A TEXTBOOK OF PLANT GENETICS

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CONTENTS

CHAPTER 1

PLANT GENETICS: INSIGHT INTO HISTROY, DEFINATION AND SCOPE

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ABSTRACT:

Plant genetic has made progress in creating plants that can survive in different types of difficult conditions. Molecular control mechanisms for dealing with non-living stress involve turning on and controlling certain genes that help us handle the stress. Scientists have used many different genes to make plants that can survive difficult environmental conditions. These genes help the plants produce proteins that help protect them from things like drought or extreme temperatures. More and more people are starting to value the use of regulatory genes to create plants that can withstand stress. This method seems to be better than other methods. This means that when one gene controls the behavior of many other genes, it can cause a lot of changes in how a living thing responds to its environment. We are still working on identifying, separating, and copying genes that are involved in responding to non-living stressful conditions. However, scientists have found many genes that respond to stress and could be used to make plants that can handle tough conditions. The development of plant transformation has made it possible to improve plants' ability to withstand harsh conditions. This can greatly help to increase crop production.

KEYWORDS:

Genes, Genetics, Mendel's, Plants, Traits.

INTRODUCTION

Plant genetics is the scientific study of genes, genetic diversity, and heredity in plants. Most people think of it as a part of biology and botany, but it is also related to other life sciences and the study of information systems. Plant genetics is similar to animal genetics in many ways, but there are some important differences. Gregor Mendel, a scientist from the late 1800s who was also a priest, is known for finding out about genetics. Mendel studied how parents pass down traits to their children. He found out that living things pass on traits in separate "units of inheritance. " These units are called genes and we still use this word today, although it is not a very precise definition. Mendel's plant research is still very important in modern plant genetics. Plants, just like other living things, use DNA to pass on their traits. Plant genetics is the study of how plants inherit traits from their parents and ancestors. This can be challenging because plants can often fertilize themselves, while most animals cannot. Many plants have certain genetic traits that make it easier for them to change and form new species. Plants are unique because they can make energy-rich carbohydrates through a process called photosynthesis, using special parts inside them called chloroplasts. Chloroplasts, just like mitochondria, have their own DNA that looks very similar. Chloroplasts help store more genes and genetic diversity. They also add an extra level of genetic complexity that is not found in mammals [1], [2].

Plant genetics has a big impact on money: many important crops have been changed at the genetic level to produce more, fight off pests and diseases, survive weed killers, or become

more nutritious. Plants, like other living things, use DNA to pass on their characteristics. Plants are the only living things that have chloroplasts. Chloroplasts and mitochondria can have genetic changes. These changes can pass on to future generations more easily in plants, as the flowers are formed at the ends of the branches where these changes occur. This has been known for a long time, and mutant branches are called sports. If the fruit on the sport is economically valuable, a new type of plant can be obtained. In certain plants, they can fertilize themselves. Some plants mainly fertilize themselves. This means that a plant can be both a mother and father to its offspring, which is something that animals don't usually do. When scientists and plant enthusiasts want to combine two plants, they have to be careful to make sure the plants don't fertilize themselves. People create hybrids between different types of plants for economic reasons and because they think they look nice. This is done in the field of plant breeding. The amount of maize produced has increased about five times in the past century, thanks to the creation and spread of hybrid corn varieties. Plant genetics can help predict which combination of plants will create a strong and healthy hybrid plant. Similarly, studying the effects of mixing different plants together has led to many important findings in plant genetics [3], [4].

The plant used for studying genetics in plants is called Arabidopsis thaliana, also known as thale cress. A thaliana has helped us learn about plant genetics, just like how Drosophila, a kind of fruit fly, has also contributed to our understanding of genetics. The organism's genetic information is small, which makes it easier to analyze at an early stage. This thing has a 125 Mbp genetic code that has around 25,000 genes. A database called The Arabidopsis Information Resource (TAIR) was made to store information and data about a plant. This includes the complete genetic information, details about genes and their products, how genes are expressed, stocks of DNA and seeds, maps of the plant's genetic makeup, markers for genetic and physical traits, published studies, and general information about the plant. All research related to thaliana is found in TAIR. There are many naturally inbred types of A. There are different types of thaliana plants called accessions or ecotypes. These plants have been helpful in studying genetics. Scientists have studied this random change to discover genes that help plants and animals resist both living and non-living challenges. Genetically Modified (GM) foods are made from organisms that have had their DNA changed using genetic engineering methods. Genetic engineering is a different way of changing the characteristics of living things. It is different from traditional methods like selective breeding and mutant breeding. Genetic engineering lets us add new traits and have more control over traits [5], [6].

