulent and had a smooth capsule, while the R strain was nonvirulent and had a rough surface, lacking a capsule. When injected with the S strain, the mice would die within a few days. Through many experiments, Griffith understood that by heating the bacteria, the virulence of the S strain would be destroyed. He found that when the heat-killed S bacteria and the R bacteria was combined and injected into the mice, both of which would not cause the mice to die when injected alone, causing the mice to die. By isolating the bacteria that killed the mice from within the hearts of the dead animals, he observed that the mixture of the two bacterias had adopted the trait of the S bacteria, having smooth capsules. Based on this, Griffith concluded that a certain chemical component from the S cells transformed the R cells into a more virulent S form with a smooth capsule. Unfortunately, Griffith could not identify the "transforming principle" that gave the S cell the ability to transform other cells.

Based on Griffith's experiments, in 1944, Oswald Avery demonstrated that it was DNA that has the ability to transform the properties of cells. They identified DNA as the "transforming principle." Until then, biochemists had believed that deoxyribonucleic acid was a relatively unimportant, structural chemical in chromosomes, but they now understood that DNA was the agent that could produce an enduring, heritable change in an organism (12). After this, several other discoveries paved the way to the advancement of genetic modification. In 1954, Watson and Crick discovered the double helix structure of DNA, and the "central dogma – DNA transcribed to messenger RNA, translated to protein – was established" (6).

The first ever genetically modified crop was produced in 1983, using a tobacco plant and Petunias. Fraley, Rogers and Horsch from Monsanto generated transgenic petunia plants resistant to the antibiotic, kanamycin. Then, Chilton and her team succeeded in the insertion of the kanamycin resistance of the

petunia into tobacco (11). In 1994, the genetically modified "Flavour Saver Tomato" was approved by the Food and Drug Administration for marketing in the USA. The modification allowed the tomato to put off ripening after the initial picking and helped contain its freshness. Nowadays, there are a plethora of genetically modified food species and crops which are displayed in our supermarkets. Examples of common genetically modified foods include cotton, soybean, canola, potatoes, eggplant, strawberries, corn, tomatoes, lettuce, cantaloupe, and carrots (4).

So how is this process done? How is DNA transferred and introduced into a new species? In simple terms, gene transfer technology is a sophisticated version of a cut-and-paste operation. The two organisms involved in this process are the initial organism that is modified and introduced to a new trait, and the organism in which a desired trait is removed in order to transfer that trait into the initial organism. The first step of this process is to identify the desired trait. Comparative analysis is used to decode what part of an organism's DNA includes the desired trait. This comparative analysis is an item-by-item comparison of the DNA in the desired cell to determine which one contains the suitable and coveted trait that is wanted to transfer into a new cell. Once the location of the gene coding for the trait is identified, the next step is to isolate the gene to transfer the DNA into the initial organism.

The two most widespread and effective methods for this are Microparticle bombardment and Agrobacterium-mediated transformation. As shown in Figure A below, Microparticle bombardment uses 'gene guns.' In 1987, Klein and colleagues discovered this method of shooting a desired gene into a cell. This is done by coating the surface of microscopic gold particles with hundreds of copies of the desired DNA. These gold, DNA- coated particles are used as 'bullets' which bombard the initial plant tissue via the 'gene gun'. The transferred DNA now becomes stably integrated into the DNA of the initial plant, and the plant then translates it and expresses the new gene as if it were its own. Over time, this results in offspring with those desired traits. Other scientists take advantage of the natural bacteria, called *Agrobacterium tumefaciens.* The agrobacterium cell contains a bacterial chromosome and a tumor inducing plasmid called "Ti Plasmid." A plasmid is a DNA molecule that is separate from the chromosomal DNA, located in the cytoplasm. In this Ti plasmid is T-DNA, which is a DNA portion of the Ti plasmid.

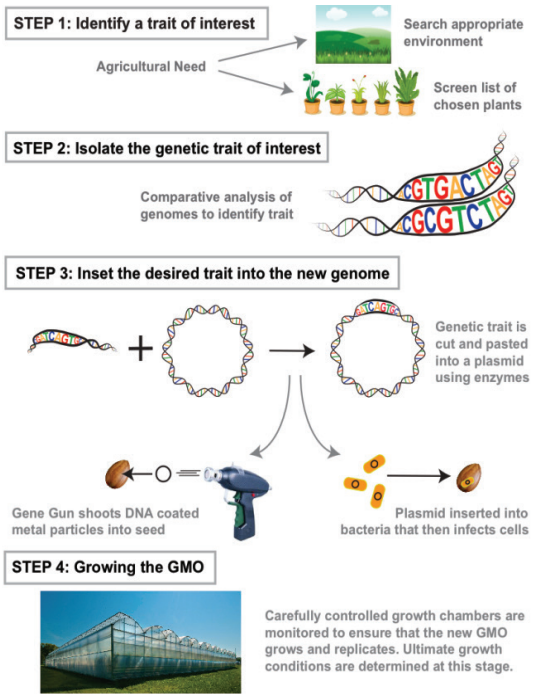


Figure A describes the process of the Microparticle Bombardment Method via "gene guns." As explained in steps 1 and 2, once a gene of interest is identified and isolated, the gene gun gets coated with the gene of interest. The trigger of the gene gun is pulled and the desired DNA bombards the crop and stably integrates into the DNA of the crop (5.)

As shown in Figure B below, this plasmid is now removed from the agrobacterium cell and the T-DNA is fragmented by an enzyme. Next, the desired DNA gets inserted into the T-DNA. The T-DNA now contains the

desired gene. The modified plasmid is then re-inserted into the agrobacterium and the bacterium then naturally invades the initial plant's genome and inserts the T-DNA into the cell. The initial cell now contains the desired DNA (10). With this, the desired DNA is transferred and now offspring these desired traits into new generations.

Genetically modified plants have offered many benefits to our society. Scientists are able to manipulate plants by inserting desired genes, which offers innovative solutions to prevent different issues within a plant. The benefits include a higher nutritional value, less insect damage, fewer pesticides, less toxins, cheaper crops, faster growth, better taste, *increase* of shelf life, and the ability to harvest seasonal crops all year round (1).Genetically engineered foods are reported to be high in nutrients and include

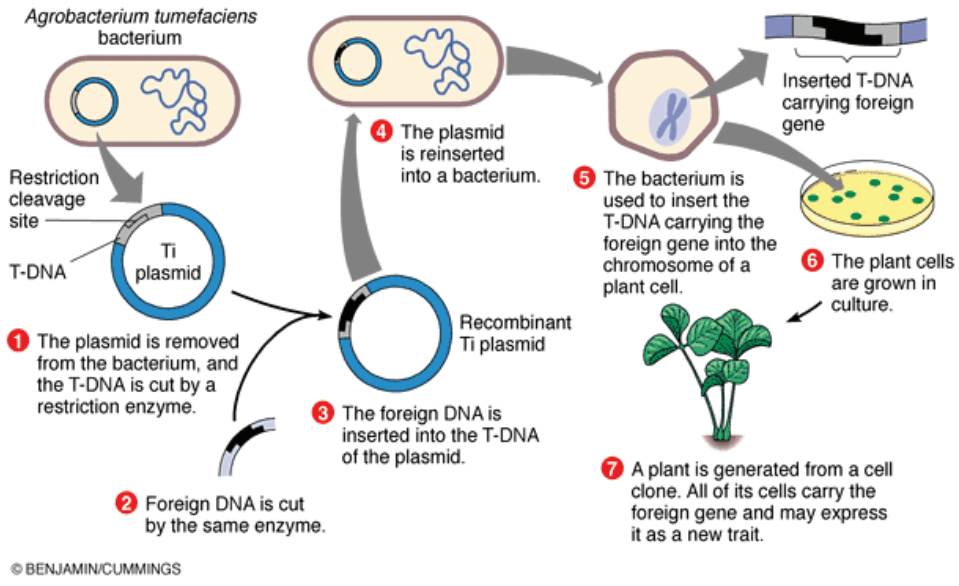


Figure B explains the process of the Agrobacterium-mediated Method (10). After identifying and isolating the gene of interest, the Ti-Plasmid within Agrobacterium tumefaciens bacteria is removed. It is then inserted with the gene of interest. The Ti-Plasmid is then returned to the bacterium, and the bacterium naturally invades the desired crop. This crop now contains the gene of interest (10).

more minerals and vitamins than those found in organic foods. These foods are also known to taste better, and have more lasting freshness. They have an increased shelf life, so there is less fear of foods getting spoiled, and that is why many supermarkets opt for genetically modified versions of produce (4).

For example, an analysis published in February by researchers in Pisa, Italy in "Scientific Reports" found that genetically engineered corn has a significantly higher yield than organic varieties, while also containing lower amounts of toxins produced by fungi. This is because scientists genetically engineered this corn to resist insect damage. With this, farmers have been able to use fewer pesticides while also increasing yields, which improves safety for farmers and the environment, while lowering the cost of food and increasing its availability. Yields of corn, cotton and soybeans have risen by 20 to 30 percent

with the use of genetic modification (3).

GM foods are also useful in preventing certain diseases. By modifying the genes of crops, many properties that can cause allergies are eliminated. In addition, genetically engineered foods grow faster than the foods that are grown traditionally. This sustains the population with more food. Also, genetically engineered food crops can be grown at places with adverse climatic conditions too. A normal crop can grow only in specific seasons or under certain climatic conditions, while genetically engineered crops can withstand unfavourable weather conditions. The U.S saw a significant increase of more than 370 million crops during the years 1996–2012. One-seventh of the increased yield can be accredited to GM crops. The global increase in agricultural income due to genetic engineering techniques had reached $116 billion in 2012. GM foods are thriving and improving conditions for farmers economically. According to the estimation from James and Brookes, about 42% of the universal economic gain for farmers is because of the increased yield due which is attributed to genetic engineering and resistance to pests and weeds (9). Genetically modified products are proven to be beneficial to the economy, as well as the producer and consumer of the product.

On the other hand, many people are concerned about the risks that genetic modification may bring along. Although, many concerns are very theoretical. Some of the risks include the possible introduction of unexpected or harmful genetic changes, undesired changes in nutritional content, and/or the creation of allergens and toxic effects on our organs (3). Critics of genetically modified crops express the need to learn more about the possible long-term effects of genetically modified plants on the environment and the crops themselves before mass-production and purchase of these crops. However, billions of edible animals are fed crops containing GMOs, with no evidence of harm or damage to their health. In fact, being fed genetically engineered crops improved animal health and growth, according to a 2014 review in the Journal of Animal Science (3).

Ninety percent of scientists and associations believe that GMOs are safe and unharmful, such as the American Medical Association, the National Academy of Sciences, the American Association for the Advancement of Science and the World Health Organizationonly. However, only slightly more than a third of consumers believe in the safety of GMOs. Being that common consumers are much less knowledgeable than scientists about this matter, many customers are skeptical about GMOs. Scientists say that there is no possible way to prove a food is safe, only to say that no hazard or harm has been shown or detected to exist. The fears of GMOs are still theoretical. According to an interview in Scientific American with Robert Goldberg, a plant molecular biologist at the University of California, Los Angeles, he states that such fears have not yet been dispelled despite “hundreds of millions of genetic experiments involving every type of organism on earth and people eating billions of meals without a problem” (3). The GMO controversy is still ongoing because of scientist’s inability to definitively prove that genetically engineered crops are safe. While the risks of genetic modification are still all hypothetical, GMOs are not yet to be proven non threatening and “safe,” with scientists still researching the possible long-term effects of GMOs.

The GMO labelling confusion has led to increasing consumer mistrust. Approximately 70% of all processed foods in the United States contain at least one genetically modified ingredient, from cereals to soups to yogurt. However, most consumers are not aware that the foods they are eating include these genetically modified ingredients. There are initiatives in place to begin the requirement to provide clear genetically modified labeling on all processed foods containing GMOs. This would make it easier for people with allergies to keep away from foods that might pose a danger to them, and it would allow those who oppose genetically modified foods to avoid buying them (5).

Congress addressed this controversy by passing the National Bioengineered Food Disclosure Standard, which President Obama signed into law in July 2016. This bill created national standards

for mandatory GMO food labeling. However, the U.S. Department of Agriculture was given two years to determine the exact rules and regulations for the GMO labels. With a new administration in place, Trump's new Secretary of Agriculture, Sonny Perdue, has altered the course of the labeling proposal. Instead of requiring food manufacturers to use widely-known labels such as "GMO" and "genetically engineered," Perdue's Department of Agriculture has established label requirements with less familiar terms like "BE" and "bioengineered." It seems that Perdue's Department of Agriculture is attempting to avoid or "tiptoe" around the words "GMO" and "genetically engineered," as to not drive consumers away from buying these genetically engineered products. However, voluntary labeling has created its own issues that increase consumer mistrust. For example, the well-known brand Tropicana Orange Juice voluntarily labels their juice as "non-GMO," even when genetically modified oranges are yet to exist. Beth Kowitt of Fortune wrote, "It's a little bit like labeling oranges gluten-free" (8). Genetic modification labelling appears to be misleading, whether by using unfamiliar terms like "BE," or promoting foods as non-GMO when there is no genetically modified alternative.

Genetic modification is an efficient and promising process which allows producers to improve plants and combat issues in crops. Although proven to be advantageous to our economy and society, many oppose it due to its theoretical risks and labelling controversies. GMOs are significant and considerable in our supermarkets and notably beneficial to our economy and society.

