FIELDSTON SCIENCE BULLETIN

ISSUE 1 // VOLUME 1 // DECEMBER 2024

THE WINTER BREAK ISSUE

Table of Contents

EDITOR IN CHIEF..... CRISTINA ELLIS

FACULTY ADVISOR..... MS. BEATON

LET IT SNOW? THE EFFECT OF GLOBAL WARMING ON SNOWY HOLIDAYS

Cristina Ellis

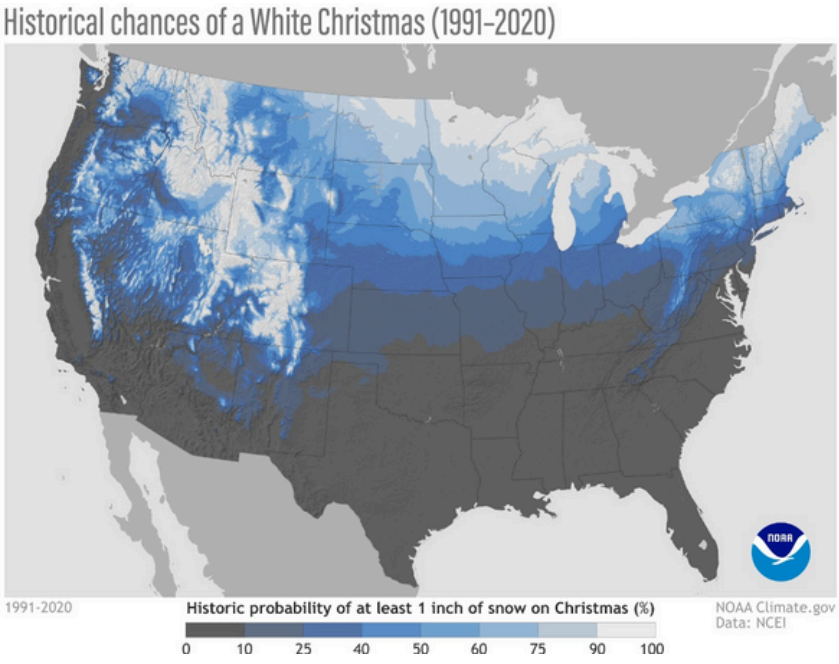
The holiday season has become inextricably intertwined with the idea of snow. From songs such as Bing Crosby's, I'm Dreaming of a White Christmas, to movies filled with snowy holiday magic, the idea that it should snow during December has proliferated the public consciousness. So why is it that so often our Decembers are utterly devoid of snow? The answer is simple, it is snowing less.

Global warming has driven the average temperature of the Earth up by around 2.11℉. You might think the implications of this small shift aren't that meaningful, but even small shifts in average temperature can change weather patterns. For example, during the last ice age, the average temperature of the Earth was about 11℉ cooler than it is currently. At that time, vast swaths of the earth were covered in glaciers, including the entirety of Canada and the northern United States. Sea levels were far lower, exposing masses of land that are currently underwater, linking several continents to various islands.

But what does all of this have to do with snow? Well, it does not take that much of a decrease in average temperature to create an icy world, so conversely, it is logical that as the average temperature of the Earth

increases, the amount of snow and ice will decrease. Justin Mankin, an associate professor of geography at Dartmouth who co-authored a study on the the impact of climate change on Earth's snowcap, told CNN that we shouldn't expect a gradual linear decrease in the amount of snow, instead once the planet reaches a certain temperature, "we should expect the losses to accelerate." Mankin continued by stating that it is nowhere safe from the decline in snow, "We can expect a lot of the places that haven't exhibited massive snowfall declines to maybe start to exhibit them with just a little bit more warming."

According to CNN, since 1973, there has been a 2.7% decrease in snow across the globe, particularly affecting the northern hemisphere's middle latitudes, where the United States is situated. In parts of the southern and western United States, there has been a 30% decrease in snow totals. This decrease has very serious implications for people who live in these regions because melting snow, aptly called snowmelt, is a crucial source of water. As the amount of snow continues to decrease, that supply is drying up, resulting in droughts. This problem is not unique to the United States, as parts of southern Asia, the Mediterranean, and Northern Africa also rely on snowmelt as a source of water. Unless we make significant changes in how we address climate



change and this pattern of reduced snow is reversed, significant changes to water distribution systems will be needed.