One of the most important parts of biology is cytogenetics, which is the study of cells and their inherited traits. Cytogenetics is a combination of different sciences throughout history. It includes the study of cells and heredity. It is often noticed that mango seeds grow into mango plants, and dogs have puppies, not other animals. People have babies who grow up to be humans. When children have traits that are similar to their parents, we call it heredity. The study of how traits are passed down from parents to children is called genetics. Genetics tries to explain why siblings can look different from each other and why some people have dark skin while others have fair skin. In simpler terms, why do individuals of the same kind have differences. This lesson discusses heredity and why individuals of the same species vary. This text explains how scientists use different methods to figure out how certain things work in humans, like how our sex is determined, how our blood groups get passed down from our parents, and how genetic disorders are inherited. It also talks about a technique called amniocentesis that helps scientists learn about a person's genetic makeup before they are born. Louis Pasteur showed that living organisms cannot just suddenly appear out of nowhere. This idea says that living things come from other living things. It made people think

about parents and children and probably helped start studying how traits are passed on from one generation to the next. Children look a lot like their parents, but they also have some unique characteristics. Offspring looking like their parents is because they inherit traits from them, while the differences come from variations. Genetics is the study of how living things inherit and have different traits, and what causes these traits to change. The first person to try mixing different plants together was a German plant scientist named Kolreuter. He looked at how certain traits get passed down when two tobacco plants are combined. He noticed that the babies looked like both their parents. One of the most significant figures in Genetics is Gregor Mendel, who was an Austrian monk. He is considered the father of Genetics. He was the first person to study inheritance closely and use math in biology. New research suggests that your parent's lifestyle and experiences can affect your DNA. This is called Epigenetics. These tags impact how the genes in your DNA work. For instance, think about twins. Identical twins are basically copies of each other. They have the same DNA since birth. When twins are little, they appear identical. As twins grow up, they experience different things in their surroundings. What people eat and what they do can be different, which can cause them to have different body shapes like being overweight and different health problems such as heart disease and cancer. During important times like puberty and pregnancy, you are more likely to be affected by epigenetic changes. Epigenetics is the study of how genes can be turned on or off without changing the actual DNA. This can lead to changes in appearance or behavior without changing the genetic code itself [7], [8].

DISCUSSION

When a baby is born into a family, the family members start to think about how the baby's eyes, facial features, skin, and hair color resemble those of the parents, siblings, and grandparents. The reason why people look similar or different to their family members is because of the genes they inherit from their parents and pass down to their children and future generations. This passing down of genes is called 'heredity' and the study of reasons for heredity is called 'Genetics'. New people acquire characteristics based on the genes they get from their mom and dad. Heredity means passing on traits from parents to their children. It is noticed that brothers and sisters who have the same parents are special and are not the same as each other, except for identical twins. These differences are called variations.

Variation means the differences that can occur between parents and their children, between siblings, or between individuals within a group. Differences among individuals in a group of organisms are very significant. It helps the population to survive. This happens when the environment changes, and some individuals are able to adjust to new situations and prevent the whole population from dying. Variation occurs when there is a change in the genes, often caused by a mutation or sudden alteration. Variation happens when genes are mixed and exchanged during the formation of gametes, creating new gene combinations. During fertilization, the paternal and maternal chromosomes mix randomly, resulting in different gene combinations [9], [10].