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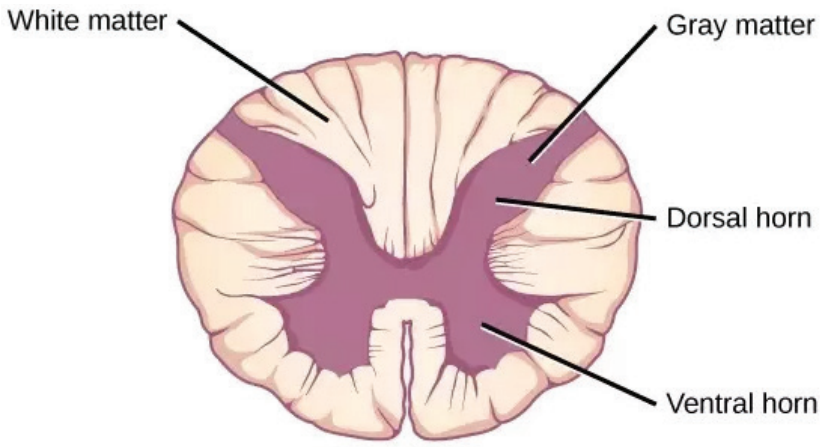
GENIUS GENES: YOU MIGHT NOT HAVE THEM

Michal Englander, 11th Grade

When you get back a bad test grade and the girl sitting next to you gets a 100, do you ever wonder why she got a better grade, is she more intelligent? What some don't realize is that the development of intelligence is much more complex than they think, and it doesn't just have to do with how much you study.

The cause of human intelligence has interested philosophers and scientists since the days of the ancient Greeks. Through modern technologies, such as magnetic resonance imaging (MRI), functional MRI (fMRI), electroencephalography (EEG), and other measures of brian structure-activity, there have been more opportunities to measure what causes human intelligence. (1) However, it is important to define intelligence and to remember that most studies talk about intelligence as scoring high on intelligence quotient tests (IQ test). Although the IQ test is the most widely used test to measure intelligence, it does not measure all types of human intelligence or cognitive abilities. (9)

Many scientists have said that the causes of human intelligence are solely in the brain, but what if that's not the case? Psychologists have had other theories unrelated to the brain, suggesting the causes for success and intelligence. In his book *Outliers*, Malcolm Gladwell proposes that success and intelligence are not based on innate talent, as some may think. Success is based on hidden advantages that some may have. One of Gladwell's most famous theories is called The Matthew Effect, where Gladwell claims that children who are born at the beginning of the year are often more successful in school. The reason for this is that when teachers start tracking children for different reading or math groups, they often "confuse maturity with ability," and the older children who are often placed in the higher-level groups learn more skills, so the next year when they are tested they will do even better and will again be placed in the higher-level tracks. Gladwell says, this "locks children into a pattern of achievement and underachievement," and this pattern travels with the children throughout their entire life. These theories do have merit; however, scientists have proven that



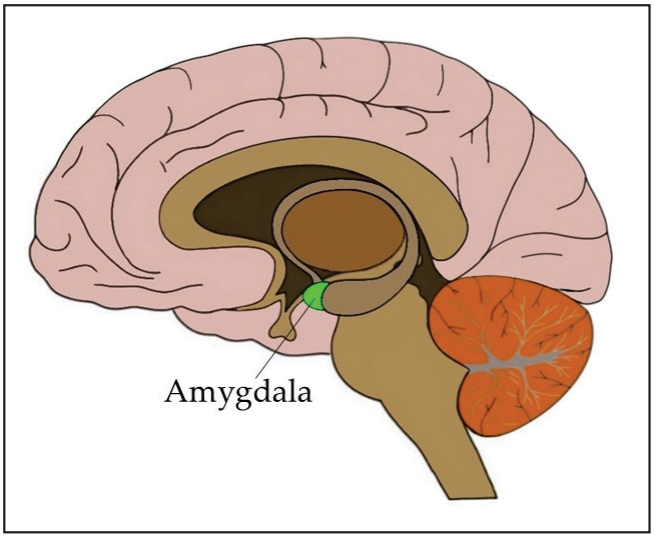
This image explains where grey matter and white matter are located in the brain

intelligence does stem from brain development and is not solely based on birthdates or backgrounds. (5)

However, many prominent thinkers believe that intelligence is related to the brain, whether they believe the cause is brain volume or other factors. According to some, there is a correlation between brain volume and high IQ levels. The results of a few search teams, using different tactics, populations, and scans have found that IQ and brain volume correlate at about the 0.40 level. (6) The volume of the brain is mostly made up of different tissues, such as grey matter, the darker tissue of the brain and spinal cord, white matter, the lighter tissue of the brain and spinal cord, and cerebrospinal fluid, the fluid which clogs the space between multiple layers of the brain. The amount of grey matter correlates to the number and the density of cell bodies and dendritic branches, which collect information from other cell bodies. The amount of white matter correlates to the number of axons and their myelination status, the formation of myelin around a nerve to allow neural messages to travel faster. Therefore, we see that the amount of grey matter "reflects the capacity of information processing centers" and the white matter reflects "the efficiency of inter-neuronal processing centers." Based on these findings neuroscientists think that individual intelligence may be based on brain volume, and more specifically the volume and density of the brain's tissue. (1)

In contradistinction, other neuroscientists believe that there is little correlation between brain volume and intelligence. Consider the honeybee; it can recognize faces, inform other bees of the quality and location of food, and successfully navigate confusing mazes because of the memories it stores in its short term memory. The honeybee effectively does all these operations with less than one million neurons, which is less than one millionth of those contained in the human brain. (10) But are humans really a million times smarter than the bee? Certainly not. Scientists have conducted studies about the correlation between brain volume and high IQ scores, and the results have shown that the correlation between the two are in the 0.3-0.4 range (9), which means brain size is responsible for between 9-16 percent of the overall composition of general intelligence. (10) Clearly there is more to intelligence than just the brain size, or geniuses with normal-sized brains like Albert Einstein would not have achieved the success that they attained. Microscopes give neuroscientists the ability to look at the brain with a magnifying glass, which causes some to believe that the complexity of cellular and molecular organization of synapses, which are neural connectors, is what truly makes up the brain's computational capacity. Therefore, intelligence depends more on how quickly different parts of your brain communicate with each other, and not as much on brain size. (9)

A study was done by researchers of the Kennedy Krieger Institute, in Baltimore, in which the researchers used MRI brain images to analyze the cerebral formation and development in 85 healthy children from the ages of 5-17. The results showed that on average the cerebral volume is 10% larger in boys' brains than in girls' brains. However, both boys' and girls' cerebral volumes don't really change after the age of 5. The main reason why men and boys have larger cerebral volume is because of the increased amount of grey matter, as opposed to white matter, thereby supporting the hypothesis that since male brains have larger cerebrum, the cerebral cortex has more neurons. A child's IQ often positively correlates to their total cerebral volume, particularly with the volume of cortical grey matter in the prefrontal region of the brain. (2) Other scientists have researched the influence of cortical development in children, primarily in the prefrontal region, on intelligence. During the early childhood stage, we don't see a connection between intelligence and cortical thickness, while in late childhood and beyond we see a positive correlation between intelligence and cortical thickness. High IQ levels in children are often seen in correlation with the prefrontal cortex, which has both structural and functional relations with intelligence. (3) Often, more intelligent children were found to develop cortical thickness and more consistently over longer periods of time, than less bright children. (3)



Throughout history, men were often seen as more intelligent and women less, but is this true? Male and female brains do have a few anatomical differences. Male brains and amygdalas are bigger The amygdala is associated with experiencing emotions and the recollection of emotional experiences. However, a woman's hippocampus is larger. The hippocampus is the part of the brain which is responsible for learning and memorization. In 2000, a study was done in which men's and women's brains were scanned while watching either emotionally neutral movies or highly aversive ones. The researchers expected to get strong negative emotions from the amygdala, which is situated in both of the brain's two hemispheres. The amygdala did in fact give a strong response as expected. Surprisingly, however, this strong response was observed only in women's left amygdala, and only in men's right amygdala. Researchers know that women remember memories of emotion more vividly, strongly and quickly than men do. (7) There is a lot of research that suggests that emotional intelligence is very critical in both men's and women's work performance. 58% of people's success in different occupations is due to emotional intelligence, and 90% of top performers have high emotional intelligence levels. When women were tested, they were shown to have a significantly high emotional intelligence level. (8)

One might think that intelligence is just a knack that some have and others don't, just like some people are good at art while others are fast runners. This is not the case, intelligence is much more complex and intricate, involving many more factors than just luck. So the next time you fail your math test, know it may not be completely in your control.

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"I NEVER LIE" IS THE BIGGEST LIE: WHO LIES AND WHY?

Leah Harris, 11th Grade

Did you know that on average, people lie between eight and nine times daily? Just kidding, I lied. Although that statistic may not be true, by the age of four, 90% of children have grasped the art of lying. (7)

There's no reason to be concerned by this fact because lying actually plays a natural role in cognitive growth. Still, just because it's natural, doesn't mean it's okay. Nearly every religion, society, and moral code condemns lying. One can understand the negative consequences of lying, but what's the science behind it? Why do we lie?

According to Dr. Kang Lee, there are two main components to the act of lying: intentionality and conventionality. The intentionality component dictates that speech is a deliberate action, meaning that our resting state is not speaking, rather we have made a conscious decision to lie. In order to lie effectively, one has to determine the recipient's mental state, as well as his own, and think of a way to conceal the truth, and convey a falsehood. Sometimes, younger children will lack this component, which is why it can be easy to determine whether or not they are lying. Take the scenario of a child who broke a glass. The child may claim that a dinosaur came and broke it, or that aliens told him or her to do so. When young children lie like that, they aren't trying to be funny, rather, they lack the "inability to represent their parents' mental state", and therefore, may think up an impossible situation (4,6,9)

The second component is conventionality. This component dictates that before one lies, he or she analyzes the social situation and determines whether or not lying is acceptable. Once again, young children are deficient in this component, which can hurt others. It is well known that when someone is looking for an honest opinion on the taste of a dish, or the flattery of a dress, to go to a child. Because children have experienced fewer social situations in their lifetime, they don't know when it's socially acceptable to lie. When a child receives a gift, and says he doesn't like it, it isn't because he wants to come across as rude, rather, he doesn't understand that in this situation society permits one to lie. (4)

Research states that we are more likely to lie in stressful situations so that we can rationalize. Dan Ariely conducted an experiment that went as follows: volunteers are given 20 math examples. Within five minutes, they are to answer as many as they can. For every question they answer correctly, they are paid. At the end of five minutes, they are given an answer key, and told to shred their paper. Then, they report how many they got correct. The trick, however, is that the shredder isn't actually a shredder. (6)

On average, volunteers claim to have solved six examples correctly, when in truth, they really only solved four. The question is: why don't the volunteers lie more? Ariely states, "Here we give people a chance to steal lots of money, and people cheat only a little bit. So something stops us -- most of us -- from not lying all the way." Ariely's belief is that people want to perceive themselves as honest. (6) No one wants to think of himself as a pathological liar, so people minimize how much they lie in order to justify their actions, and avoid a guilty conscience. (6)

In 2016, Nature Neuroscience published an article discussing how lying alters a person's brain. fMRI research has shown that when people lie the amygdala bursts with activity. (3) The amygdala is the part of the brain that controls emotions and survival instincts. (See figure 1) It makes sense that the same brain region that processes emotional information from personal interaction is active during deceptive behavior. (d) This is similar to the aforementioned lying component of conventionality. It is

also connected to the reason we lie under pressure; it's our survival instinct kicking in.

According to a study at UCLA, the first time someone lies, the amygdala responds with much activity, but after a few times, the amygdala lights up less and less. This is called "emotional adaptation." (2) For example, the first time someone cheats on a test, his amygdala will flood with negative emotions. But by the second time he cheats, his brain will have already adapted. Therefore, there is less of a negative reaction to hold him back. (2)

Nobuhito Abe at Kyoto University and Joshua Greene at Harvard University used fMRI to scan subjects. They found that those who acted dishonestly showed a greater activation in the nucleus accumbens, which is used in reward processing. (See figure 2) Joshua Greene states, "The more excited your reward system gets at the possibility of getting money [...] the more likely you are to cheat." (3) This too, aligns with the topics above. The nucleus accumbens controls our reward system, which explains why people will lie when there is an incentive or a reward -- they will lie to their personal benefit.

In 2005, Yaling Yang conducted brain scans on three groups: 12 adults with a history of frequent lying, 16 who had antisocial personality disorder but were not repeated liars, and 21 who were not antisocial and who did not have a lying habit. The results were that the frequent liars had, at minimum, 20% more neural fibers by volume in their prefrontal cortices than either other group. (3) The regular function of the prefrontal cortices is focus, predicting consequences, as well as impulse control. (8) This study suggests that frequent liars have more connections in their brain. It may be that due to an increased number of connections, it is easier for them to think of lies, and they can do it faster than their peers. (3)

As these articles discuss, although we tend to think of lying in exclusively moral terms -- considering whether a person was or was not justified in telling a specific lie -- the reality is that people lie or tell the truth for complex reasons and with complex goals. In fact, as brain studies show, we often do not even know our true motivations and reasons for lying. Understanding better why we lie will hopefully teach us how to be better people. Of course, as G-d fearing Jews, despite the compulsion for lying and its illusory benefits, we know that our Torah demands integrity, which is one of the fundamental qualities that define a Jew. Truth and integrity are also one of the most vital components of Jewish education.It is crucial that we train our children that lying is reprehensible and destructive so that they do not become naturally disposed to lying, Integrity is a bedrock of Torah living.

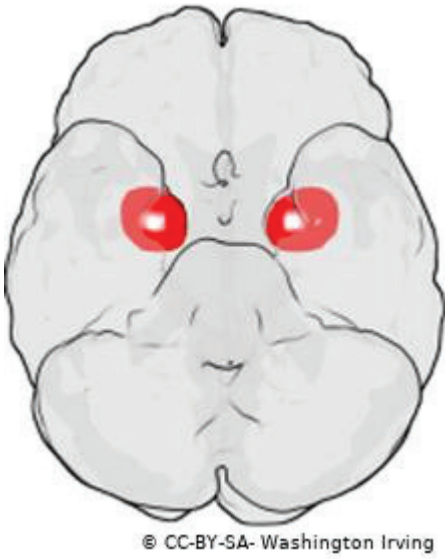

Figure 1: The amygdala highlighted in red.

