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A Dance of Life and Death: How cancer works and why it makes a great source of choreographic inspiration

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How cancer works and why it makes a great source of choreographic inspiration

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Abstract

Cancer is a problem that has plagued humans our entire existence, and with that, has come the quest for a cure. Over many years of research we have discovered that the cure wasn't easy to find and that cancer was a far more complicated disease than originally expected. Cancer is a disease characterized by uncontrolled cell growth, that begins with genetic mutations. With advances in science we came to understand our genetics and then we found some potential causes for cancer, in our genes. It is hoped that if we can reverse the faulty genetic mutations that cause cancer, we may be able to cure it. The mutations occur in two groups of genes, oncogenes and tumor suppressor genes, and the latter is the focus of this paper.

Tumor suppressor genes stop the cell cycle and can cause the cell to go into apoptosis, also known as programmed cell death. As the name suggests, these genes help prevent tumors and cancer from forming. When mutated they lose their function and thus their ability to prevent tumors, which can help give rise to cancers. Without active tumor suppressor genes, cells can grow out of control, which can lead to more mutations being gained and a tumor being formed. The genes I looked at are TP53, INK4, and PTEN-they have been chosen as they represent the range of functions tumor suppressor genes have and have been seen in cancers across the body. I looked at where the mutations are

occurring within the genes and examined potential differences between reproductive and nonreproductive organ-based cancers.

Through the data I collected and the results of my analysis, I planned to create a dance to be performed in the Spring Dance Concert in 2020. This dance will be inspired by American and German theatrical Modern dance and will be an abstract representation of how tumor suppressors work and how they act when mutated.

For my thesis, I blended art and science through the analysis of data and the creation of a dance. I explored how cancers and patients are affected by tumor suppressor genes in reproductive organ-based versus non-reproductive organ-based cancers. The reason behind this comparison lies in the difference in how the different organ system's cells grow in terms of rate. It is hoped that a difference will be found and that may help understanding how cancer works. And then that can be explored through a dance and shared.

Literature Review

Cancer research has come a long way since it began, and we now understand that there are genes in our bodies that when mutated can contribute to cancer growth. With that knowledge, we have gone out to find these genes and see how they work, as well as what causes them to be involved in cancer.

Although commonly grouped, tumor suppressor genes are not proto-oncogenes. Though genes in both of these broad groups can cause cancer when they mutate, they do so in different ways. Both sets of genes help control the cell cycle to regulate growth, however, tumor suppressor genes do this by stopping or pausing the cycle while proto-oncogenes do it by sending signals when they think the cell is ready to move onto the next stage. Proto-oncogenes are genes that check that everything the cell needs is there and if so tells the cell to continue to go through the cell cycle. Proto-oncogenes are called oncogenes when mutated and they tell the cell to just keep going through the cell cycle which causes quick tumor formation (Lodish, 1970). Proto-oncogenes are the normal non-mutated form of these genes while oncogenes are the mutated form. Because these two gene types function differently, their effect when mutated is also different.

Tumor suppressor genes work to stop the cells of the body from dividing and growing out of control. They rely on outside cell signals to tell them when

there is space and a need to grow. They are also believed to be a possible key in stopping and controlling cancer. If we could unmutate these genes we could potentially have the body fight the cancer itself and prevent the cancer from coming back. To unmutate would be to return the gene to its normal functioning form. This study examines the current theories on tumor suppressor genes and also briefly touches on what is being done in cancer research as a whole. Tumor suppressor genes have different subcategories: cyclin-dependent kinase inhibitors, genes that create proteins to stop the cell cycle to fix damaged DNA, and genes that create an enzyme to stop the cell cycle and perform apoptosis (programmed cell death) (Elviser, 2007). Tumor suppressor genes are genes that tell the cell cycle to stop if there is a missing factor or mutation in a gene. A tumor suppressor gene when mutated no longer tells the cell to stop when something is wrong, which can allow a proliferation of mutations.