A common source of variation is when genes mix together, which is called genetic recombination. Heritable differences usually occur due to changes in genes or when genes get mixed together during reproduction. Important words in genetics. In genetics, a factor is a unit that controls the inheritance and expression of a specific characteristic. These factors, also known as genes, are present in pairs in cells from parents and individually in reproductive cells. A part of the DNA that decides how a certain characteristic is inherited and expressed. Alleles or Allelomorphs are different versions of a gene.

For instance, in a pea plant, there are two different versions of the gene for seed shape: one that makes the seeds smooth and another that makes them wrinkled. The genes for smooth and wrinkled seeds are different versions of each other, and they are found in the same spot on similar pairs of chromosomes. A trait is a visible characteristic that can be seen on the outside of an organism's body, like its shape or physical features. The color of the flower and the shape of the seed. When there are two versions of a trait, the one that is seen in an organism that has different versions of the trait is called the dominant trait. The other version, that is not seen in the organism but can show up in their offspring, is called the recessive trait. So, if an organism has the combination of alleles Tt, and the allele T (which represents tallness) shows itself but the allele t (which represents dwarfness) does not, then T is the stronger allele and tallness is considered dominant over dwarfness, which is represented by t.

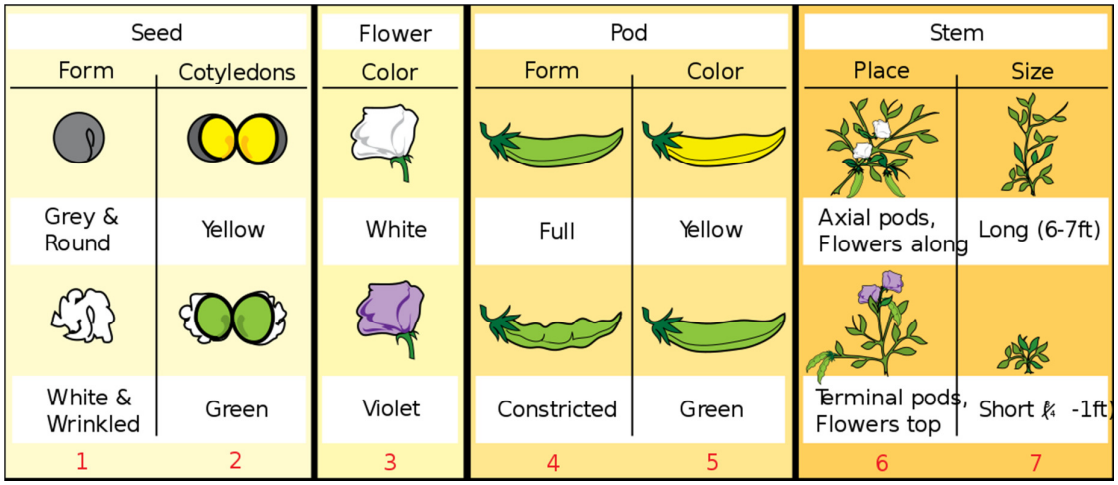

Figure 1: Representing the different trait used in the mendelian genetic [Byju's].

A recessive trait is a trait that is hidden when there are two different forms of the trait. It is called the recessive trait or allele because it is not expressed in the first generation (F1) when the two forms are mixed together (Figure 1). But the recessive gene only shows its effects when there are two copies of it (homozygous state). Genotype refers to a group of individuals that share the same genes and how they reproduce. For example, a pure smooth-seeded parent pea plant has a genotype of SS and will always produce smooth seeds. But if plants with the genotype Ss reproduce with themselves, they will create a population with a ratio of 3 smooth-seeded plants to 1 wrinkled-seeded plant. Phenotype is a group of individuals identified by how they look on the outside for a specific trait. Seeds can have a smooth or wrinkled shape, which shows two different appearances. Homozygous means that an individual has the same alleles for a certain trait. SS means that an organism has two of the same alleles for the smooth seeded character in garden-pea. Heterozygous means having different forms of a trait, such as having one allele from each parent. Ss means that the garden pea has both smooth and rough seeds, with smooth being the dominant trait.