But now, before we conclude this article, let's return to the question of whether we can look forward to snow this holiday season. In the Greater New York City area, during the past thirty years, 10 percent of Christmases have had at least one inch of snow. While this statistic may be discouragingly low, it is important to remember that the Holidays are a time of hope. In addition to wishing for a happy holiday with friends and family, wish for more snow, not only here, but around the world.

DOES TURKEY MAKE YOU SLEEPY?

Kevin Nam

People have long believed that turkey makes them sleepy, but is this true, or just an old wives' tale? Science has an answer.

Turkey contains an essential amino acid called tryptophan. This acid creates serotonin, which in turn makes melatonin. Both of these hormones help regulate mood, and the latter also help with sleep cycles. However, despite tryptophan being present in turkey, it does not appear in excess amounts compared to other

foods; research shows that chickens have more tryptophan than turkeys. Tryptophan is also found in most other kinds of poultry, as well as nuts and seeds.

So if turkey isn't the culprit, why do people feel tired after Thanksgiving? This fatigue is most likely caused by overeating because digesting a large amount of food (2 or 3 plates worth) requires a lot of energy, causing you to feel tired. Another reason may be the excess amount of carbohydrates consumed, which release insulin that helps remove glucose and amino acids from the blood. This allows tryptophan to pass throughout the body more easily, and thus get into the brain and release serotonin and melatonin. In general, the holidays are a stressful and exhausting time, contributing to people feeling tired.

So, how can you feel more awake this Thanksgiving? Experts say that exercising, eating more slowly, and drinking less alcohol can all make you feel less tired. In addition, eating a protein and fiber-filled breakfast will help prevent you from overeating during Thanksgiving dinner.

Turkey has long been unfairly blamed for the tiredness people feel during Thanksgiving; In reality, a variety of factors cause people to feel this fatigue that aren't related to turkey.

HOW DO SNOWFLAKES FORM?

Francesca Master

Snow is one of the most important aspects of winter, yet very few people understand the science behind it. Have you ever wondered how snowflakes form and why each one is unique? We have the answers for you!

Snowflakes aren't actually pure water, instead, they are drops of very cold water which freeze onto pollen or dust particles in the sky, forming an ice crystal. As the ice crystal falls to the ground, water vapor freezes onto it, creating new crystals that develop into the six arms of the snowflake.

So, why do no two snowflakes look the same? While all ice crystals are symmetrical and have six sides, several factors influence their individual characteristics. For instance, at 23 degrees Fahrenheit, ice crystals tend to be long and needle-like, whereas at 5 degrees Fahrenheit, they take on a flatter shape.

The atmospheric conditions also play a significant role in determining the appearance of a snowflake. Changes in temperature or humidity as the ice crystal descends can cause it to grow in various ways. Since each snowflake takes a unique path to the ground and encounters different conditions, none look identical.

Snowflakes are a special yet often misunderstood part of winter. While many people are aware of certain aspects of their creation, they may not fully understand how or why each snowflake's formation is unique. Take a moment to appreciate their beauty this winter.

FIELDSTON SCIENCE BULLETIN FUN FACTS

FSB Editors

The holiday season is full of distinctive traditions and symbols, such as turkey, candles, pine trees, and (sometimes) snow. Here are some scientific fun facts related to traditions and objects we all love.

Why is Thanksagiving Turkey golden brown?

Browning of food such as roast turkey, french fries, or biscuits due to a chemical reaction called the Maillard Reaction. It occurs when amino acids and sugars are heated to around 300 Fahrenheit. The chemical reaction produces complex compounds that, in addition to producing a brown color, gives food distinct flavors and aromas that we all love.

What happens when candles burn?

Parafin wax consists of hydrocarbon molecules. When a candle is lit the heat of the flame melts the wax near the wick. This liquid wax is then drawn up the wick where it is vaporized by the flame and hydrocarbons break up into hydrogen and carbon molecules. These molecules react with oxygen creating heat, light, water vapor, and carbon dioxide.

How do pinecones form?