In an article published by the American Cancer Society, recent fields of interest in cancer research were presented (*Expert Predictions: Cancer Care 10 Years From Now*, 2015). Eight of the ten doctors mentioned DNA being used to treat cancer in some way. This field of using DNA for cancer treatment is called immunotherapy and comes out of epigenetics. Immunotherapy is a form of therapeutic medicine that uses knowledge of the immune system to treat the body. Epigenetics studies, tags that exist on genes and is a subcategory of

genetics. Epigenetics studies when the code of the DNA is unchanged but the chemical makeup and the way the DNA is read is changed. The most common way this happens is through methylation is the addition of a methyl group which causes a section of DNA to be more tightly wound making it read less often. Looking towards the future, many of the researchers in this article mentioned individualized cancer treatment. And the field of epigenetics has been working towards a future of individualized medicine where people would be treated with drugs that would turn on or off certain epigenetic factors in order to treat them. This is also hoped to be the future of cancer medicine if the field can figure out which genes are involved and what happens when they mutate, we could potentially undo what has gone wrong and potentially cure the patient without the current harm that's done through chemotherapy, radiation and the many drugs that come along with them (*Expert Predictions: Cancer Care 10 Years From Now*, 2015).

The genes I am interested in are TP53, INK4, and PTEN, they represent the major functions of tumor suppressor genes. They are also present in both sex-organ based cancers and cancers throughout the body. They are also all being studied already as potentially important tumor suppressor genes.

TP53 creates protein p53 which attaches to the DNA and assesses when DNA is damaged, whether it can be fixed or if the cell needs to enter apoptosis.

Apoptosis is programmed cell death so the cell will kill itself if it recognizes that it is too damaged to be fixed. If the nucleus of the cell thinks the cell can be fixed than the p53 will activate the proteins necessary to fix the DNA ("TP53 Gene Tumor Protein p53.") In a paper written for the National Cancer Institute the major research that has been done on TP53 was summarized (TP53 gene, 2020). There are three main factors about TP53 that allow it to contribute to cancers. First, this gene can have a somatic mutation occur which has been shown to cause cancers. Second, TP53 in germline mutations has been found to be the cause of Li-Fraumeni syndrome which causes many early-onset cancers. Third, TP53 is highly polymorphic in its coding which means that there are many different forms of TP53 which increases its chance of causing cancer. A lot of the research that has been done has been using a human form of TP53 in mice using a knockout technique. A knockout is when the gene being looked at is removed to see what occurs when it is not there. With the amount of data we have gathered on TP53 we still need to do more research as we have found TP53 to have unique biomarkers as well as diverse forms which could make using it in cancer therapy difficult. What is meant by that is TP53 can shift and change and be hard to track due to its biomarkers so trying to find and change it for curing cancer could be difficult. A biomarker is a section of DNA that is suspected to cause a disease.

The purpose of the INK4 gene is to code for proteins that specifically bind to CDK4 and CDK6 and inhibit their activity (Sherr and Roberts, 1995). The presence of a CDK such as 4 or 6 causes a cascade effect that allows a cell to move from G1 phase to S phase which moves the cell closer to being able to replicate, by inhibiting this activity INK4 can stop replication and DNA can be checked for mistakes, which lowers the risk of the cell becoming a cancer cell. INK4 proteins are found from early embryonic stages all the way into adulthood. (Zindy et. al. 1997). INK4 when mutated can cause cancer both when the protein made by the gene is deleted or downregulated and also when it is being overexpressed (The Cancer Genome Atlas Program, 2020). Both aging and stress have been shown to cause an increase in the expression of INK4. INK4 is also known to be a part of cellular processes such as apoptosis, cell invasion, and angiogenesis which may be the clues as to why its overexpression can cause cancer (Cánepa, 2007). For those cancers being caused by overexpression of INK4, we may be able to use it for diagnosis and as a marker of the progression of the cancer. On the other hand, for cancers being caused by the under-expression or loss of INK4 we may be able to reinsert it and use it to stop the cancer from growing as has been shown in a murine model. Research being done on the INK4 gene currently involves looking at its effect in both its normal and mutated state. This is being done by knocking out the gene in mice, looking at gene expression in

a cell line, and performing genetic screens on human tumors (Roussel, 1999). Mice are being used both as excellent lab subjects as well as sharing 90% identity to the corresponding proteins that are made in humans by the INK4 gene (Roussel, 1999). It has been found that mutations in this gene are linked to a large percentage of sarcomas and lymphomas (Kamb et al., 1994; Ruas and Peters, 1998).