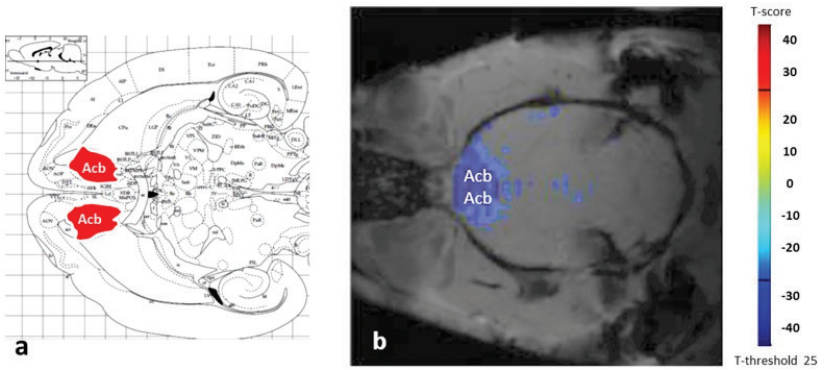

Figure 2: Nucleus accumbens highlighted in red

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NEMO NEEDS HELP FINDING HIS WAY

Adina Hoffman, 10th Grade

Imagine you booked a half day cruise (pre-COVID of course) on a glass-bottom boat off the coast of Eilat. You were anticipating all week long to the snorkeling experience of the spectacular display and vibrant colors of the undersea world but to your chagrin, there was nothing.

You throw some crumbs hoping to lure these shy creatures, but to no avail. What a disappointment. The coral reef had only white, short coral, no clams or oysters, and the few fish swimming had no sense of direction. "What has happened?" you sullenly wonder as you head back to shore.

Understanding greenhouse gas effects on our environment in general, and on our oceans in particular, has been a long standing and historied enterprise. Although most of the world's attention is given to greenhouse gases such as carbon dioxide (CO2) and its deleterious effect on the atmosphere and climate change, ocean environments are negatively impacted as well. Carbon dioxide created through human activity, stays in the earth's biosphere for about forty years, until being released back into the atmosphere through decay.

On the other hand, estimates ranging from one quarter to one-third of the CO2 emitted each year is quickly absorbed into the oceans, causing ocean acidification (1, Figure 1). Carbonic acid (H2CO3) is formed when water (H2O) and CO2 mix, releasing hydrogen ions (H+), which can then bond with other molecules. Seawater with a lower pH becomes more acidic with increased levels of hydrogen ions. Acidification is an interplay between H+ and pH where acidity reflects the concentration of H+ ions in a solution. An acid is a substance that releases H+ ions, and pH is the scale used to measure the concentration of H+ ions. Ocean pH has dropped from 8.2 to 8.1 since the industrial revolution, and is expected to fall another 0.3 to 0.4 pH units by the end of the century. A small change in the pH of seawater has been shown to have detrimental effects on marine life, impacting chemical communication, reproduction, and growth (3).

Ocean chemists have documented the accumulation of fossil fuel generated CO2 in the upper ocean, and its penetration to the deep-sea. Unfortunately, current calculations estimate the rate of CO2 invasion exceeding one million tons per hour with total estimates of 530 billion tons of fossil fuel CO2 already contained in the oceans (2). This disruption of the delicate geochemical balance in the ocean has impacted ocean ecosystems and is only now beginning to be understood. Imagine snorkeling in a coral reef without huge masses of coral and fish to marvel at.

Skeleton building in marine animals is particularly sensitive to ocean acidification. In order to produce calcium carbonate (CaCO3) shells, marine animals such as corals and oysters need to combine calcium ions (Ca+2) with carbonate (CO3-2) from surrounding seawater, releasing CO2 and water in the process.Both calcium ions and hydrogen ions tend to bond with carbonate, but the hydrogen ions have a greater affinity to carbonate than calcium. A bicarbonate ion (HCO3-) is formed when hydrogen bonds with a carbonate. Unfortunately, shell-building organisms are not able to extract the carbonate ion from the bicarbonate, which prevents these organisms from employing the carbonate to grow new shells. The hydrogen, in essence, has a greater affinity for and binds up the carbonate ions, making it harder for shelled marine organisms to build their protective coverings (4).

Coral reefs are negatively impacted since calcifying marine organisms building their skeletal structures are extremely sensitive to ocean acidification. As the temperature of the water rises, coral calcification increases, algal endosymbionts are lost in a process more commonly known as coral bleaching (Figure 2). In addition, as pH decreases, the amount of carbonate ions decreases, which corals use to build their calcium carbonate skeletons, and consequently, the skeletons do not calcify as well. Location of the corals can further impact this effect. Inshore reef environments are more adept at adapting to different environments, mainly because of their shallow depth, as opposed to offshore reef environments (4). At this point, imagine the same coral reef as before, but without any color. You are just snorkeling with small, short, white coral surrounding you.

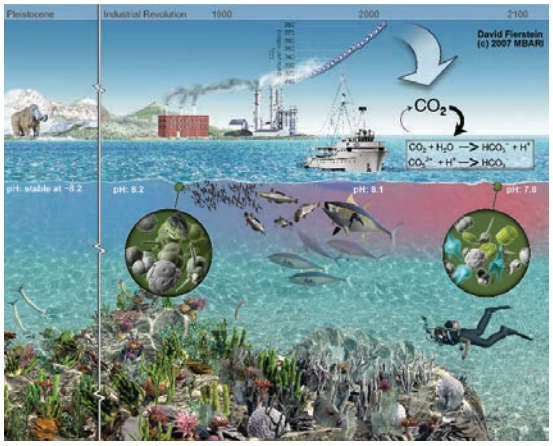

Figure 1: Artist's rendition of anthropogenic CO2 and the resulting Ocean Acidification

Reprinted from Peltzer, Edward. "Emerging Science of a high CO2/Low pH Ocean." *Monterey Bay Aquarium Research Institute, Accessed February 11, 2020, https://www.mbari.org/science/seafloor-processes/emerging-science-of-a-high-co2low-ph-ocean/*

Even if these marine animals were able to build skeletons in more acidic water, they would have to expend more energy to do so, depleting resources from other activities like reproduction. Moreover, increased hydrogen ions, with insufficient bonding molecules, can result in the breakdown of calcium carbonate molecules and even existing shells and skeletons made from calcium carbonate can begin to dissolve (Figure 3). Marine life that produces calcium carbonate structures have to spend extra energy either repairing their damaged shells or thickening them to survive. Some shellfish that produce calcareous shells, particularly in the early stages of development, are especially vulnerable to elevated CO2 levels (5). For example, massive oyster "die-offs" have been reported in the United States Pacific Northwest because the acidic seawater caused their shells to disintegrate before they could even form (6).

Another example, pteropod shells have been reported to be dissolving in the Southern Ocean, where more acidic water from the deep sea rises to the surface, accelerating ocean acidification effects. These sea snails, like corals, are particularly susceptible to ocean acidification because their aragonite shells are made from a delicate form of calcium carbonate that is fifty percent more soluble in seawater (7). Foraminifera has even been predicted to become extinct by the end of the century due to the rapid dissolving of their shells which are also particularly susceptible to high acidity (8). Imagine that same coral reef, but without any clams or oysters.

Many fish are excellent at altering blood chemistry to accommodate changing seas. However, a considerable body of research investigating the impact of elevated CO2 levels in seawater has shown that carbon dioxide in the ocean acts like alcohol on fish, impairing their senses and leaving them less able to judge risks. Elevated CO2 disrupts brain signaling in a manner common among many reef fish, with noted olfactory (9), auditory (10) and visual (11) impairments and behavioral changes including increased boldness and activity and impaired behavioral lateralization with compromised propensity to turn left or right (12). Baby reef fish exposed to high CO2 and placed back in the wild died five times more often. Even when baby reef fish and predators were both exposed to high CO2, young fish were

bolder, ventured further from home and died twice as often (12).

Figure 2: Coral Bleaching

Reprinted from https://www.cnn.com/2014/06/18/world/asia/australia-great-barrier-reef-whc/

Elevated CO2 has been found to interfere with neurotransmitter receptor function, specifically the gamma-aminobutyric acid (GABA-A) neurotransmitter receptor. When GABA, an inhibitory neurotransmitter, binds to its receptor, a flood of negatively charged chloride and bicarbonate ions rush into the neuron and prevent it from firing. Ion distribution is altered when CO2 accumulates in the fish, so that when the receptor opens, chloride and bicarbonate ions escape out of the cell, causing an excitatory response as opposed to an inhibitory one, with derivative negative consequences on the function of neural circuits in the brain.

Adult clownfish (*Amphiprion percula*), initially reared in high levels of CO2 (900 microatmospheres), swam towards rock cod's (*Cephalopholis cyanostigma*) scent around 90 per cent of the time when given a choice between a water stream containing the odor of common predators such as the rock cod or a stream lacking predatory odors. However, those reared in normal (450 microatmospheres) levels of CO2 avoided the predator's scent more than 90 per cent of the time (13). Treating clownfish bred under CO2-rich conditions with gabazine, a GABA-A receptor blocker, helped the fish regain sensory function with evidence of decreased swimming towards the predatory smell only twelve per cent of the time. Imagine that same coral reef again, but with little Nemos swimming around trying to get their way home. In this situation, their dad won't be coming to rescue them because all the clownfish are experiencing the same problem of losing all sense of direction. A second experiment employing juvenile damselfish (*Neopomacentrus azysron*) from Lizard Island in the Great Barrier Reef, revealed that in certain situations, high levels of CO2 destroyed their natural tendency to turn left or right, a crucial factor in shoaling. Gabazine restored this "handedness" (13). Similarly, ocean acidification was also shown to impair retinal function in damselfish through interference with GABAA receptors and was counteracted by treatment with gabazine (11).

In conclusion, ocean acidification has been shown to damage coral reef environments by impairing the ability of marine life to build (4) and sustain (5, 6) their shells and skeletal structures. Further increased levels of acidity has been shown to disrupt cognitive function and to induce behavioral disturbances (12,13) by impairing sensory function (9,10,11) with alterations in GABA receptor processing (13). These behavioral alterations have been shown to significantly affect mortality rates (6, 8) with a derivative negative impact on community structure, population replenishment and overall ecosystem function (1-4). As a possible opposition, high CO2 levels which have been implicated in these abnormalities, specifically chemosensory function, have been shown to be effectively reversed by treatment with gabazine, a GABAA antagonist (11, 13).

Future research should investigate the possibility of employing this technology as a tool in the management and conservation of ocean environments in the hope of reversing the negative implications of CO2 buildup in threatened ocean systems. Interestingly and on a positive note, on April 23, 2020,

CNN reported that the Florida Aquarium made a breakthrough in creating coral in the Apollo Beach Laboratory which they are beginning to place in devastated coral reefs. So far, more than 350 coral babies have been released into the marine ecosystem (14). Hopefully, we can all go on that cruise and enjoy snorkeling among colorful coral reefs, teeming with life.

Figure 3: The acidic waters from the CO2 seeps can dissolve shells and also make it harder for shells to grow in the first place. (Laetitia Plaisance)

Reprinted from "Ocean Acidification." Smithsonian Ocean, April 2018. Accessed February 2020, https://ocean.si.edu/ocean-life/invertebrates/ocean-acidification

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DNA AND GANG VIOLENCE IN SAN DIEGO

Musia Kirschenbaum, 11th Grade

On April 24, 2014, Gregory Benton was murdered in a drive-by shooting as a result of complicated gang dynamics. Benton, a member of the West Coast Crip Gang, had been walking home after a cigarette run with his cousin when two unidentified males got out of a car and began to shoot at them.

While his cousin ran for cover, Benton was shot multiple times and died on the street. All that was left behind was his body and multiple bullets strewn about the streets of San Diego. Previously, this would have been a cold case, as bullets could not provide individual evidence, proof that points toward a specific person; the only thing the bullet could provide the forensics departments with was information about the gun it was fired out of. While the police suspected that it was a member of a rival gang, the "5-9 Brim's," a group that frequently had 'turf wars', they had no real evidence to arrest any specific members, just their own speculations. However, lucky for the San Diego Police Department, scientists in the Netherlands had just developed a new method of extracting usable DNA from bullet casings, a method currently employed by their local crime lab. By using this new method, the S.D. Crime lab was able to extract DNA and match it to two men, both affiliated with the "5-9 Brim" gang, who were tried in court for murder and sentenced to 38 and 89 years in prison, respectively.

Prior to the Benton murder, the crime lab in San Diego, inspired by the research of the Dutch scientists, conducted their own study on the new method. The lab in San Diego gathered a group of ten people to load guns and shoot them over and over in different settings and environments that mimicked those of a crime. Then, the bullets were collected and sent back to the lab where they were analyzed by the forensic scientists using the new method of collecting DNA off of ammunition. The S.D. Crime Lab was able to collect viable DNA from 34% of the bullets they fired, and in 2014 they officially instituted it as a method in their lab.