This means that the parents chosen for the initial mating are considered the first generation of parents. The new generation of offspring that comes from two parentss is called the First filial or F1 generation. The F2 generation is when the children of the F1 generation are bred with each other or with themselves. A monohybrid cross is when two parents have only one pair of different traits. The F1 offspring from this cross is called a monohybrid. The ratio of how traits appear in the second generation from Mendel's experiments is called the monohybrid ratio. It is when three dominant traits appear for every one recessive trait. A dihybrid cross is when two parents with different traits in two pairs of characteristics are studied together to

understand the inheritance pattern. The ratio of different physical traits observed in the second generation from a cross between two individuals with two different traits is called Mendelian dihybrid ratio (9 : 3 : 3 : 1), and the first generation individual is called a dihybrid.

Creating new and improved traits in offspring by mating different species together. A test cross is when the offspring from the first generation are crossed with a parent that has two copies of the recessive trait. If the F1 offspring has different genetic characteristics, then a test cross will always result in equal numbers of each characteristic. A reciprocal cross is when the parents switch genders. If the first cross had a short father and a tall mother, then in the second cross, the short parent will be female and the tall parent will be male. Plants can reproduce in different ways. One way is through vegetative reproduction, meaning they can grow new plants from their existing parts. Methods can be categorized into two types: asexual methods and sexual methods. The way plants reproduce without seeds is different for each type of plant, like bacteria, algae, fungi, mosses, ferns, and flowering plants. Asexual reproduction means that different types of spores are produced in algae, fungi, bryophytes, and Pteridophytes.

Sexual reproduction is a method of reproduction in plants where they create special sexual organs like archegonia, antheridia, cones, Strobilus, and highly modified flowers in angiosperms. Sexual reproduction occurs when an organism creates and releases special cells called gametes. These gametes are made through a process called meiosis. During a process called meiosis, something called crossing over happens. This crossing over creates variety in the sex cells and the organism as a whole. Flowers in angiosperms come in many different shapes and sizes, and can have different colors and smells. Some flowers can be either male or female, while others have both male and female parts. These characteristics greatly impact the ability to have offspring. Flowers can change their structure to make cross pollination and self pollination happen.

Passing on acquired traits from one generation to the next. He believes that if a person is healthy, then their children will also be healthy. The children of a weak person will also be weak. According to Charles Darwin, the traits seen in one generation are often seen in the next generation too. ii) Epigenesis and Pangenesis: The idea of pangenesis was introduced by Charles Darwin in his theory of Pangenesis. He wanted to explain how traits are passed down from parents to children and how these traits affect the growth and development of the children. He came up with the term gemmules, which means the imaginary particles that are released by all cells of the body and carry inheritance.

The theory says that an organism's environment can change the tiny particles in its body, and that these changed particles would gather in the reproductive organs of the parent to be passed down to their children. If a part of a living thing is changed or becomes different, the small parts that make up that living thing will also change or become different for that part. The changed gemmule will now pass on to the next generation through the gametes and cause a similar change in the corresponding organ of the offspring. This is called pangenesis.

When biologists stopped believing in the theory that traits can be passed down from parents based on the experiences or acquired characteristics of their ancestors, which was a partial basis for the pangenesis theory. In the 20th century, biologists changed their ideas about how traits are passed down from parents to offspring. They stopped believing in pangenesis and started believing in germ plasm theory, and then later in chromosomal theories of inheritance. They also stopped using the term "gemmules" and started using the term "genes" instead.

Epigenetics is a scientific concept that was introduced by Waddington in 1942. This word comes from the Greek word Epigenesis, which was first used to talk about how genetics

affect development. This is a modern science that is part of genetics. It studies the changes in genes that can be passed down from parents to children and affect how they appear or behave. These changes don't affect the actual DNA sequence, instead they modify the histones and DNA methylation, along with other similar processes.