PTEN acts as a phosphatase, which means it breaks down/removes phosphate groups from proteins - this is a common way that cells can send signals to one another. Without the phosphate group the cell is inhibited from migration and adhesion to other cells which is an important part of the cell cycle. PTEN mutations have been found to cause disorders other than cancer, including Cowden, Proteus, Proteus-like, and Bannayan–Riley–Ruvalcaba syndromes, as well as autism spectrum disorder (PTEN Hamartoma Tumor Syndrome, 2018). They have also been linked to squamous cell carcinoma of the head and neck, endometrial and prostate cancer, as well as Glioma 2 (GLM2), and Macrocephaly/autism syndrome (MCEPHAS). The problem that arises when trying to use PTEN in cancer therapy is that it undergoes complex regulation which makes it hard to track. PTEN has been studied quite a bit as a potential biomarker to track the progression of cancer (Uniport PTEN, 2020). Although pten mutations have been found in sex organs there is high expression of PTEN in

adult tissues of the heart, brain, placenta, lung, liver, muscle, kidney and pancreas (Steak et al 1997).

Definition of terms - adapted from the National Human Genome Research Institute's Talking Glossary of Genetic Terms

The most common DNA mutations occur as substitutions, deletions, or insertions. A substitution is when one nucleotide is switched for another, so it doesn't change the number of codons but it does change which codons are there. Deletions are when a nucleotide is lost. Insertions are when one or more nucleotides are added to DNA. A nucleotide is a single letter of DNA, this includes ATGC. A codon is a sequence of three nucleotides that creates a specific amino acid when read.

Within those three mutations there are many consequences that can result. A frameshift occurs when an insertion or deletion has occurred and has changed the number of nucleotides, causing a shift in how the gene is read. Stop gained is when the codon reads as a stop before the original stop codon which results in a shorter protein. Missense is when a substitution occurs that causes the resulting amino acid to be different. Inframe deletion is when a deletion occurs that doesn't change how the rest of the gene is read and results in the loss of amino acids. And an inframe insertion is when a section is inserted that

doesn't change how the surrounding codons are read but results in more amino acids overall. 3 prime UTR is a section of non-coding mRNA that follows after the stop codon, what this means as a mutation is that since this chunk is important for binding it can change the structure and shape of proteins. The same goes for a 5 prime UTR though this section comes before the start codon, it regulates translation so when mutated that regulatory function may be lost. The intron is a portion of a gene that doesn't code for a protein, usually, these are cut out in the translation process but sometimes they are accidentally left in. Start lost is when the start codon of a gene isn't included when transcribed, which means that it's unreadable as it lacks its starting point. Protein altering is a mutation that alters what protein is made by a gene, this consequence also means that none of the other mutations that could change what protein is being made have occurred. Splice mutations occur in the messenger RNA before translation when the parts needed to make the final protein are not all there, or parts are added. Downstream genes are normally activated and allow for cellular processes like DNA repair, cell-cycle control and apoptosis to occur. A mutation in a proto-oncogene or tumor suppressor gene can cause it to no longer activate the downstream genes and is referred to as a downstream gene mutation.

There are also many different types of tumors. Neoplasms are tumors that result from cells dividing when they shouldn't be. Squamous cell neoplasms are

neoplasms specific to squamous cells which include the surface of the skin, the lining of hollow organs (bladder, kidney, and uterus), and respiratory and digestive tracts. A cystic neoplasm is when the tumor forms a cyst which is a sack with liquid on the inside. A serous neoplasm is when the tumor forms from the serous layer of epithelial tissue, which is what lines the outside of organs. A mucous neoplasm is when the tumor forms from the mucus lining. A ductural neoplasm is when the tumor forms at a duct. A lobular neoplasm is when the tumor forms at a lobe such as in the breast. A fibromatous neoplasm is when a tumor forms from connective tissue. A mesothelial neoplasm is when a tumor forms from the mesothelial cells which line a part of the lungs. An epithelial neoplasm is when a tumor forms from epithelial cells. A germ cell neoplasm is when a tumor forms from germ cells also known as eggs and sperm.