So what is this new DNA method helping to solve numerous shootings? According to Kristin Beyers, you should think of it sort of as "soaking your dishes." The bullets are swabbed for DNA by the forensic analysts, first at the scene, and then again at the lab to make sure there was contamination during transport. (*See the bullet on the right - the bottom half is usually swabbed as it is most likely to be touched when loading a gun, meaning it is most likely to have incriminating DNA on it.)*

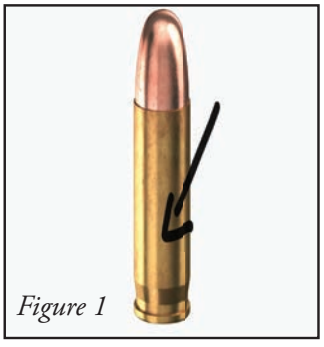

Figure 1

The bullets are then soaked for half an hour in a chemical mixture that separates the DNA from the metal. The chemical mixture is essentially a complex detergent-based lysing solution geared to rupture the cell membranes in order to get the DNA out. Detergents are designed to bond to the grease on dishes and pull it off. The reason they can do this is that detergents are amphipathic solutions, meaning they contain both hydrophilic and hydrophobic parts. The hydrophilic parts of the detergent bond to the water in the sink and the hydrophobic parts bond to the grease, which is an amphipathic lipid (fat). Together with the combined heat and water in the sink, the detergent aids the water in pulling the grease off the dish. This is helpful in DNA extraction because in the same way the

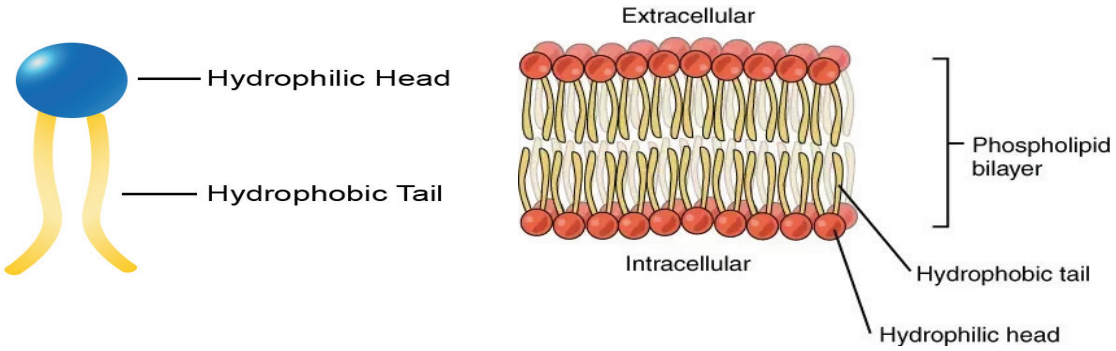

Figure 2:
Left image: *amphipathic phospholipid that surrounds the cell.* ***Right image:*** *phospholipid molecule that makes up the grease on plates. Notice the hydrophilic heads and hydrophobic tails of the phospholipid bilayer that protects the cell's internal organelles, mimicking the lipids in the grease stuck to the dishes. This accounts for the similar ways in which the detergent and the lysing solution work- the detergent's hydrophobic parts attach to the grease and pull it off. The lysing solutions hydrophobic parts attach themselves to the cell membrane and pull it off.*

grease on the dish is an amphipathic lipid, the cell membrane, in which the DNA is located is also an amphipathic phospholipid with the hydrophilic heads facing outwards. This means that the detergent-based solution is able to attach to the cell membrane the same way it attached to the greasy plate and pull it off, leaving the insides of the cell, where the DNA is located, exposed *(see figure 2.)* For the best DNA samples, some scientists subject their solutions to sonification during the soaking process- they use sound waves of high frequencies to further upset the particles in the solution and help the detergent bases to break apart the cell membrane.

The scientists in the lab then swab the soaked bullet and place both the original crime scene swab. the swab from the lab, and the lysate solution1 in an incubator along with the enzyme proteinase K, which functions as a protection against the degeneration of the DNA by breaking down nucleases, enzymes that break down the phosphodiester bonds present in the nucleic acid *(see figure 3.)* After it is incubated for an hour, the entire lysis solution is placed in a centrifuge where the DNA is separated from the solution. At the same time, the DNA is removed from the incubated swab. Now with the two viable DNA extractions, the scientists in the lab can perform any form of DNA amplification on them, ranging from PCR to isolate specific parts (such as STR) of the DNA to HDA, helicase dependent isothermal amplification, which requires no thermal incubation, decreasing the risks of DNA degeneration.

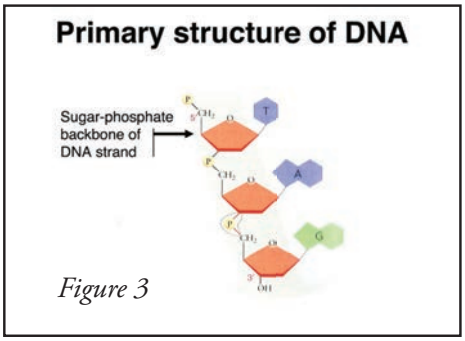

Figure 3

This process of extracting DNA from bullets is not foolproof, although nothing is in forensics. There are a variety of circumstances that would degrade the viability of the DNA found on the bullet casings. The first is the kind of metal the bullet casing was made from; copper, for example, is a metal that degrades DNA due to its high oxidation levels. The copper bullet oxidizes and corrodes and when it does this it releases ROS, reactive oxidation species, that infiltrate and alter the double helix structure of the DNA.The scientists also must worry about thermal degeneration. When a bullet is fired it generates

a lot of heat, heat that one would assume would denature the DNA on the bullet. This point is not a huge issue though, as recent studies have come out showing that the casing outside of a bullet usually does not get hot enough to completely denature the DNA on its surface.

Not only did the bullets in San Diego help solve the murder of Gregory Benton, they also helped to solve another murder. The bullets collected by the police were run through NIBIN, National Integrated Ballistic Information Network (the CODIS of ammunition), and matched to a gun that had been used in a San Diego shooting two days prior to Benton's. This meant the police now had evidence linking the same gang member who killed Benton to this murder, and were able to arrest and convict him.

This new method surfaced at just the right time, not just for San Diego, but for the entire United States. Since 2014, gun deaths have increased by 16%. Of these deaths, 35% of them are gun homicides. Gun violence is a rising problem in America and methods like these are crucial to the prevention, containment, and punishment of its perpetrators.

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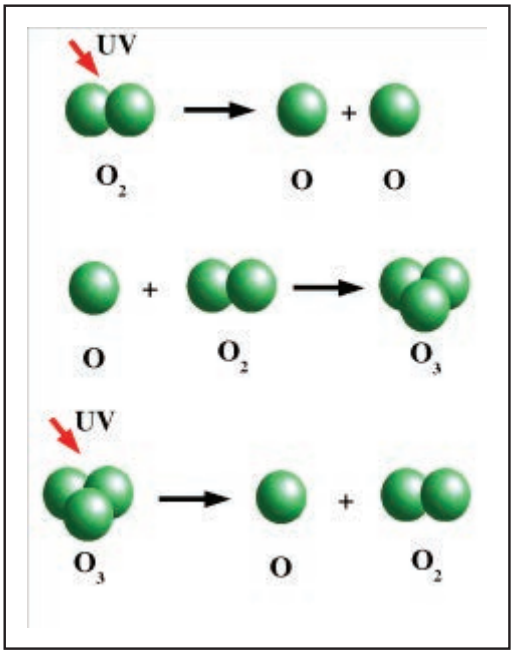
THE ICE-MAN COMETH

Rikki Klein, 10th Grade

Try to imagine a life without refrigerators or air conditioners. Before the availability of refrigerators, people used to stock up their ice boxes daily with fresh batches of ice to keep their foods from spoiling. Many times, food would not last as long as expected and grow mold.

In the scorching heat of the summers of the 1800s, before air conditioners were introduced, people in the sweltering crowded New York City tenements were forced to sleep on the fire escapes in order to find refuge from the unbearable heat and catch a breeze. Luckily, in the mid 1800s, refrigerants were invented. In the beginning, these refrigerants were made using toxic chemicals that were not safe for human use, and an alternative was needed.

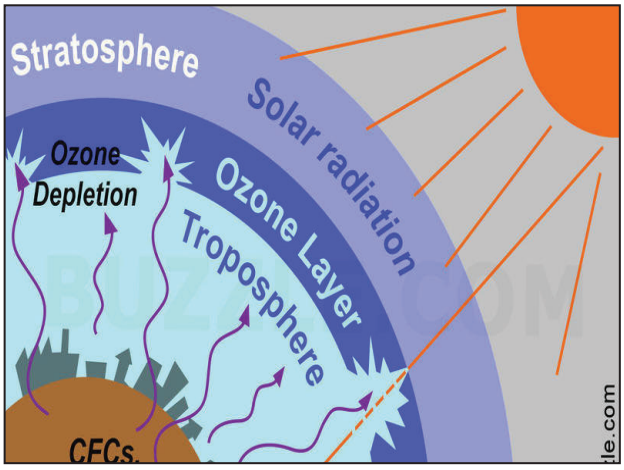
Chlorofluorocarbons, also known as CFCs, are a group of chemicals that contain atoms of chlorine, fluorine, and carbon – hence the name. These chemicals were mainly found in the manufacturing of refrigerators, air conditioners, aerosol cans, and packing materials. CFCs were first synthesized by Thomas Midgley in 1928 as a safer, non-toxic alternative for the deadly gases like ammonia, methyl chloride, and sulfur dioxide that were being used as refrigerants for refrigerators at the time. Previously, these gases were seen as acceptable and safe options to be used. However, in the 1920s, the methyl chloride started leaking out, causing many casualties (4). It was so dangerous that people started to leave out their refrigerators in their yard to protect themselves from the toxic chemical leak. As a result, the American refrigerator corporations started seeking out a safer alternative, and this is when Midgley first discovered the potential benefits of CFCs. They were referred to as a "miracle compound" and replaced the deadly chemicals that were formally being used as refrigerants. This is because CFCs have the ideal boiling point- around 32 degrees fahrenheit- for refrigerants. It is ideal for the boiling point of refrigerants to be low because as the cold liquid of the refrigerant enters the evaporator, a fan blows the cool air around, removing all the heat and thereby lowering the temperature of the space. As the liquid refrigerant evaporates and turns into a gas, a compressor returns the gaseous refrigerant back to the liquid state where it can be recirculated. In the 1930's, once they were recognized as safe and had a record of nontoxicity, CFCs were being used as a coolant in many air conditioning systems. In the 1940's CFCs were being used in the manufacturing of aerosol cans, and as the years went on, CFCs were being found in millions of US goods (1).



Although CFCs may seem like a completely safe and useful alternative, they can in fact be extremely harmful. Our Earth's atmosphere is comprised of many layers. The ozone layer is found in the second to lowest layer, called the stratosphere. It is called the ozone layer because the majority of the atmospheric

ozone is concentrated there. Ozone is formed when sunlight breaks apart an oxygen molecule into two separate atoms which then each combine with another oxygen molecule, forming ozone, or O3. The ozone layer protects us on Earth's surface by absorbing a portion of the sun's harmful radiation, and absorbing specific UV rays called UBV that can cause major harm to crops, sea life, and even cause skin cancer (2). UV rays cause DNA damage, which results in mutations. Since our skin is on the front line of UV damage, skin cancers result. (See "A Moment in the Sun" by Sarah Strauss in this publication, and the effect of UV radiation on patients with Xeroderma Pigmentosa.)

In the United States, after the introduction of CFCs in refrigerants, skin cancer cases increased between 20-40%. The cancer development does not happen immediately, and detection may take years to notice. Hence the observed skin cancer cases worldwide increased 90% in 1974 since the 1950s, where CFCs were starting to be used more and more. The ozone acts as a filter, absorbing all of the UBV radiation, preventing its travel from reaching Earth's surface. In other words, the ozone layer is protecting us from many potential harms by absorbing certain energies in the atmosphere. Unfortunately, when the chlorine atoms from CFCs come in contact with an ozone molecule, they destroy it, causing ozone depletion. Ozone depletion is when there is a gradual thinning of the ozone layer in the stratosphere. This happens because UV radiation breaks a chlorine atom apart from a CFC molecule, which then breaks an ozone molecule apart by bonding to one of its oxygen atoms - because an ozone molecule is made up of three oxygen atoms. When the ultra violet waves strike CFC molecules, a carbon-chlorine bond is broken, producing a chlorine atom. Since one chlorine atom can destroy up to 100,000 ozone molecules, ozone was starting to be destroyed more frequently than it was being created, thus causing ozone depletion (5).



One example of ozone depletion is the ozone hole over Antarctica. Back in 1957, before an apparent ozone crisis, researchers began measuring the ozone concentration in our atmosphere for the International Geophysical Year, a scientific project which aimed to learn everything possible about our environment (6). Ever since then, the British Antarctic Survey has continued to measure the ozone levels. In the early 1970s, when the British Antarctic Survey was conducted, they noticed a slight and gradual decrease of ozone levels over Antarctica as the months passed by. They compared this graph to another graph measuring CFCs, and noticed that as the amount of CFCs rose, the amount of ozone fell. They then concluded that the CFCs were in fact harming our ozone layer. There is not actually a hole above Antarctica, but rather a significantly low concentration of ozone in the atmosphere (3). The lack of ozone is a very serious matter because we need enough ozone in our atmosphere to protect us from the harmful UV rays and radiation (7). Additionally, if the ozone layer wouldn't successfully shield us from those UV rays, it would be very difficult for all life on earth's surface to survive. For example, plankton, which serve as a food source for most marine life, would not be able to survive with heavy exposure to UV rays, which would cause immense loss to all sea life.

Due to the destruction that the CFCs were causing to the ozone layer, in 1987, twenty seven nations signed a global environmental treaty in order to reduce the production of substances containing CFCs. This treaty restricted the manufacturing of those goods in order to protect the atmosphere. In

1996, the nations met again to make the Montreal Protocol, where they updated the treaty, applying trade penalties on any foreign goods imported with these CFCs (8). A total of 148 countries are included in this and have agreed to ban the production of these goods. Businesses have now found safer alternatives that are better for our environment than CFCs. For example, now hydrochlorofluorocarbons replace CFCs because they are significantly safer. The ban of CFCs is affecting the ozone layer and our overall atmosphere for the better.