Surprisingly, pinecones are a type of woody fruit that contains pine tree seeds. They develop when pollen from male pine flowers fertilizes the egg cells in female pine flowers, which

then take about a year to develop into mature pinecones. Most pine trees have both male and female flowers on the same tree. In the spring, male flowers or cones grow at the tips of branches; they are smaller and softer than the female flowers which mature into the brown, scaly cones most associated with pine trees. Male cones generally grow on the lower branches of pine trees, and female cones usually grow on the upper branches of pine trees. Pine pollen from the male flowers is distributed by the wind.

Why does snow look white?

While sunlight looks white to us, it is actually composed of different colors with different wavelengths. An object's color is determined by the wavelengths of light that it does not absorb, and hence reflects. Clean snow looks white because it reflects back most of the visible sunlight. On the other hand, dirty snow can take on a whole host of unpleasant colors.

Why is the winter solstice the shortest day of the year?

The Earth is tilted 23.5 degrees relative to the sun, so the Northern and Southern Hemispheres receive different amounts of sunlight depending on the time of the year (this causes the seasons!) In the Northern Hemisphere, the winter solstice occurs when the North Pole is at its farthest tilt of 23.5 degrees away from the sun, which causes the area to be plunged into total darkness. While this is going on in the north, the south pole is simultaneously experiencing its summer solstice or the day with the greatest number of daylight hours. This year's winter solstice will occur on December 21st.

How are Reindeer feet adapted

to snow and ice?

Reindeer (Rangifer tarandus) are a species of deer found in the Arctic tundra, Greenland, Scandinavia, Russia, Alaska, and Canada. (If you're wondering why you've never heard of reindeer in the United States, it is simply because we call them caribou.) While there are a myriad of interesting facts about these animals, one in particular is about their foot adaptations. Their feet are very broad (like snowshoes), which prevents them from sinking into the snow. They also have cloven hoofs and fur on their feet, which acts like treads beneath their hooves. This allows them to move quickly over snow while maintaining a firm grip on slippery and icy surfaces.

ONE COPY OF CX3CR1 CRE ALLELE IS AS EFFECTIVE AS TWO AT GRIP1 SUPPRESSION THROUGH A CRE-ERT SYSTEM

NATHANIEL ROZOFF (FORM VI)

Abstract:
Microglia, the resident macrophages of the CNS, can promote both MS and its remission due to their innate phagocytic properties. The GRIP1 protein influences the role of microglia in MS and is the focus of ongoing research to develop treatments for the disease. To enable future investigations, methods for GRIP1 suppression must be confirmed. This experiment examined whether or not one copy of the Cre promoter would be as effective as two copies of the Cre promoter at mediating GRIP1 expression through the Cre-ERT system. Using RNA isolation, RT reactions, and qPCR assays, researchers found that one copy of the Cre promoter was indeed as effective as two copies of the Cre promoter at inhibiting GRIP1 activity in mouse microglia. The researchers hope this work will enable future research regarding GRIP1's role in inflammatory diseases.

Introduction:
Multiple Sclerosis (MS) is an immune disease that affects over 1.8 million people worldwide. Various forms of MS exist. Clinically Isolated Syndrome (CIS), characterized by early neurological symptoms, is often the first sign of MS.

Relapsing-Remitting Multiple Sclerosis (RRMS) is the most common form of the disease and is characterized by periodic onsets of symptoms - relapses - followed by a period of remission in which patients experience little discomfort. Secondary Progressive Multiple Sclerosis (SPMS) can present after a patient has RRMS.

A patient with SPMS does not experience the same relapses and remissions that characterize RRMS - they are continuously affected by their symptoms. Finally, Primary Progressive Multiple Sclerosis (PPMS) entails a gradual progression of symptoms from the initial onset, with no clear relapses or remissions (World, 2023). Symptoms vary among patients; vision problems, difficulties with walking, balance, and processing information, weakness and stiffness in the arms and legs, depression, and fatigue are all common.

MS can affect anyone, but some demographic, genetic, and epigenetic factors correlate with instances of the disease. Demographically speaking, MS occurs 2 to 2.5 times more frequently in women compared to men. It also occurs most frequently in those of Northern European descent (Calabresi, P. A., 2011). In terms of genetics, relatives of MS patients have a significantly higher chance of developing the disease as opposed to members of the general population (Noseworthy, J. H., Lucchinetti, C., Rodriguez, M., & Weinshenker, B. G., 2000). Epigenetic factors include smoking, obesity, life at lower latitudes, Vitamin D deficiency, and infections like Eppstein-Barr Virus (Ascherio, A., & Munger, K., 2008) (Ascherio et. al, 2015).