Adenomas/adenocarcinomas are cancers that begin in gland/secretory cells. Gliomas are cancers that begin in the glial cells, which are the cells that surround the nerves in the brain. Lymphomas are cancers that begin in the cells of the immune system. Leukemias are cancers that begin in blood-forming tissues, like bone marrow. Papillomas are cancers that begin growing on the tissue that lines the inside of organs and has growths that are long, and thin. Sarcomas are cancers that begin growing in bones or soft tissues.

Introduction

There are two major aspects to this paper, first being the science side which is a metadata analysis on data from the national cancer institute on three tumor suppressor genes. This data is input by scientists who work in hospitals across the country on the genetics of people with cancer. The data is gathered in the hopes of finding patterns and a better understanding of how cancer forms to figure out how to cure it. The data includes every mutation found on a number of genes that may possibly be linked to cancer, which includes oncogenes and tumor suppressor genes. The second aspect is the dance created from this data that was supposed to be showcased in the Spring Dance Concert.

The cancer research being done right now is largely focused on gene mutations and trying to identify them and hopefully fix them in the future with CRISPR technology (American Cancer Society, 2015). The logic behind that is that if we can remove the cancer cells and fix the mutation, that the cancer won't be able to come back as the mutation that caused it in the first place will be gone, also potentially removing the mutation during the treatment of cancer could stop cancer from growing further. This research fits within that, as a metadata analysis on gene mutations linked to cancer looking for patterns that may help identify what has gone wrong. Looking at the causes of mutations and how we may be able to fix those mutations. Another main aspect of the research is using tumor

suppressor gene protein products as biomarkers to detect cancer as well as track the progression of the cancer.

But before we can do those things we need to better understand the genes and their mutations which my thesis aims to potentially add more understanding on three of the major tumor suppressor genes. The majority of my knowledge of this was gained through the exploration and use of a database that was created by the National Cancer Institute (The Cancer Genome Atlas, 2020). It is important to understand the causes of cancer and what things we as people might be doing to increase mutation rates. Being able to understand the causes may lead to the cure for cancer and understanding causes of mutations may lead to avoidance of those things which could lower rates of cancer.

The dance is meant to show what is happening at a microscopic level when cancer occurs, this is something that is hard to see and understand, and having an artistic representation of it is meant to be helpful. The hope is that this dance encourages thoughtfulness about cancer and the research that is going on about it. It is also meant to help those who find learning about complex scientific ideas by using a more accessible medium.

Hypothesis

I predict that sex organed based cancers will have more severe genetic problems in comparison to cancers located in non-sex organs. With that, I expect

to see more mutations in number and severity (vital status and how hard it is for the cell to fix). Severity implying more loci affected by the mutations, as well as the number of places within a loci affected and mutation consequences that affect more codons. This prediction is based on the fact that the cells in sex based organ keep growing for the majority of one's life, and more growth leads to more potential mutations to occur and accumulate.

Why I chose the genes I chose

The genes I choose are representatives that are found both in reproductive and non-reproductive cancers. They are also widely studied genes, which was important for me as it allowed me to have enough data to pull from to have the possibility of having statistically significant findings. They also have all been considered as potential markers for a cancer's progression which means that we have been studying their importance in cancer as well as their level of mutation in relation to the stage of a cancer is at.

The plan was to collect data is to input information found from the National Cancer Institute's GDC Data Portal. I took information on the location of the cancer, the number of mutations, sex affected, vital status, type of DNA mutation, and whether its a sex organ or not. Sex organs are invovled in reproduction and include ovaries, testes, breasts, the prostate gland, the vaginal canal, the penis, and the uterus. The location of the cancer is important in understanding the pattern of each of the genes and where they mutate. The number of mutations is important in seeing the ease at which each gene mutates, along with comparing the number of mutations to the vital status to see if there are any correlations there. The sexes affected will be listed as male/female or both. This database does not take into account gender or other sexes besides male and female, so my report will not be able to consider transgender or

intersex people. The vital status is whether the person with the cancer lived through its removal or died from the cancer. The type of DNA mutation includes substitution, insertion, and deletion and from those, there are several mutation consequences. Understanding how the mutations can form is important to understanding how to stop them, which is why I am including this as part of my research. I included whether the cancer was in a sex organ or not, and this will allow me to scan my charts quickly to look at overall distribution of the data and whether the mutation is found in a sex organ or not.