In conclusion, our society today has made many changes and improvements in the process of creating and manufacturing goods. Due to the recent global warming scare, humans as a whole have been trying to raise awareness and act more responsibly with our resources in order to protect our environment. Additionally, specifically due to the continuing ozone crisis, CFCs' emissions have been drastically reduced by all sorts of companies worldwide. For example, all aerosol cans that are being manufactured today, ranging from spray deodorants to hairsprays to anti-rusting sprays, now have a small label on them that clearly says "no CFCs." This is both to raise awareness of their harms and to show that the product was legally manufactured and is safe to use. This law has been in place since the late 1970s as part of the CAPCO's, or The Consumer Aerosol Products Council's, goal to confute the myth that toxic CFCs were still being used for the manufacturing of these products. Hopefully, as time goes on, the use of harmful chemicals in products will stop completely, making the world a safer, happier place.

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THE PLASTIC BRAIN

Chana Lipschutz, 11th Grade

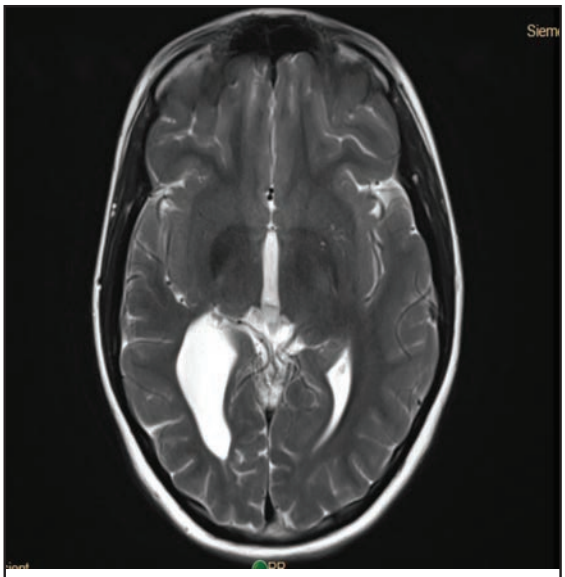
After extensive medical testing, my parents received the news that I had suffered a stroke. I wasn't over 65 when it occurred, which is the median age for people who experience strokes. I was not a young adult. In fact, I wasn't even born.

Although most are familiar with strokes in regard to the elderly, anyone can have a stroke. Perinatal stroke is defined as an insult occurring from 20 weeks gestation and 28 days postnatally (1). A stroke can occur in about 1/2300 full term births, in comparison to about 7/100 in preterm infants.

There are two types of strokes that can occur. An ischemic stroke is the result of a carotid artery's inability to send a blood supply to the brain. This blood contains the oxygen that the brain needs to function. Without the supply of blood, there is no oxygen being delivered to the brain and so brain cells and tissue begin to die. These type of injuries result in infarcts or localized areas of dead brain tissue resulting from loss of blood supply (2). Hemorrhagic strokes occur when there is either bleeding inside the brain or between the brain and the membranes that cover it. This bleeding can also be defined as germinal matrix hemorrhage when referring to preterm babies.

The process of diagnosing stroke in newborns is not a straightforward one. There are two specific ways in which stroke will present itself. Acute Perinatal Stroke is diagnosed when an insult occurs shortly before or right after birth and will manifest in seizures or other encephalopathies (2). Presumed Perinatal Stroke is diagnosed as a remote infarction. This can be described as the injury having occurred in the past, presumably in utero, but the symptomes and the diagnoses had presented at a later stage. The signs of Presumed Perinatal Stroke, with which I was diagnosed, are varied. They include vision impairment and early pathological handedness. People diagnosed with perinatal stroke often have frustrating experiences due to their limitations but can still lead very fulfilling and successful lives.

Strokes among the elderly are unfortunately very common and research has shown that geriatric strokes are often the results of blood clots due to unhealthy daily habits that lead to hypertension, diabetes or obesity. However, there is very little information as to why a stroke might occur in unborn or newborn babies if they themselves have not developed



This is an MRI of a presumed perinatal stroke with a stable appearing remote right periventricular injury, likely ischemic sequela. Additionally there is right parietal volume loss.

These factors caused left hemiparesis and dystonia with some peripheral vision loss

The injured area, represented by the gaping white space on the left side, is clearly visible

Table 3

Genotype distributions among 13 white control infants with perinatal arterial ischemic stroke.

Gene	Polymorphism	*n*	Common Homozygote (%)	Heterozygote (%)	Rare Homozygote (%)
TNF-alpha	− 308 G/A	13	77	23	0
IL-6	G to C	13	39	62	0
Lymphtotoxin	804 C/A	13	39	54	8
Factor V Leiden	506 G/A	13	100	0	0
MTHFR	677 C/T	13	54	39	8
MTHFR	1298 A/C	13	31	54	15
Prothrombin (F2)	20210 G/A	13	100	0	0
Apolipoprotein E	ε3/ε4	13	54	23	15
Apolipoprotein E	ε3/ε2	13	54	8	0

This table describes the genotypes that were researched and the data collected on the thirteen case infants

these bad habits. Therefore, one could easily hypothesize that genetic disorders or mutations are the most common cause of perinatal stroke.

Personally, the insult I suffered is linked to a genetic disorder. It is a genetic mutilation called Prothrombin G20210A. Prothrombin is an inactive protein that is converted to the active thrombin protein when the blood coagulates. This protein both regulates and promotes clotting when necessary. The prothrombin gene disorder will lead to elevated levels of thrombin protein being produced in the blood. This in turn dysregulates the clotting process and produces clots at a more frequent and faster rate. This gene is not directly responsible for a stroke occurrence. A harmful incident or a recurring issue will have needed to cause the defective gene to produce an excess of blood clots which would likely result in a stroke. Especially if the child was unborn at the time of injury, it is nearly impossible to know the direct cause of injury. Although much research is being done in order to better understand the mechanisms of perinatal stroke, one can truly understand how neuroscience is a complex and evolving field of science from the limited information scientists have today about perinatal strokes (7).

Although blood clotting disorders such as Prothrombin G20210A are seemingly the most common cause of perinatal stroke, there are a lot of other hereditary conditions that can possibly cause injury. According to research done done on thirteen infants who suffered arterial ischemic perinatal stroke, the babies who have the apoE ε4 allele were most likely to have suffered an insult with 15% of the 13 control cases possessing that allele. (3) Apolipoprotein E is a protein involved in lipid transport and metabolic reactions. It is secreted into the extracellular space by astrocytes where it is involved in the binding of cholesterol. ApoE ε4 allele is most commonly known as the variant of the ApoE gene responsible for late onset Alzheimer's disease.

One can make a case for the environmental causes that may lead to stroke. In the study analyzed above, elements such as gender, maternal age, whether the baby was born by caesarian section or if the mother experienced preeclampsia are taken into account. According to the study, male infants are more predisposed to experience stroke, with ten out of the thirteen babies who participated in the study being boys. Eight percent of the mothers whose infants had suffered an injury experienced preeclampsia versus four percent of control babies mothers. However, for the rest of the clinical markers, the percentages for both case and control infants were very much the same. For instance, both cohorts had an average maternal age of around 28. This study proves that although clinical factors most definitely play a role,

ultimately it is a baby's genetic makeup that will cause a stroke. The chance of stroke occuring more than once in an infant is relatively low. However, the cases in which stroke was the result of prothrombotic issues do have a higher chance of recurrence (3).

If genetic disorders are a major factor in the cause of a brain injury, the question arises as to what is the Halachic status of those with such genetic disorders that may affect their performance of Mitzvot. In Yevamot 64B, hemophilia is mentioned in reference to circumcision. There is a debate on the course of action that should be taken after a woman has multiple sons who passed away after circumcision. Rabbi Yehudah Hanasi states that the mother should not circumcise her third son after the other two passed away after the circumcision. Rabban Shimon Ben Gamliel argues that only after three sons die can one conclude that the fourth son must not be circumcised. Further on, genetic blood clotting disorders are clearly mentioned; the Talmud refers to them as דרפי דמא ("thin blood" or hemophilia) and דקמיט דמא ("thick blood"or thrombophilia). It is interesting to note that both of these disorders are transmitted genetically from females to their male offspring, a fact that the Gemara is seemingly aware of when they consistently use female pronouns (e.g. היא and לא תמול) throughout this specific discussion (4). An answer to this question can be found further on, in Yevamot 70a. A Mishna mentions the case of an uncircumsized Kohen and Ramban comments that this is referring to a Kohen whose brothers passed away as a result.

While there is no mention of perinatal stroke in the above discourse, we can gain invaluable insight from this passage. First, it is truly amazing to see how the knowledge of Talmudic sages was all encompassing and advanced. Second, while stroke is not mentioned in this debate, one should be aware that both thrombophilia and hemophilia are genetic mutations that can possibly cause perinatal and childhood stroke.

The good news comes in the form of brain plasticity and physical therapy. Brain plasticity is the ability of the brain to change and adapt throughout a person's life. Because a fetus and newborn brains are constantly developing, other areas of the brain are able to compensate for the injured section.

Brain plasticity has also been proven to be an important factor in maintaining intellectual abilities of a person who suffered perinatal stroke (7). Seizures are common among patients who suffered perinatal stroke and research indicates that the presence of seizure activity will significantly interfere with intellectual and cognitive development. Children who did not experience seizures maintained the nearly the same or very similar rates of development as children who did not suffer a stroke. Lastly, it is important to note that the size of the lesion does not affect chances for intellectual progress.

Intensive physical therapy is crucial to the development of an infant who suffered brain injury. My parents took me to therapy even before they received the official diagnoses. Although I often resisted over the years because it was often difficult and frustrating, today I am eternally grateful that they did so. Therapy can promote brain plasticity and even use of the injured limb. This practice is referred to as constraint-induced movement therapy (CIMT), in which the unaffected limb is restrained by use of a glove or the like. The patient is forced to use the affected limb and becomes accustomed to engaging in various activities with the limb even though it may be more difficult. Some other general methods are attempting fine motor skills, balance and hand-eye coordination through exercise and play. Whichever route the child's caregiver takes, putting any emphasis on improvement is key. It can be physically and psychologically harmful to ignore the affected limb.

No matter the cause, however, perinatal stroke is a serious injury that has life altering repercussions. It can be extremely frustrating to grow up in an environment that isn't always equipped to best accommodate such limitations. I'm extremely fortunate to grow up in a supportive environment where my family and friends were aware of my disability and yet viewed me no different than the rest of

the world. However, not everyone is aware of perinatal stroke and the science behind it. I am hopeful that by documenting my experiences in this way, people will realize that differences in ability may be part of a person's definition but is not the defining feature of a person.

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FACE IT- RECOGNIZING FACES ISN'T EASY

Sima Leah Mandelbaum, 11th Grade

Have you ever been in a room full of strangers without one familiar face? Were you filled with feelings of loneliness, constant struggle, confusion, and embarrassment? This is how prosopagnosics, individuals who have prosopagnosia or facial blindness, feel in every situation, every day.

The word "prosopagnosia" is coined from the greek words "prosopo," meaning "face," and "elleipsi gnosis," meaning "lack of knowledge." Prosopagnosics have trouble recognizing people solely based on their faces, even those they see daily, such as family members, classmates, and friends. (2) There are many facets of prosopagnosia, with only two distinct types, developmental and acquired. Developmental prosopagnosia is when face recognition abilities are lacking from early on in a person's life due to neurodevelopmental impairments. On the other hand, acquired prosopagnosia is when someone develops facial blindness due to brain damage. Atypical development of the neural face network causes developmental prosopagnosia, whereas acquired prosopagnosia is when the network becomes impaired after being developed properly. (1) Just like most neurological impairments, there is a spectrum. Each prosopagnosic is unique in the sense that each one sits in his or her own place on the spectrum.

In some cases, people who suffer from prosopagnosia can't easily imagine the face of someone they know or have trouble knowing if they've seen a specific face before. (1) Prosopagnosics can also have a hard time recognizing someone "out of context" since obvious factors are taken away. For example, their teacher at the grocery store; the teacher is no longer the only one standing at the front of the classroom therefore, isolating factors cannot be used to deduce who the person is. The possibility of not being able to recognize friends and family "out of context" gives prosopagnosics anxiety and fear. They may experience lows in self confidence because of social embarrassment and isolation. (3) Additionally, prosopagnosics have a hard time differentiating between objects, new faces, old faces, and even their own faces! In order to make up for these incapabilities, prosopagnosics rely on non-facial information for recognition. Using hair color, skin color, distinct clothing styles, voice, and/or gait are few of the many strategies prosopagnosics use to attempt to recognize people. (2) Many prosopagnosics have commented that following movie plots is difficult since they can't continuously follow individual characters throughout a film due to outfit and setting changes. (3) Although there are multiple different symptoms of prosopagnosia, there are only two distinct types of prosopagnosia.

Bodamer, an extremely influential writer on prosopagnosia, published one of the most noteworthy papers on prosopagnosia. That paper included his report from 1947 about face recognition inabilities in wounded soldiers. From those statistics, the scientific field learnt that prosopagnosia can originate from encephalitis, trauma, temporal lobe resection, stroke, degenerative atrophy, and tumors. When facial blindness develops because of brain damage, it is called acquired prosopagnosia. (5) (See Figure 1.)