The specific pathogenesis of MS is unknown and is the subject of significant ongoing research. Generally, researchers understand that the degradation of the myelin layer around nerve fibers causes the symptoms of MS (Multiple Sclerosis (MS), 2023). Myelin, made of proteins and lipids, insulates nerve fibers like insulation around electrical wires. When the myelin layer, known as the myelin sheath, is damaged, electrical impulses that travel through the nerve fibers slow or even cease (Myelin: MedlinePlus Medical

Encyclopedia, 2020). This disruption of neural signals causes the symptoms of multiple sclerosis and other neurological diseases.

Research by the International Multiple Sclerosis Genetics Consortium surveying the entire human genome identified 200 gene variants linked with MS predisposition. The vast majority of the allele variants it identified were in genes involved in the immune response, suggesting that the immune system is centrally involved in MS pathogenesis and that immune cells are the cells damaging the myelin sheath (International Multiple Sclerosis Genetics Consortium et. al, 2011).

The domain of MS development is the Central Nervous System (CNS). However, many types of immune cells, like microglia and macrophages, play a role in the pathogenesis of the disease. CD4+T-cells, CD8+ T-cells, B-cells, antibodies, and other cells in the adaptive immune response all have proven links to MS development (Rodríguez Murúa, S., Farez, M. F., & Quintana, F. J., 2022). But, cells in the innate immune response play an even greater role (Hemmer et al., 2015). Mast cells open the Blood-Brain Barrier, allowing other immune cells to enter the CNS, and release promoters for neurodegeneration. Astrocytes promote MS pathogenesis through interactions with cytokines and chemokines (immune signalers) that prompt the involvement of other immune cells. Natural killers, in contrast, actually seem to mediate MS and are found at lower levels in MS patients. Specifically how natural killer cells do this is unknown, but certain signalers they release have been found to induce T-cell apoptosis, which may contribute to their mediating effect. Macrophages and their CNS resident form, microglia, are the most important type of cells in the innate immune response related to MS. They play a varied role in MS pathogenesis, with different states of activation leading the cells to contribute to the development of the disease through signaling and phagocytosis or to clear debris in the CNS and

promote remyelination (Rodríguez Murúa, S. et al., 2022).

The potential of macrophages and microglia to be both agents of MS and promoters of remyelination is why they are a key focus of study in the context of the disease. GRIP1, also known as Glucocorticoid Receptor Interacting Protein 1, is a key factor in the dual functions of microglia in MS and MS models. The protein is a transcriptional coregulator in the immune system that mediates cytokine repression by glucocorticoids (Yurii Chinenov et al., 2012). It also plays a role in macrophage polarization and development - regulating the homeostasis of the immune system (Coppo et al., 2016).

Research suggests that GRIP1 has both positive and negative effects on MS pathogenesis. Mice in the EAE MS model with the GRIP1 gene knocked out experienced less severe symptoms of the disease, with decreased levels of demyelination, less infiltration of the nervous system by immune cells, and reduced microglial activation. However, GRIP1 suppression also reduced the therapeutic effects of treatments for the disease (Mimouna et al., 2020).

The papers in the preceding paragraphs are works of research of the Rogatsky Lab at the HSS Research Institute, where this investigation took place. The lab has advanced the understanding of MS and the role of genes like GRIP1 over many years of work, but questions remain. GRIP1 research is ongoing. To enable future research, inhibition methods for GRIP1 in research mice need to be verified.

The Rogatsky Lab uses a Cre promoter on the CX3CR1 gene to suppress GRIP1 expression in mice. This promoter takes the place of the normal CX3CR1 promoter and works like a set of genetic scissors, splicing sites, and genes marked by lox-p markers out of the genome (Kim et al., 2018). The CX3CR1 gene is involved in mice immunoregulation. Its function does not have relevance to this experiment, but the gene is activated regularly during normal homeostatic activity in the immune system - which is

important for the removal of GRIP1. This process can be taken a step further through Cre-ERT, where the Cre promoter can only function after the mouse receives a Tamoxifen injection. The Cre-ERT process allows gene suppression to be induced at specific times for an experiment (Donocoff, R.S. et al., 2020).