Methods

The overall plan for the scientific part of my paper was to collect and enter my data which is outlined in the Data Collection section and then analyze that data as is outlined in the Data Analysis section.

Data Collection

I started by typing my gene of interest into the search bar of the National Cancer Insuites database, then I collected the data by choosing a project ID and then collecting data. On the main page of each project ID, I would record the vital status, sex, number of mutations, location, and cancer type- as well as recording whether that project included a sex organ-based cancer. I went into each project IDs mutation page and recorded the number of each type of mutation and of each mutation consequence. I went through and did this for every project for a gene and repeated it for each gene. These project IDs represent specific groupings of cancers and is how this database organizes its information.

Data Analysis

I used three programs to do my data analysis, Microsoft Excel, R which is an open-source statistics program, and Primer. Microsoft was mostly used as a place to store the data as I could turn it into the types of files the two other programs required. R allows for many statistical tests between two variables and is an easy program to make clear graphical representations. Primer allows for

more complex analysis with many variables through looking at things like resemblance between variables. Resemblance begins with you running a test that equalizes all of your data and then it looks at the percent difference between different categories. I used an ANOVA to check to make sure all of my data is significantly different. I used a t-test on vital status and the number of mutations to look for correlation. I also used a t-test on the number of mutations between those found in somatic versus germline mutations. I compared the number of mutations versus the number of cases with a t-test, which was meant to check for any sort of correlation between the number of mutations that occurred and the number of people affected by that cancer. This will be done as a two-sample unequal variance t-test. A binomial test was done for death count versus reproductive organ-based or not-with yes reproductive organ-based set as the success or 1 variable, and not reproductive organ-based set as the failure or 0 variable. I compared which was the most common mutation consequence across the three genes which was done using the resemblance test in Primer. I compared the number of reproductive cancers across the three genes; which will also be done as a resemblance test in Primer. I compared vital status to mutation number, to check for correlation between the rate of death and rate of mutations. This will be done as a two-sample unequal variance t-test. This was all

my original plan, most of which was not accomplished due to Covid-19 which is explained in the Covid-19 update section.

Inspirations for Developing the Dance

Modern is the basis of my technique and is a style I am inspired by. There are different eras of Modern where certain things came into popularity, for me the 1960s-70s is the era I am most inspired by. This era is identifiable by its use of new technology, the rebellion of older Modern styles, and chance dancing. My style fits within that, as I work in a very formal and minimal way as a lot of the choreographers of that time had.

A large part of the inspiration for the dance I will be creating is Alwin Nikolais' "Tensile Involvement". This dance is done by a large group of dancers who hold ribbons that are attached to rafters on a stage. The music is techno-based and there is use of multicolored lights to shift the colors of both the costumes and ribbons. This dance was created during the second wave of Modern dance which occurred in the 1960s/1970s. I am most drawn to this era of Modern as it is highly gestural and is largely what I have been taught. The use of ribbons in this dance is what inspired the idea of using fabric attached to the costumes to create the imagery of DNA strands. I plan to have the dancers each grab someone else's fabric, and twist it with theirs creating a double helix image. Also in the particular resetting of "The Green Table" that I have seen, the side lighting on the dancers in blue and red which is something I would like to incorporate into my own dance as DNA is typically colored as blue and red. Alwin

Nikolais' dances are inspired by science which is another thing that draws me to him as a creator. Another aspect of this era of dance that I am inspired by is the costumes, which were simple colored unitards that were popular at the time. I think unitards can do a great job of showcasing what movement is occurring while also obscuring and bringing the attention away from the fact that it's a human on stage which can transport the dance into being less of a formal performance and more of an experience. These qualities are important to me in creating my dance.