Most cases that were reported before the 21st century were as a result of brain damage, such as neurodegenerative disease, head trauma, or stroke. People who have acquired prosopagnosia are usually able to recognize that there is an issue since they previously had face recognition abilities. (1)

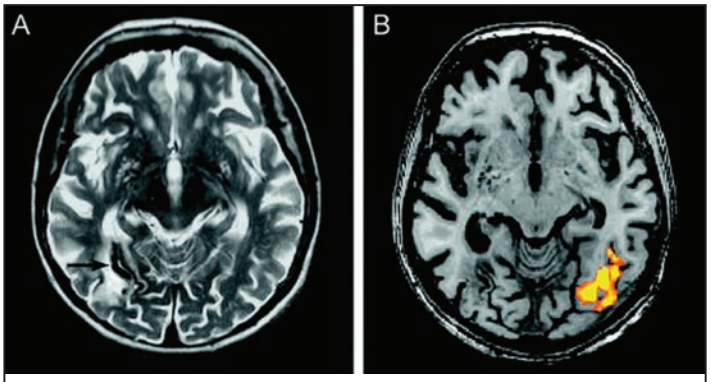

Figure 1: MRI images of a patient who developed transient prosopagnosia after an ischemic stroke.

Developmental prosopagnosia is when face recognition abilities are deficient from early on in a person's life due to neurodevelopmental impairments. (1) When looking at a brain image of a developmental prosopagnosics, there are no apparent lesions. (5) People who have normal face recognition abilities fall within a range from excellent to poor. Developmental prosopagnosics are seemingly on the low end of this range while super recognizers are close to the excellent side of this range and have outstanding face recognition abilities. (1) Developmental prosopagnosics might not realize their abnormal incapability to recognize faces and may even think everyone experiences recognition challenges since they've never experienced anything else. (3)

When individuals without prosopagnosia look at a face, their scanpaths show favor to the eyes and both the upper and center of the face, which are the facial areas that are most crucial to face recognition. In a study individuals with normal face recognition abilities showed bias towards the eyes more than the mouth, the upper face more than the lower, and the central face more than the peripheral of the face. Acquired prosopagnosics' fixations were more diverse, they focused on less crucial face regions. In contrast, developmental prosopagnosics results were similar to the control group. The conclusion of this study was that even though developmental prosopagnosics have lifelong face recognition inabilities, their scanning behavior of faces is still guided by a normal internal facial schematic. (7)

Numerous developmental prosopagnosics have other family members who have issues with their face recognition abilities and there have been families who have many developmental prosopagnosics. It can be logically deduced that there is a genetic contribution to developmental prosopagnosia. In fact, there are even studies of twins that show that inconsistencies in face recognition ability are mainly a result of genetic differences. (1) It is probable that developmental prosopagnosia is caused by genetic mutation or deletion since it seems to run in families. (2) Familial pedigrees have shown up to 10 developmental prosopagnosics within 2 generations. There also seems to be a genetic component in normal face recognition. Monozygotic twins' face recognition abilities match up more nicely than dizygotic twins'. (5) Monozygotic twins are identical and matured from the same ovum, while dizygotic twins are fraternal and are matured from two separate ova.

Functional MRI's, among other brain-imaging methods, have shown that multiple areas within the brain will react more in response to faces than to other visual groups. The right hemisphere of the brain is more crucial for face recognition than the left hemisphere, but both of them are involved. Areas in both hemispheres of the brain are linked to each other and all areas related to facial recognition operate in response to images of faces. Prosopagnosia occurs when there are issues in the face network. (1) Neurology, life experiences, and genes are all factors in the cause of and risk for developmental prosopagnosia. Approximately 2-2.9% of the population is affected by developmental prosopagnosia. To help ensure normal face recognition abilities, it is vital to expose a child to faces early on in their

life. (3)

In contradiction to a common belief, there is no connection between prosopagnosia to learning disabilities, memory loss, or memory dysfunction. Damage, impairment, or abnormalities in the right fusiform gyrus seem to be the cause of prosopagnosia. (See Figure 2.) The right fusiform gyrus is a "fold in the brain that appears to coordinate the neural systems that control facial perception and memory". It's common, but not always the case, that children with Autism and Asperger's Syndrome experience some level of prosopagnosia, which might be the reason for their impaired social development. (2)

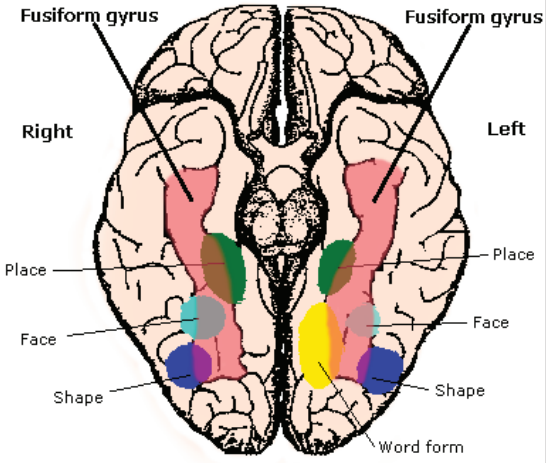

Figure 2: On the right side of the brain the right fusiform gyrus is highlighted in a darker shade of pink.

There is a correlation between prosopagnosia and medial occipitotemporal lesions, specifically on the right. FMRIs have shown a section in the right fusiform gyrus that is activated directly in face perception. (4)

Oftentimes, prosopagnosia can lead to dangerous situations. These may include, children who get lost in a public area and might not be able to find their caregiver, or children accidentally approaching a stranger thinking it's someone they know. Additionally, prosopagnosia can generate stress for students with social interactions and at school. (3) Prosopagnosia can have many social consequences for children. It may be hard for them to make friends and be a part of social interactions, they can experience extreme fear of losing a teacher/parent and being unable to relocate them, have different behavior in school than at home since they've adapted to relying on non-facial aspects of their family members to recognize them. Lastly, they may turn down jobs that have to do with recognizing people such as returning classwork to students. (3)

More than 150 years ago, the first case of acquired prosopagnosia was found. (5) It's hard to believe, but our current understanding of prosopagnosia on a neurological level is merely a glimpse of the full picture. So the next time your old friend doesn't recognize you right away, don't be insulted. You never know, they might have prosopagnosia, whether it's clinically diagnosed or not.

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A STUDY ON THE NATURE OF CONCUSSIONS

Dassi Mayerfeld, 11th Grade

One moment is all it takes. The same amount of time it takes to take a step or to snap a finger. A millisecond that to all those around is insignificant, just another moment faded into the millions they've experienced. But for one, that moment can change everything.

While almost 1.7 million people receive mild traumatic brain injuries in the US every year, little is known in the field regarding the extent of the symptoms, and more importantly, how to treat them (4). What are some of the causes of the symptoms? Why is every brain injury so different? Do concussions leave behind any symptoms permanently?

One possible cause for the symptoms of a concussion patient is a dysfunction of the autonomic nervous system (ANS). This can cause a wide range of symptoms since the ANS has many different functions in a person's body. First are the sympathetic and parasympathetic branches which are responsible for the fight and flight or rest and digest reactions. Next, the ANS has a major role in cerebral perfusion, the flow of blood in a person's brain. The ANS also helps organ systems in a person's body adjust to change both internally and externally. The ANS does this though fibers that connect it to the different organs in a person's body. Without its connection to the ANS through these fibers, the organ is unable to adjust to internal and external changes. For example, if these fibers are cut, it can affect one's ability to adjust their blood pressure, body temperature and metabolism.

According to the article, Concussion and the Autonomic Nervous System: An Introduction to the Field and the Results of a Systematic Review, it is likely that concussions affect the performance of the ANS, causing many of the different symptoms. One example of this is a concussion's effect on the blood flow in the brain. When a person performs different tasks, he uses different parts of his brain. As he uses a certain part of his brain, that part requires more blood flow in order to function properly. The ANS is responsible for sending an increased amount of blood flow to areas of the brain that are being used, bringing them the resources they need to function. One of the causes of the post-concussion symptoms experienced by many patients is a change in cerebral blood flow, thus preventing different areas of the brain from receiving the extra blood flow they need while they are being used. As a result, these patients do not experience optimal brain performance that is enabled through this function of the ANS. Another observation that points to the fact that ANS dysfunction is the cause of many post- concussion symptoms is that patients who have other medical conditions, especially those with dysfunctions in some of the ANS processes above, suffer from similar symptoms as those who received a concussion. These symptoms include headaches, nausea, anxiety, memory and attention dysfunction and fatigue. This observation suggests that a concussion may affect the ANS, thus giving both types of patients similar symptoms. Since the ANS controls so many functions of a person's body, ANS dysfunction post injury can explain the range of symptoms as well as the uniqueness of the symptoms in every case due to where the patient was hit and which areas of the ANS were affected (3).

Another explanation for the uniqueness of each concussion, the severity of its symptoms and the duration of the symptoms is related to the axons that are damaged during the time of injury. It has been observed that depending on the group sampled, the answers to these questions are different. For example, it has been found that symptoms last longer in non-sports related brain injuries (3). A difference has also been noted between samples of women and those of men. Some research has suggested that

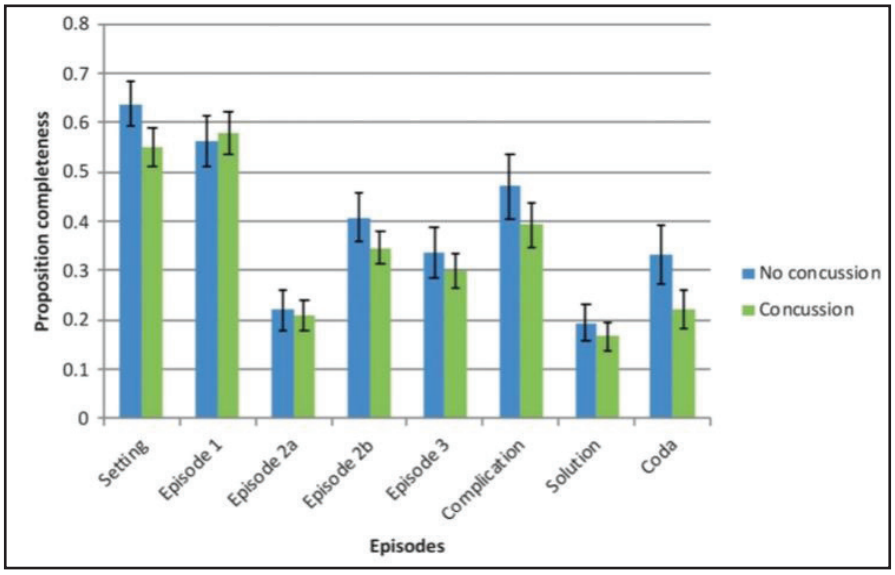
women are more prone to receiving concussions and can experience more severe symptoms for a longer period of time than their male counterparts. One suggested cause for this difference is the difference in the structure of a woman's axons compared to a man's axons. When a person receives a concussion, axons are damaged and that is one cause of the post- concussion symptoms. Because women's axons are thinner, they undergo more structural damage during a concussion than those of a man and as a result they suffer from worse and longer lasting symptoms than men do (2).

Some of these symptoms include deficits in a person's cognitive and linguistic skills. In the past, many studies have observed language deficits right after a person receives a mild traumatic brain injury. These deficits can affect one's verbal fluency, ability to repeat over sentences, and ability to transition between two topics. It can also affect one's ability to relate a story with an introduction, conflict and resolution, in a clear manner.

Additionally, deficits relating to a person's attention span and abilities to process, remember and recall new information increase as they are asked to perform more tasks. One cause for this is that as they do more, their cognitive resources are reduced and as a result, they cannot process the information as efficiently. But, these deficits post injury are believed to heal over time. Contrary to this belief though, many people who received concussions report to finding cognitive and linguistic tasks more of a challenge even after healing from the injury, suggesting that these deficits might have a more permanent nature.

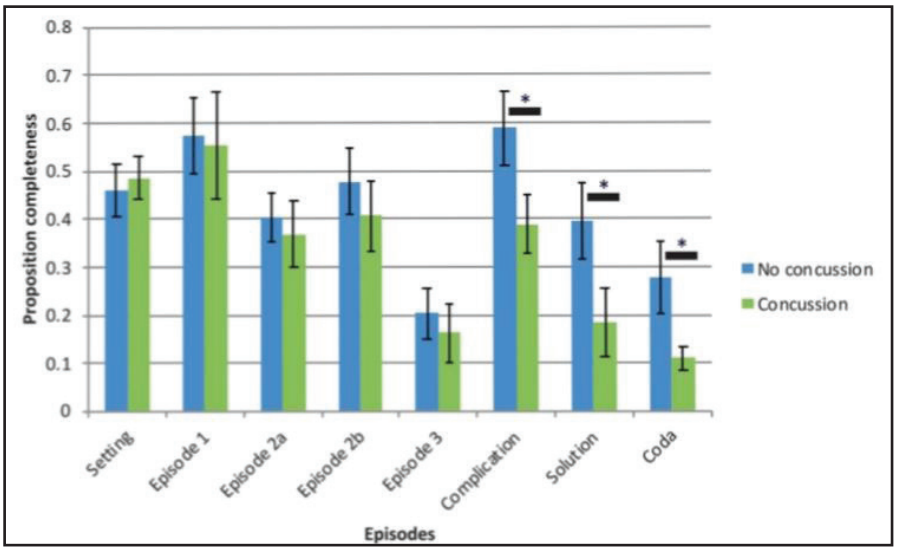
In their experiment, Melissa D. Stockbridge an Rochelle Newman study a group of subjects between ages twelve and forty and compare their abilities regarding different cognitive skills. During the experiment, the test subjects had to relate a narrative that they were familiar with, the story of Cinderella, and then a narrative of a new story they were shown in a video.

Those in the test group who suffered from a concussion at some point in their past performed almost equally as well as those who never experienced a brain injury in the tasks that required them to use one skill at a time, like recalling and retelling the Cinderella story.