This experiment examined whether or not one copy of the Cre promoter (a Cre promoter on only one of the two CX3CR1 alleles) would be as effective as two copies of the Cre promoter (on both of the CX3CR1 alleles) at reducing GRIP1 expression. The lab prefers to run experiments with only one copy of the Cre promoter so that the effects of CX3CR1 suppression
do not skew results. It was hypothesized that one copy of the Cre promoter would be as effective as two copies of the Cre promoter at inhibiting GRIP1 expression.

Methods:

Mouse Prep

This experiment used a total of 18 mice (9 males and 9 females) born during October and November of 2022 at the HSS Research Institute. The mice were split into tw experimental groups and one control group based on their specific genotypes. One experimental group had mice with the Cre promoter on both CX3CR1 alleles, while the other had mice with the Cre promoter on only one CX3CR1 allele. Mice in both of these groups received Tamoxifen to trigger the Cre-ERT process. The mice in the control group had the Cre promoter on both alleles but were not given Tamoxifen. Three mice were selected for each group from each sex.

Members of the Rogatsky Lab began experimental work in late April of 2023 and conducted all research in the shared facilities of the HSS Research Institute. First, they gave injections of Tamoxifen in corn oil solution to the experimental group mice and injections of simple corn oil to the control group mice. Each injection was 150 uL and contained 3mg of Tamoxifen for the experimental group mice. These injections were given daily for the first four days of the experiment. The researchers euthanized the mice on the seventh day of the experiment, when Cre activity was high and GRIP1

expression was suppressed, before isolating the spleen and spinal cord cells. Tissues from these organs were digested and frozen at -80℃.

RNA Isolation

RNA Isolation was the foundation of this experiment. To measure GRIP1 expression, its RNA had to be isolated from each sample. RNA isolation was done with a QIAGEN RNeasy Mini kit and along a protocol outlined in their handbook (with slight modifications). First, the frozen tissue samples were thawed in a water bath before they underwent a PBS wash and centrifugation process to remove their storage media. Cells in the tissue samples were subsequently lysed with RLT buffer.

Afterward, each sample was homogenized with a 20-gauge needle. Ethanol was then added to each sample before they were spun in RNeasy spin columns. RW1 Buffer and RPE buffer were added before further spins. Finally, the RNA was eluted from the spin columns with RNAse-free water.

To accurately calculate solution ratios for the RT reaction to follow, each sample had its amount of RNA quantified using a Nanodrop Spectrophotometer. Any quantity of at least 60ng/uL was acceptable to use in the subsequent RT reaction. All samples from the spleen successfully passed the threshold, although all spinal cord samples had to be removed to do poor RNA yields.

Reverse Transcription

The RT reaction transcribed RNA back into cDNA. For each sample, ½ uG of RNA was needed. This was diluted to create a solution of 10uL with Nuclease-free water. 6uL of a mix of dNTPs and dN9 were then added to each sample. After the sample tubes were placed into a PCR machine correctly calibrated for the reaction, the RT reaction began. At a middle step in which the samples stayed at 4 ℃ , 4uL of a second mixture of RT Buffer, RNAsin, the reverse transcriptase M-MuLV, and Nuclease-free water was added to each sample. The samples remained stored at 4℃ - before qPCR analysis.

qPCR

expression for each control group sample is 1, as these are the values that experimental samples were quantified against. The standard deviation of this group is 0. For the male homozygous experimental group, M(+/+), the mean relative expression is slightly above 0.69. The female homozygous experimental group, F(+/+), has a mean relative expression slightly higher than 0.79. The standard deviation for the homozygous experimental samples is slightly below 0.27. The group's coefficient of variation is 35.8%. The male heterozygous experimental group, M(+/-), has a mean relative expression slightly above 0.57. The female heterozygous experimental group, F(+/-), has a mean relative expression slightly lower than 0.82. In the heterozygous experimental group, (+/-), the standard deviation is slightly below 0.26. The group's coefficient of variation is 37.2%. Figure 4 provides the mean relative expressions for homozygous experimental, heterozygous experimental, and control group samples as a whole, combined across both sexes. The mean relative expression for homozygous experimental samples is slightly greater than 0.74, and for heterozygous experimental samples the mean relative expression is slightly less than 0.70. Figure 5 and Figure 6 represent these expressions graphically, broken down by sample sex and treatment/genotype, and treatment/genotype respectively. Figure 8 shows the P-values of T-tests comparing the relative expressions of samples in each treatment group. The difference in expression between the control group and the homozygous experimental group (+/+) is not significant but trending towards significance ($0.05 < P < 0.07$). The difference in expression between the control group and the heterozygous experimental group (+/-) is significant ($P << 0.05$). The difference in expression between the two experimental groups is not significant ($P > 0.05$). Figure 9 shows the results of a Krusal-Wallis test run between the relative expressions of samples