Another major influence on my dance is dance theater, this refers to a subcategory of Modern dance that includes more gesture movement and more use of theatrical elements. The two styles of dance theater I am most inspired by are German and Japanese Modern dance. Both focus on storytelling and use elaborate costumes and sometimes also sets and props. I plan to have a clear plot in my dance which is common to those styles, as well as using theatrical tools to add to my story rather than just adding in the visual effect as other Modern dance styles would do. Both German and Japanese dance theater styles use heavy makeup/masks which is something I am considering for my dance as I would like to obscure the human face to help the audience understand that the dancers aren't dancers portraying genes but are instead the genes themselves. I also am inspired by the way slow and subtle movements are used in Japanese

dance theater, and my opening section for my dance was inspired by that way of moving.

Thankfully, I have many other dances based on science to be inspired by, from the beginning of Modern dance scientific ideas have been incorporated into dances. And the Modern dances of the 60s/70s were heavily inspired by science, which may be why I am so drawn to that era of dance. Also, there is a foundation with a catalog of videos of turning the theses into dances ("Dance Your Ph.D"). I plan on watching many of those dances and reading about the processes people used to adapt a thesis into a dance. Dance and art in general have been used to explain complex ideas in ways that are easier to understand outside of the discipline for a very long time. Dance is a great way to represent DNA as it is already three dimensional and does move.

When I think of DNA, I think of something that is so immaculately and beautifully designed. The way in which DNA is able to be a source of so many different structures with vastly different functions is incredible. I picture it as the perfect machine, which is why I find I was so drawn to balletic movement to capture DNA. Ballet is seen as this very precise, rigid, and beautiful form of dance which is why it felt so perfect to be the starting inspiration for how my dancers would move. I personally have roots in hip hop and see that as a large inspiration for how I want the mutated dancers to move. The classic popping and locking

with the sharp staccato movements is something I plan to use when creating the mutated version of the movement. And overall I plan to use Modern movements to draw it all together.

Plans

My overall concept for my dance is to show how cancer works on a microscopic level, with each of my dancers representing a gene within a single cell. An important part of cancer is mutation and I plan to show that through having one of my dancers begin the dance doing a mutated version of the choreography and slowly have the other dancers join till they are all mutated, I want to do it this way as it is rare to have few mutations cause cancer. Most cancers are caused by a combination of many mutations. A mutated movement to me is largely twitch movement which is something I am naturally drawn to, it feels chaotic and spontaneous which to me fits what a mutated gene is like. I want the mutated movement to be passed on in the many ways DNA mutations can be passed, which includes randomly without any correlation between the two mutation events, a mutation causing another right next to it, and mutation skipping down the DNA to a section farther away. I plan on using the eight dancers to help create the imagery of the machine that is DNA. The idea of DNA as a machine will be shown through hard labor like movements that lean to the robotic side as far as their look. I also want an even number so that when the dancers pair up, they can pair up evenly. I also want the dance to start in a large clump and then separate into smaller groups to represent the cell dividing. I picture it beginning with the dancers being so close together that it is hard to

distinguish their bodies from one another. Slowly mutations will form and accumulate till all the dancers are doing mutated movement and in the end, they form a tumor and that kills the host that will be symbolized through self and other mutilation based choreography (I picture this as scratching and also pulling at the fabric) until they finally fall all the way apart and stop moving. My choreography is very focused on how the structuring of the dance changes the meaning of the movement, and so I plan on using graphs and tables I created to help shape the forms I use when creating the dance.

I plan to use colored lights and gobos (stencils put on lights, can be specific shapes like trees or can be abstract) to break up the human form and give the illusion of something that is not normally seen. Creating a knot in the fabric once the dancer is mutated, will be the way to signify a change to the audience that a mutation has occurred. For the costume, I want a unitard that has blue and red fabric twisted along the front left shoulder to the right hip and then for the two pieces of fabric to come off the unitard so that there are two stretch bands to be used in the choreography.

One of my first steps in creation is to find the music I want to use, and then to create a music map. A music map, sections the music into parts and has the number of counts in all of the parts. I then use this to decide the overall

structure of my dance like where the climax will go, and if there is any sort of intro and the length of the ending.