August Weismann, a person who studied genetics a long time ago, came up with this idea. And questioned the widely accepted idea that traits acquired during an individual's lifetime can be passed on to their offspring, as well as the belief in Pangenesis. He is trying to make progress in understanding heredity. This theory says that everyone's body is made up of two different types of tissues: somatoplasm and germplasm. The somatoplasm is made up of cells in the body that are important for the organism to stay alive and work properly. These do not help with making babies. So, the changes that happen inside a living organism cannot be passed on to the next generation. On the other hand, germplasm creates cells called gametes that are important for sexual reproduction. The changes that are present in the genes are passed on to the next generation. In animals, gonads are the reproductive organs and they are different from somaclones. Some plants can have babies without needing another plant. However, most plants that have babies this way do not look exactly like their parents. This theory is a big step forward in understanding genetics.

Transformation allows for the expansion of the gene pool in plants, making it possible to manipulate the genetic material of all living beings and their DNA sequences. Plant transformation is a useful tool for studying genes, making lots of important proteins and substances in plants, and improving crops. It has been widely used since it was first shown in 1983. In theory, every plant has the ability to be changed. Basically, new ways to change plants have been created for almost all kinds of crops and some smaller ones too. In the past, scientists successfully changed the DNA of plants like tobacco and petunia because these plants were good for studying genetic modification. They could easily grow in a lab and had a high chance of growing new plants from their cells. They were also sensitive to certain chemicals and could be easily transformed using Agrobacterium to deliver new DNA. Since the first demonstration of plant transformation in 1983, the methods used to change and improve plants were quickly applied to many different crop plants. The Flavr Savr tomato was the first genetically engineered food to be sold. It was changed to last longer on store shelves and was made available to the public in 1994. The Flavr Savr product was not very popular or successful in the market. However, it was significant in history because it was the first genetically modified product to be developed and approved by regulations. This paved the way for future commercial launch of genetically modified crops. The Roundup Ready soybean was made available for farming in 1996. This, along with other genetically modified crops, led to a large increase in their production. Going from a small experiment to making a product for sale in many different types of plants within just 13 years is a really impressive accomplishment in technology and business. Crops with transgenic traits that help control weeds and pests are being adopted by farmers very quickly. This adoption rate is faster than any other crop improvement in the history of farming.

CONCLUSION

In recent years, genetics has become very important in biology. It is used to study how traits are passed down and how living things are different from each other. It has also given scientists new ways to study different processes in plants and other living things. So, now every biologist needs to know a little bit about genetics. Genetics is the foundation of modern biology. The study of genetics had a big effect in medicine. Farming, growing trees, fishing, rules, and beliefs. Recently, Biotechnology has made the science of genetics more important by introducing new technologies like genetic engineering, recombinant DNA, and tissue

culture. Researching how plants grow and change when they are exposed to different kinds of radioactive materials helps us understand how they are affected by various types of radiation. Based on what we currently know, it seems that many plants can be harmed by being exposed to certain things. This is shown by the various instances of genetic damage and unusual growth problems that aren't commonly seen in normal circumstances. Smaller amounts help plants grow either by changing their genes directly or by controlling how their cells work. On the other hand, larger amounts can harm genes, create harmful molecules, and impact the ability of plants to grow and develop. This can prevent seeds from sprouting and make the plants have unusual growth patterns. It is hard to say the best amount of radiation to use.

REFERENCES:

[1] P. Ronald, "Plant genetics, sustainable agriculture and global food security," *Genetics*. 2011. doi: 10.1534/genetics.111.128553.

[2] S. Pflieger, V. Lefebvre, and M. Causse, "The candidate gene approach in plant genetics: A review," *Molecular Breeding*. 2001. doi: 10.1023/A:1011605013259.

[3] Y. S. Chung, S. C. Choi, T. H. Jun, and C. Kim, "Genotyping-by-sequencing: a promising tool for plant genetics research and breeding," *Horticulture Environment and Biotechnology*. 2017. doi: 10.1007/s13580-017-0297-8.

[4] H. T. Nguyen, "Plant genetics," in *Experiments in Crop Science*, 2015. doi: 10.2135/1990.experimentsincropsci.c2.

[5] P. McCourt and D. Desveaux, "Plant chemical genetics," *New Phytologist*. 2010. doi: 10.1111/j.1469-8137.2009.03045.x.