On the other hand, those who had suffered from a concussion faced more of a challenge when asked to summarize the content of a new and action packed video they had just been shown, a task that required the use of many cognitive skills. Many of the people who had a concussion in their past left

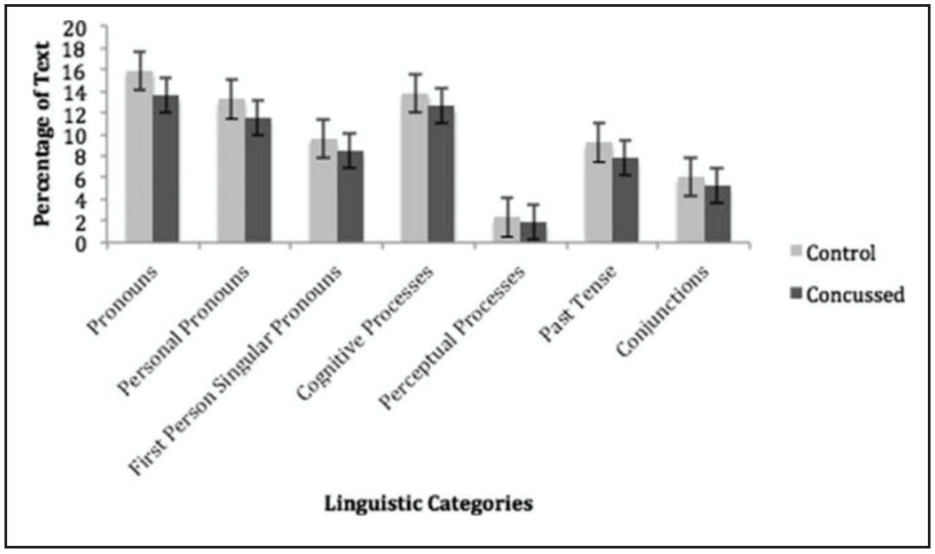
out important details when retelling the story or ran out of five minute time frame to relate the details.



This study shows that a concussion can affect one's ability to do tasks that require the use of multiple cognitive and linguistic skills and that these effects can last many years post the concussion even after most of the concussion symptoms have gone away (4).

Memory is another area affected by concussions. The concept of memory is also something that is very important in Judaism. As Jews we have Mitzvot to remember things from our past every day. In שולחן ערוך אור החיים סימן ס׳ מגן אברהם סעיף קטן ב׳, the שש זכירות are mentioned, the source for the six things that we are supposed to remember every day. These remembrances are about events from our past, not as individuals, but as a nation. They include זכירת יציאת מצרים, זכירת מעמד הר סיני, זכירת מעשה עמלק, זכירת מעשה העגל, זכירת מעשה מרים וזכירת שבת. This source discusses how we fulfil this obligation of remembering through hints to these six remembrances in אהבה רבה. Today many people also recite the שש זכירות after davening. This is just one example of a מצות עשה that we must fulfill through our memory.

Nicole C Barry and Jennifer L Tomes performed a study to observe whether a concussion can permanently impact one's memory. In their study, they asked a group of individuals to relate autobiographical memories, personal memories of experiences they had. They found that test subjects who had suffered from a concussion described memories that were more general and simple while those who had not received a concussion recalled more vivid memories, remembering specific names and places and used an increased amount of perceptual process words, past tense words, and cognitive process words as seen in the results below.



One cause of this is the vulnerability of the medial temporal and frontal lobes. When one receives a concussion, both of these areas are vulnerable to injury and both of these areas are responsible for retrieving memories. As a result, a concussion that causes damage in those areas can have a more permanent effect on one's memory(1).

Whether caused by axon damage, ANS dysfunction or another cause, concussions can have a permanent effect in areas including language, cognition and memory.

That moment, so minute in size but giant in impact, can change a life forever.

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ADDICTION FICTION: HOW CAFFEINE GETS YOU HOOKED

Tziporah Pinczower, 10th Grade

Thomas Jefferson commented that coffee is "the favorite drink of the civilized world (1)." Since time immemorial, intellectuals would congregate in coffee houses, forming an impromptu democratic club, open to everyone for the price of a cheap cup of coffee.

Every guest could sit for hours to talk, write, play cards, receive posts, and above all consume an unlimited number of newspapers and journals. Others were opposed to coffee as they feared it would threaten their beer-focused economy, or as Fredrick the Great declared in order to preserve societal norms, " My people must drink beer." Coffee houses attracted diverse thoughts and discussions, prompting Mark Pendergrast to publish *Uncommon Grounds: The History of Coffee and How It Transformed Our World* in 2010. In places such as Boston, coffee became the ever-increasing popular alternative to tea after the Boston Tea Party. Not long after, European Jews began arriving on the shores of America and along with the influx of Jewish migration came coffee cakes. New York became a hot-spot for Jews to begin marketing coffee eventually producing Starbucks, founded by the Jewish Howard Shultz in 1987. (2)

The American Psychiatric Association defines addiction as a "complex condition, a brain disease that is manifested by compulsive substance use despite harmful consequences (3)". While one may apply this definition to alcohol or drug abuse, the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders have included caffeine withdrawal as a mental disorder in their 2013 publication (4). According to the Food and Drug Administration (FDA), more than 80% of Americans rely on their daily dose of caffeine, calculating to around 280 milligrams per day (5). Despite this ever-escalating number, professionals such as Dr. Griffiths claim, "People talk about being addicted to coffee. We know from laboratory studies that people can get physically dependent on caffeine and show tolerance to it, but that doesn't mean they have great difficulty giving it up (5)." Dr. Griffths maintains this view due to the American Psychiatric Association's four criteria for identifying an addiction: "withdrawal symptoms, development of tolerance over time to the effects, use of the substance in spite of aggravation of medical or mental problems, repeated unsuccessful attempts at quitting (5)". Defending his claim of ability to halt caffeine consumption, Dr. Griffiths points to the last requirement as grounds to disregard the remaining three. In contrast to this view, reports of distressing and painful caffeine detox cause many to speculate the seemingly innocent caffeine consumption. Symptoms reported include headaches, lethargy, constipation, irritability, depression, muscle pain, nausea, and insomnia (6).

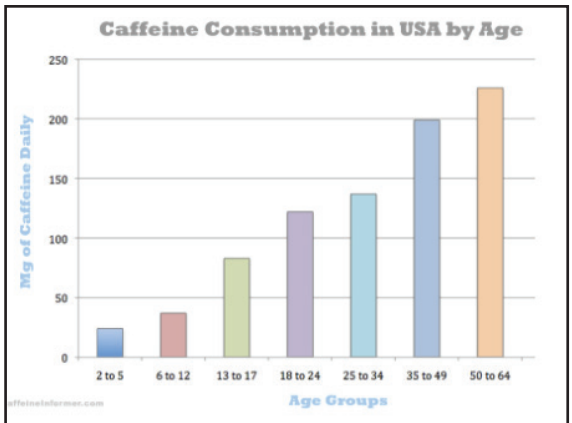

Figure 1: The average caffeine intake of Americans, showing our increasing reliance upon it.

Set on determining the percentage of *true* (those who fulfill the fourth requirement) caffeine addicts, Dr. Griffiths conducted a controlled experiment with eleven individuals,

under the impression that this was determining the effect of food on one's mood. Assessing whether something is an addiction is harder than simply calculating the intake of the average individual as metabolism varies as well. Each participant was given a pill to take every day during the two-day duration of the experiment. Some were given pills containing the same amount of caffeine they had reported digesting daily while the others were given placebos. All of the participants reported having difficulty during this time and some even made drastic changes to their days. In retrospect, Dr. Griffiths reports that although caffeine is a drug and can clearly be difficult to withhold from, one can gradually cease their intake, therefore rendering caffeine a drug, not an addiction (5).

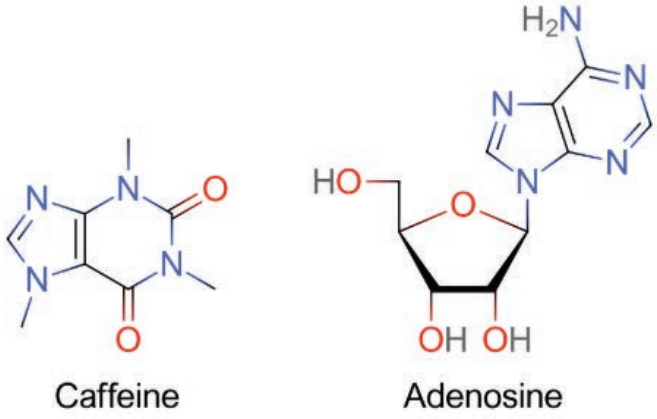

Figure 2: The similar chemical makeup of caffeine and adenosine, resulting in adenosine receptacles producing energy with caffeine in place of adenosine.

Fortunately, caffeine has positive effects as well, and a moderate intake of caffeine is actually beneficial and arouses vigilance at times when one may lack concentration. Withdrawal symptoms for those decent caffeine consumers will not provoke an outrageously difficult process (7). Many may be wondering, how does caffeine work? When one intakes caffeine it enters their bloodstream and makes its way up to their brain where the adenosine receptors are. Adenosine is meant to be accepted by the adenosine receptors and the adenosine level increases with every hour you are awake, telling your body to slow down and making you tired. Caffeine, due to its similar chemical makeup is the perfect imposter and once it arrives in the brain, connects with the adenosine receptors, blocking the adenosine from ever reaching their destination and preventing them from telling your brain to slow down, so the blood rate and pressure continue at the same pace, never dwindling. Eventually, the caffeine molecules detach from the receptors, and

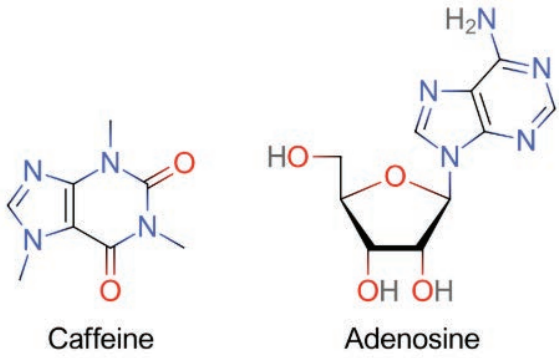

Figure 2: The similar chemical makeup of caffeine and adenosine, resulting in adenosine receptacles producing energy with caffeine in place of adenosine.

adenosine takes its place, this is when one may suddenly feel very tired and worn-out because their body hasn't been slowing down at the appropriate pace. The next morning one may have withdrawal symptoms that prompt them to have another cup of coffee, turning this seemingly innocent drink into a vicious addiction. This pattern is responsible for morning coffee becoming a staple in our modern culture, where one must have an active and engaging mind early in the morning and cannot risk the adenosine production. Those who consume a moderate amount of caffeine daily, (from 100-400 milligrams, around the amount of four cups of coffee) are still susceptible to its addictive qualities. Additionally, the routine intake of caffeine may lead to a sort of immunity to its effects, resulting in ever-increasing quantities in order to receive the same effect (8).

Coffee has a rich history across the globe and remains one of the most traded commodities today, causing some to worry that we may have become too dependent on its supply and consumption. Therefore, dear Manhattaners, if one requires a quick dose of caffeine to perform a task that requires more than momentary vigilance, this may be an efficient aid. However, it is critical to be wary of your intake and not let it dominate one's system lest they become reliant on it.

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ADVANCEMENTS IN DNA TECHNOLOGY

Ariella Shloush, 11th Grade

In August 1987, a father and son were both brutally murdered in front of a storefront in Hamden, Connecticut. The officers found the father and son's bodies next to each other on the bedroom floor of their apartment, their chests full of stab wounds, their throats slashed and both of their hands tied behind their backs.

As hard as the police tried, they could not come up with enough physical evidence to link their prime suspect, Willie McFarland – a man who had actually confessed to the crime – to the murders. Because of this lack of physical evidence, they were never able to arrest and jail the killer and this Hamden murder case was put away, labeled a cold case by the police department despite a confession and much speculation but no real evidence.

Thirty two years after the confession of Mcfarland and 'closing' of the case, the police finally arrested him. During the time that had passed between the confession and the arrest, the forensic world had been steadily researching and advancing. One of the main advancements was a new method in which investigators were able to extract very small amounts of DNA from pieces of evidence and "amplify" it, or copy it multiple times, so that it could now be read and analyzed. Other techniques were advanced and developed that allowed mixtures of DNA to be separated and analyzed. Evidence found at the crime scene that was previously useless to the labs now became viable for admission into DNA testing, and later into court as a piece of prime evidence.

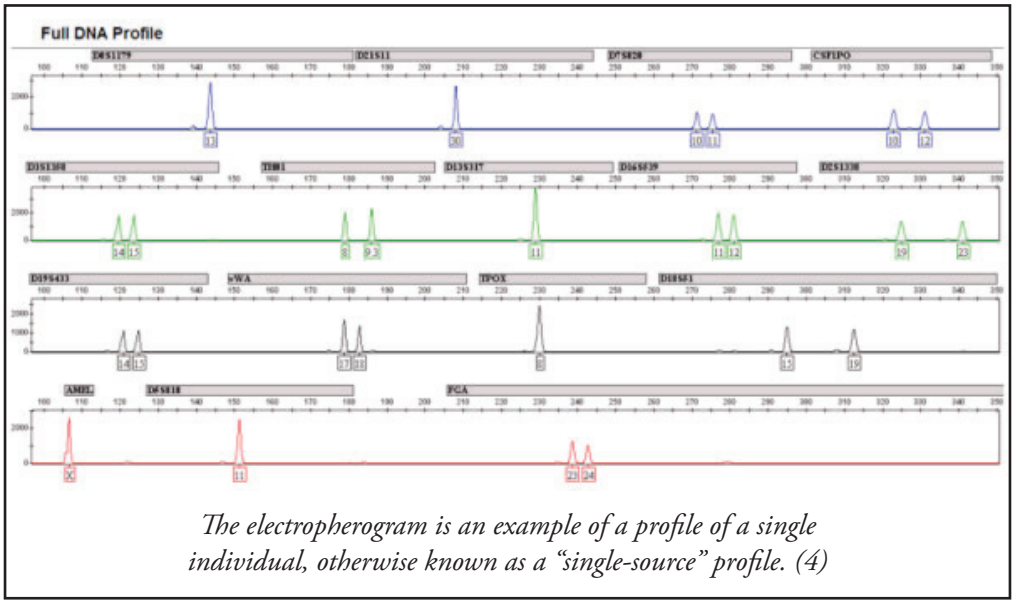
This is exactly what happened in the McFarland case: A glove found at the crime scene, previously serving no purpose in the investigation as they had no real way to link it to their suspects, was put under the new testing methods and came up with a mixture of DNA and found inside the mixture was DNA that matched Willie Mcfarland. This DNA evidence, combined with his startling personal confession, put him in jail for life. (1,2)

As technology advances, forensic scientists are able to analyze smaller and smaller biological samples to develop a DNA profile. However, in a case like this one, where the DNA found on the glove was a mixture, how do forensics specialists analyze the mixture and even more so, match the mixture to a suspect?