Dance Discussion

I always start my process by creating a music map that allows me to chunk my music into sections, making it way more manageable when working on the choreography. Then I figured out what I wanted my main motifs to be-these are the movements that are used repeatedly throughout the dance that add to the meaning. With my dancers in my mind, I made the first form-a windowed clump, meaning that they are close together but are all visible. This is where the dance begins, and it starts with some of the core movements that will be repeated throughout the dance. The dancers stay in this form for a while to set up for the audience what the DNA normally looks like in a cell. The dancers then form a circle, and then pull and stretch on each other's band to represent the replication of DNA. They then move to a straight line that represents the DNA being lined up in the center of the cell in mitosis. They pull away from each other forming two groups/cells as mitosis is completed. By this point in the dance, there are three dancers mutated and I have shown how mutations can pass themselves onto other genes as well as how they can occur spontaneously. I then separated the dancers into four groups of two representing another splitting of cells. In that section, in each of the pairs, one dancer is mutated and the other isn't, this is to show how mutated and non-mutated DNA interact. This is where I got to before no longer being able to hold rehearsals.

I found this piece of music while just listening to Spotify, and knew that it would be great to put a dance to. I then listened to many different renditions of the song and fell for the one that I ended up using. I enjoy music that has many rises and falls like a wave, and this music does that.

Covid-19 update

Covid-19 has prevented the completion of the dance aspect of my project. I am unable to work with my group of dancers due to social distancing. And I am also unable to access the dance studios, which means I do not have a safe place to work. As an adult having the proper flooring for dancing is important as it helps protect your body and allows you to dance for longer. Also having access to the mirrors in the dance space is important, as it allows one to quickly adjust and edit the choreography being created as you can see yourself. This dance was also built to be for a group, with having lifts and partnering which would have been difficult to try and transfer to a solo. So instead of attempting to fit it to a solo the dance has been left as is, and in the future I would like to revisit and finish it.

With the scientific aspect of my thesis, trying to use the data software is a bit of a challenge as I only have access to a laptop and the number of things needed to run it puts enough strain that it slows the computer. It also is now much harder to get help and be able to get quick responses to questions that come up while doing statistics. Though I was able to create my data sheets and fill them with all the information I thought might be necessary. I was also able to do the resemblance testing and set up for Primer. I also tried using some of the other testing functions of Primer with my data set. I also did some test t-tests in

excel and looked over the results of those tests to think about how I would want to continue to analyze the data.

What would have occurred

If Covid-19 had not occurred I would have finished my dance and it would have been performed at the Spring Dance Concert. A recording of the dance would have been made and would have been linked to my thesis. I would have written about how the dance had turned out and how I felt about its outcome. I would have written about what I might have changed if I had the opportunity to in retrospect. I would have also included the final lighting design and color references. A picture of the final costume would have been included. As well as the description of the dance included in the pamphlet from the concert. I would have also discussed the feedback from the audience as well as from other dancers.

I would have also been able to finish the scientific aspect of my paper which would have included completing my scientific analysis. Using the results to draw conclusions based on my hypothesis. I would have written a results section outlining the results and their scientific significance and then would have drawn conclusions from them. I would have created graphs and tables to help with the visualization of my project and those would have been included in my thesis. The graphs and tables made may have also been used to influence the dance either through floor formations or lighting.

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Appendix

TP53 Spreadsheet

https://docs.google.com/spreadsheets/d/e/2PACX-1vQQS2BULqc09Uoc3c2uMkDZzuN5LHubpUdbqJFr39OMpos8Qv8gVefNN_TIhfSt8AnHCYvVRQ9M4Vs0/pubhtml

PTEN Spreadsheet

https://docs.google.com/spreadsheets/d/e/2PACX-1vQ-vPhlifLYsYhu2BzJyb0Q2v3XizfTi5ppZNHFap0elilcOkSMFIemlxXbW3l79kw60lklxIBAYMLU/pubhtml

INK4 Spreadsheet

https://docs.google.com/spreadsheets/d/e/2PACX-1vQZYDli6NTG_Bej8k2p71gHSbV2bo1xpX0wQO9QbhWLORSxA3E-rvRWDoQKqaw-U5HJXI4Xn99dqGHY/pubhtml