Analysis and Discussion:

The results of this experiment are generally as expected. The mean relative

The process of qPCR quantified the amount of GRIP1 in the samples. First, the cDNA previously made during the RT reaction was diluted with Nuclease-free water. Then, a reaction master mix with SYBR Green and gene-dependent primer was created and aliquoted into the qPCR plate, before the cDNA of each sample was also aliquoted into theplate. The two genes quantified were the gene of interest, GRIP1, and a baseline gene, Actin. Each sample was run with two replicates for each of the two genes quantified. The qPCR reaction was executed in a QuantStudio 96-well machine. After data was collected from the machine, it was analyzed independently - as will be explained below.

Data Analysis

The research used the Livak method of relative quantification for data analysis. GRIP1 expression was compared to the expression of the baseline Actin. First, CQ values for each sample were averaged from the two replicates. Then, the ΔCQ values were calculated for each sample, using their CQ for Actin subtracted from their CQ from GRIP1. ΔCQ values were then averaged for the control group samples (GRIP +/+, without Tamoxifen). After, ΔΔCQ values were calculated, subtracting ΔCQ values of the control group from the ΔCQ values of the treatment group. Finally, expression ratios describing the relative expression of GRIP1 in each sample were calculated. The control samples had an expression ratio of 1, and every other sample was a decimal relative to that. Expression ratios were calculated by setting 2 to the power of the - ΔΔCQ for each sample. After this, the data were analyzed, graphed, and examined with standard deviation, T-tests, and Kruskal-Wallis tests.

Results:

Relative expressions of the GRIP1 gene, as quantified through the Livak method, are shown below in Figure 1. Means for each group are shown in Figure 3, and Figure 2 is a key to the labels used for the data. Figure 7 shows the standard deviations within each treatment group. As expected, the relative

of the homozygous and heterozygous experimental groups are very similar, 0.74 and 0.70, respectively. The control group has a mean relative expression of exactly 1, expected since relative expressions for experimental samples were calculated relative to these values. The relatively large coefficients of variation for both experimental groups raise some concerns about the reliability of the data. However, the T-test results and Kruskal-Wallis test results largely align with the experiment's hypothesis - suggesting that one copy of Cre is as effective as two copies of Cre at impairing GRIP1 activity. It should be noted that the difference between the homozygous experimental group and the control group is not significant, but trending very close to significance.

A principal error in the design of this experiment was the small sample size. This was due both to the limitations of experimental resources and the nature of this research. Mice with the correct genotype needed to be used for breeding and future research, and must be used efficiently according to Cornell's policy of 3Rs for animal use. Furthermore, this experiment was intended to confirm a method of GRIP1 suppression for future experiments, not establish novel protocols. 18 mice, in the context of this research, is a lot, but 18 samples are not. Some samples in each of the male experimental groups appear to be outliers. This may be attributed to how Tamoxifen, used in the Cre-ERT process, interacts with estrogen receptors. This interaction varies slightly between male and female mice but is not believed to have been a meaningful error in the experiment. The three samples in each group, when broken down by treatment and sample sex, are typically insufficient for meaningful research, but were deemed satisfactory due to the constraints and purpose of the experiment. This small sample size probably led to the large coefficients of variation for the experimental groups and the surprising lack of a significant difference between the homozygous experimental group and the control group. Once again, the lack of significant difference between the two experimental groups, the

Figure 8: P-Values of T-Tests Between Treatment Groups

(+/+) vs Control	**0.06450171628**
(+/+) vs (+/-)	**0.7650903249**
(+/-) vs Control	**0.0353284236**