These DNA samples go through a specific and certain process. First, extraction, where the DNA is released from the cell. Then Quantitation, the determination of the amount of DNA. The DNA then gets amplified and multiple copies of the DNA are made through a process called Polymerase Chain Reaction, or PCR for short. Then the amplified DNA gets seperated to process identification. After, the DNA goes through analysis and interpretation, where the DNA evidence is compared to other known profiles. Finally comes Quality Assurance, the process of reviewing the procedure and ensuring technical accuracy. (4)

The process is then continued when the DNA analysis is done on a chart called an electropherogram, which displays the genetic material present at each loci tested. In a complete profile, each person will exhibit either one or two peaks (alleles) at each locus. (7)

The way the scientists are able to differentiate between all of the DNA in the mixture is by



The electropherogram is an example of a profile of a single individual, otherwise known as a "single-source" profile. (4)

combining the process of matching up STRs with other genetic markers. STRs, or short tandem repeats, occur when two or more nucleotides repeat multiple times within the DNA. The number of repeats vary from person to person and that is one of the distinguishing characteristics of the individual. No two people have the same number of repeats at each of the STR loci. In addition, they can use SNP-STR that detect small changes in a nucleotides DNA code and DIP-STR, which detects the minor and major contributors to the mixture based on the STRs. The SNP, otherwise known as single nucleotide polymorphisms, is a random single substitution change in one nucleotide in one STR locus, further adding another dimension to the variation in the DNA from one person to another. DIP (Deletion/Insertion Polymorphism) is yet another type of variation within a STR that is a result of an extra nucleotide insertion or a nucleotide deletion. These combined genetic markers, as well as parallel sequencing technology that allows the different pieces of DNA to be sequenced at the same time, decreases the risk of the DNA extracted being a jumbled combination of all the DNA with nothing actually trustworthy in it. (5) Once the mixture is separated, and the different DNA is analyzed and profiled, the police begin the task of searching the profile in the national database. (3,6)

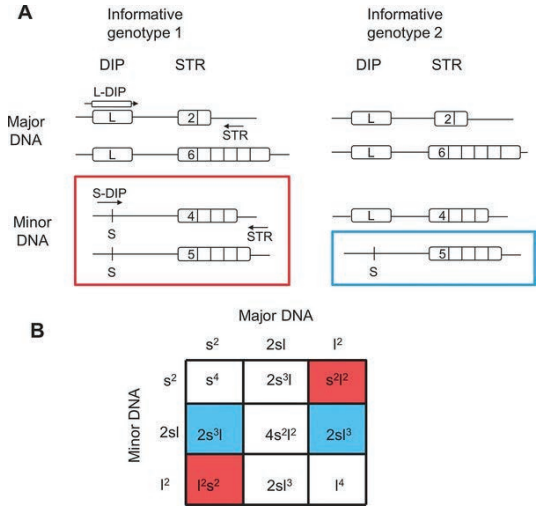


Figure A is displaying DIP-STR genotypes. Figure B is a theoretical analysis of the occurrence of the markers. In red are the probabilities of DIP genotypes to enable the identification of two minor DNA haplotypes, while the blue is showing the probabilities of the DIP genotypes enabling the identification of one minor DNA haplotype. The sum of these values shows the probability of having any informative genotype in a mixture of two DNA. (8)

To enable profiles to be searched in a national database, the FBI created the National DNA Index System (NDIS) in 1998. This national database is part of the Combined DNA Index System (CODIS) that enables investigators to share and compare DNA profiles to help investigate and solve cases. Once a laboratory enters their evidence from a case into CODIS, a weekly search is conducted of the DNA profile, and the resulting matches are then returned to the original laboratory. (4)

When describing this case, Captain Smith, the head of the police department who worked this case, said this was a case that "there was always hope of solving." The police knew what to do, they just needed a way of doing it. They had the glove with the precious DNA that could close the case. They just needed a way to make it useful to crack the case. These new methods allowed the Hamden case to be reopened, solved and closed for good, and in the future will do the same for many other cases that also lack sufficient and pure DNA evidence. This illustrates a crucial and important lesson. Never throw away any evidence. Even if it is not useful now, with future developments in technology, it may be the key to locking the door on criminals and finally giving closure to the families of their victims.

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A MOMENT IN THE SUN

Sarah Strauss, 12th Grade

The sun's rays in the morning are exciting and give motivation to start your day, but Raphi's shutters are closed. You need fresh air and so you go on a walk but Raphi is stuck inside until nightfall.

Today with Covid-19 we are all sort of stuck at home. However, people could still go on social distancing walks during the day and yet people don't stop complaining about the situation. Raphi Strauss is a Xeroderma Pigmentosum patient. Imagine being stuck at home with Raphi's condition your entire life.

Xeroderma Pigmentosum, XP for short, is an inherited skin disorder characterized by an extreme sensitivity to ultraviolet (UV) rays which can come from sunlight. (1) Sources of UV light also include unshielded fluorescent light bulbs, mercury lights and halogen light bulbs. (2) Anyone can receive damage to their skin from the sun but the body repairs the harm done. However, in XP patients the body does not fix the damage because they have a mutation. (6) This rare genetic syndrome affects all areas of skin of an XP patient that was exposed to UV rays. Depending on the type of XP a person has, is the severity of the disorder. For example, some affected individuals can even have problems with their nervous system.

One can observe signs of XP even at the young age of infancy. At such a small age, the baby will receive a severe sunburn from spending just a few moments in the sun. Over time, the effect of the UV rays is penetrated, and the first thing that a person with XP will develop is a lot of freckles. These freckles cover the areas of the body that were exposed to UV. Exposure to UV light also causes these patients to have unusually dry skin and changes in the pigmentation of their skin.

To inherit Xeroderma Pigmentosum, one must carry the two recessive autosomal genes, which means the parents of a patient with XP had to have each carried one copy of this mutated gene. Carriers typically do not exhibit any signs of this disorder; they merely possess it in their DNA. (1) There are tests that can be done during pregnancy to see if the fetus has XP or not. These tests consist of Amniocentesis, Chorionic villus sampling and Culture of amniotic cells. (10)

The first time XP was described was in the year 1874 by Hebra and Moriz Kaposi. In the year 1882, Kaposi coined the term Xeroderma Pigmentosum, which describes the symptoms of the disorder, Xeroderma meaning dryness and Pigmentosum meaning the discoloration that both occur to the skin. Later, it was discovered that Xeroderma Pigmentosum also causes patients to develop skin cancer from sun exposure. (3)

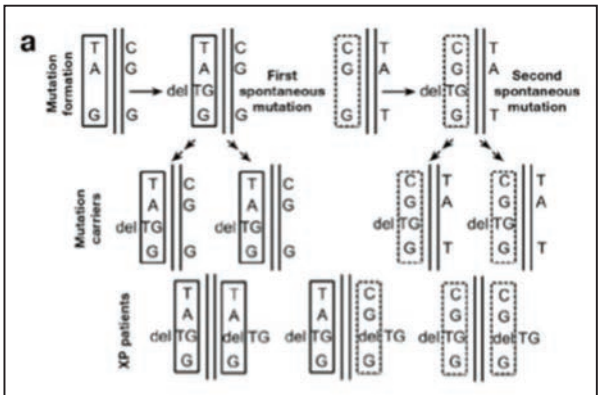
Most forms of XP are caused by a defect in nucleotide excision repair of DNA damage in skin cells associated with sun (or any UV light) exposure. This discovery was made by the scientist James Cleaver in 1968 almost 100 years after Kaposi first established the name Xeroderma Pigmentosum for this rare disease. There were recurring events that took place, rarely, but enough to make doctors wonder and start to research this topic. The phenomenon that took place over and over again was that an infant or small child was taken outdoors into the sun. After a short time, the child started to cry and became irritable. Not that long after the exposure, the child's skin turned very red, blistered and swollen. The skin did heal but very slowly. The healing didn't help since this scenario happened every time the child was exposed to UV rays. At the age of two, the child started to develop freckles all over the skin that was exposed to UV rays. As the child grew older, the cute looking freckles turned into

dangerous and even life-threatening tumors and skin cancers. That was when the doctors realized that this is a rare and irregular pattern. In simple terms, patients with Xeroderma Pigmentosum are allergic to the sun and any UV light. (5)

Individuals who have XP, develop skin cancer very often. Without sun protection most children with this condition develop their first skin cancer at the significantly young age of ten. There are different types of skin cancers, some more severe than others and most XP patients develop a range of various types of skin cancers. (1) Patients with XP must go to a dermatologist once a month to check for any suspicious spots on their skin. This is because of the high risk of the development of skin cancer that they have. If there are areas of the skin that look different than usual they will be biopsied and sent to a lab for testing. If the skin tested came back positive for skin cancer, the patient must then undergo surgery to remove it.

In a medical peer-reviewed journal on genetics, the importance of the polymerase chain reaction was tested. The gene responsible for the Xeroderma Pigmentosum (XP) group, A gene, was identified and isolated. The mutation site in the XP group A that was found in the complementing gene was recognized by a restriction enzyme, *Alw*NI. This article described the usefulness of the polymerase chain reaction diagnosis on XP group A. The scientists tried a polymerase chain reaction diagnosis on genomic DNA from a patient with XP group A and they also tested the patient's parents. *Alw*NI of the polymerase chain reaction product showed three bands one was the patient's father's and two bands was the mother's DNA. One band corresponds to normal and two bands correspond to a point-mutated gene. A point-mutated gene is when a single nucleotide base is changed, inserted or deleted from a sequence of DNA or RNA. When the point-mutated gene was replicated and passed down to the child, that is when the XP syndrom formed. A DNA sample from the patient showed two fragments indicating the patient was the only one with the mutation. They concluded that there is a way to detect the hidden mutated XP group A by the complementing gene in the pedigree.(7) Interestingly, because of this specific mutation, it is unlikely for somebody with the condition to pass it on to the next generation.(6)

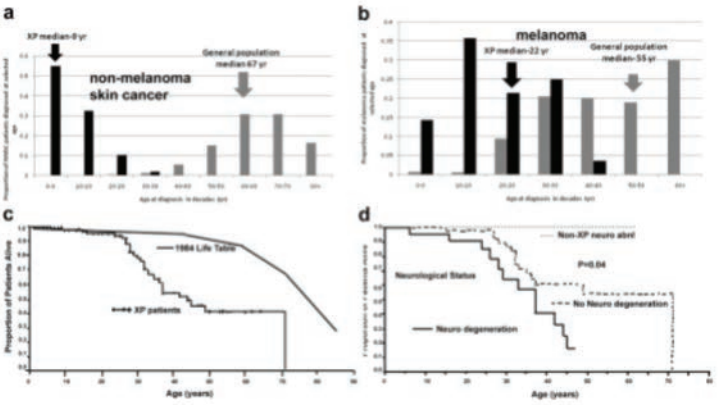
The flow chart on the right depicts the way a mutation is formed by deletion of a nucleotide because the DNA Polymerase cannot read the mutated nucleotide. The nucleotides get together by base pairing and Adenine goes to Thymine and Guanine goes to Cytosine. Therefore, when there is this mutation of TG nothing pairs to it leading the DNA to delete that base pair. "Over generations these individuals pass the delTG mutation and adjacent regions to their progeny, who are asymptomatic carriers with one mutated allele and one normal allele (center row). Eventually, carriers of the delTG mutation have children with the same mutation in both alleles (bottom row). All of these affected individuals (XP patients) are homozygous for the delTG mutation. However, different XP patients have different haplotypes (TA (delTG) G, CG (delTG) G, or both)." (8)



XP Mutation Formed By Deletion

Every person is prone to skin cancer from the accumulated damage of sun exposure for many years. This usually occurs during the mid-fifties and older. However, in XP patients, skin cancer can

develop at the very young age of nine years old. The graph shows the average age of a normal person receiving skin cancer for the first time versus the first time an XP patient develops skin cancer. (9)



Age of first skin cancer; XP Patient Versus Healthy Patient

XP patients cannot go out into the sun just like any other person. If they do need to go outside during the day they must wear appropriate attire. Raphael Strauss, who has XP, can only go outside during the nighttime when there is no sunlight at all. Some people think that if it's cloudy out he can leave the house with no special uniform. However, the sun's UV rays still shine through the clouds and damage the skin. Therefore, if it is necessary for an XP patient to leave while the sun is still shining, they must wear a mask to protect them from the UV. Raphi uses a mask which was made with material meant for astronauts.

Perhaps, now that we have experienced the extended stay at home policy due to the COVID pandemic, it is easier to empathise with someone who must be stuck at home the entire day with such a harsh disorder. However, XP patients can always find the light through the darkness and hopefully one day a cure to this disorder will be discovered. Raphi Strauss is my brother; he is the most positive, heroic person I know, and a true role model for the rest of us.

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