Figure 4: Relative Expression by Treatment

(+/+)	0.7431728604
(+/-)	0.6965475695
Control	1

Figure 5: Graph of Relative Expression by Group

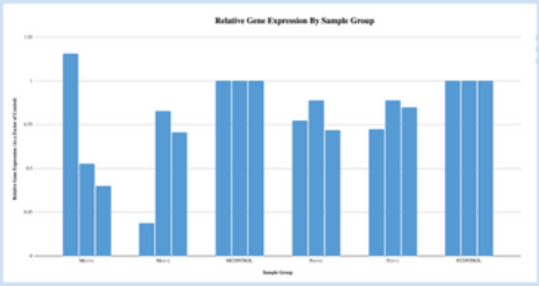


Figure 6: Graph of Relative Expression by Treatment

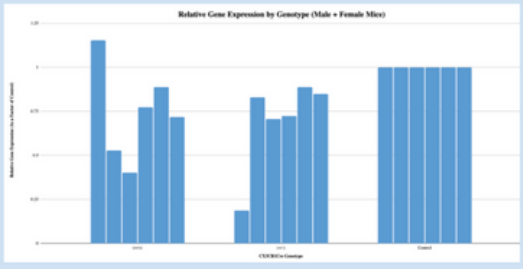


Figure 1: Relative Expression of GRIP1 By Sample and Group

	01	1.153432174
M(+/+)	02	0.5268347973
	03	0.4014102101
	04	0.1866747624
M(+/-)	05	0.8283195001
	06	0.7061260213
	07	1
MCONTROL	08	1
	09	1
	10	0.7717630493
F(+/+)	11	0.8879697987
	12	0.7176271327
	13	0.723346503
F(+/-)	14	0.886829706
	15	0.8479889243
	16	1
FCONTROL	17	1
	18	1

large significant difference between the heterozygous experimental group and the control group, and the trend towards significance of the aforementioned difference between the homozygous experimental group and the control group, all support the conclusions of this experiment.

This experiment validates the Cre-ERT GRIP1 knockout method as effective for future studies. The researchers hope that this work will enable future research regarding GRIP1's role in inflammatory diseases, and help further scientific endeavors in general.

Figure 2: Data / Group Key

- M(+/+): Male Cre (+/+) Experimental
- M(+/-): Male Cre (+/-) Experimental
- MControl: Male Cre (+/+) Control
- F(+/+): Female Cre (+/+) Experimental
- F(+/-): Female Cre (+/-) Experimental
- FControl: Female Cre (+/+) Control
- (+/+): Experimental homozygous
- (+/-): Experimental heterozygous
- Control: Control Group

Literature Cited:

Ascherio, A., & Munger, K. (2008). Epidemiology of multiple sclerosis: from risk factors to prevention. *Seminars in neurology*, *28*(1), 17–28. https://doi.org/10.1055/s-2007-1019126

Figure 7: Standard Deviation Within Treatment Groups

Standard Deviation							
(+/+)		M			F		
	0.2662152166	1.153432174	0.5268347973	0.4014102101	0.7717630493	0.8879697987	0.7176271327
	0.3582143951						
(+/-)		M			F		
	0.2597251725	0.1866747624	0.8283195001	0.7061260213	0.723346503	0.886829706	0.8479889243
	0.3728749964						
Control		M			F		
	0	1	1	1	1	1	1
	0						

SOURCES FOR ISSUE:

Let it Snow?
https://www.cnn.com/2023/11/25/weather/snowfall-temperatures-climate-change-water

Does Turkey Make You Sleepy?:
https://www.piedmont.org/living-real-change/can-turkey-make-you-sleepy

How Do Snowflakes Form?:
https://www.noaa.gov/stories/how-do-snowflakes-form-science-behind-snow

FSB FUN Facts:
https://www.chemistrylearner.com/chemical-reactions/maillard-reaction
https://candles.org/candle-science/
https://treevitalize.com/how-do-pine-trees-reproduce/
https://www.zmescience.com/feature-post/natural-sciences/physics-articles/matter-and-energy/what- gives-colour/ https://nsidc.org/learn/parts-cryosphere/snow/science-snow
https://www.livescience.com/winter-solstice
https://worlddeer.org/reindeer/
https://www.britannica.com/animal/reindeer

FIELDSTON SCIENCE BULLETIN