[6] M. Rossetto and R. J. Henry, "Escape from the laboratory: New horizons for plant genetics," *Trends in Plant Science*. 2014. doi: 10.1016/j.tplants.2014.06.011.

[7] T. R. Sinclair and T. W. Rufty, "Nitrogen and water resources commonly limit crop yield increases, not necessarily plant genetics," *Global Food Security*. 2012. doi: 10.1016/j.gfs.2012.07.001.

[8] A. T. Kramer and K. Havens, "Plant conservation genetics in a changing world," *Trends in Plant Science*. 2009. doi: 10.1016/j.tplants.2009.08.005.

[9] P. Sikora, A. Chawade, M. Larsson, J. Olsson, and O. Olsson, "Mutagenesis as a tool in plant genetics, functional genomics, and breeding," *International Journal of Plant Genomics*. 2011. doi: 10.1155/2011/314829.

[10] R. Tóth and R. A. L. van der Hoorn, "Emerging principles in plant chemical genetics," *Trends in Plant Science*. 2010. doi: 10.1016/j.tplants.2009.11.005.

CHAPTER 2

CELL DIVISIONS: PROCESS OF CELL DUPLICATION AND MULTIPLICATION

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ABSTRACT:

Cell division is when a cell splits into two or more new cells. Cell division happens as part of a bigger process called the Cell Cycle, which we will study in more detail in this chapter. In living things with a cell nucleus, there are two different ways cells divide. One way, called mitosis, makes two new cells that are exactly the same as the parent cell. The other way, called meiosis, makes cells with only half the number of chromosomes as the parent cell, and these cells are used for sexual reproduction. We have already studied the process of Meiosis, which is closely related to Mendel's Laws. In this lesson, we will examine Mitosis more closely. Cell growth and division is important for making new cells and for the growth of living things with many cells. In a cell process called mitosis, genetic information is transmitted. This process makes sure that when a cell divides, each new cell gets the same genetic material as the original cell. This means that each new cell has one copy of each chromosome that was in the original cell.

KEYWORDS:

Cells, Chromosomes, DNA, Mitosis, Meiosis.

INTRODUCTION

According to the cell theory, cells come from other cells that already exist. Cell division is the name given to the process in which this happens. Any living thing that reproduces by having sex begins its life as a tiny cell called a zygote. The process of cell division doesn't end when an organism grows up, but it keeps happening throughout its entire life. The cell cycle is the process a cell goes through between one division and the next. The cell cycle is divided into two parts Interphase, which is a period of getting ready for cell division, and Mitosis (M phase), which is the actual period when the cell divides. Interphase is broken down into three parts: G1, S, and G2. G1 phase is the time when the cell gets bigger and does its regular functions. During this phase, most of the organelles also duplicate. S phase is the stage of DNA replication and chromosome duplication. The G2 phase is when the cytoplasm grows. Mitosis is a process that has four stages called prophase, metaphase, anaphase, and telophase. Chromosome condensation happens during the beginning phase of cell division. At the same time, the centrioles go to the other ends. The protective layer around the nucleus and the part that helps make ribosomes go away, and long, thin fibers called spindle fibers start to show up. During metaphase, the chromosomes line up in the middle of the cell. In anaphase, the centers of the chromosomes split and the two parts start moving toward opposite ends [1], [2].

After the chromatids reach the two ends, the chromosomes start to get longer, and the nucleolus and the nuclear membrane appear again. This part of the process is called the telophase. After the nuclear division, the cell's cytoplasm is divided, and this process is called cytokinesis. Mitosis is a type of cell division where the number of chromosomes stays the

same in the new cells. Unlike mitosis, meiosis happens in cells that have two sets of chromosomes and will eventually develop into reproductive cells called gametes. It's called reduction division because it cuts the number of chromosomes in half when making gametes. During sexual reproduction, when the two sex cells combine, the number of chromosomes is brought back to the same amount as in the parent. Meiosis is split into two parts meiosis I and meiosis II. During the first step of cell division, the matching chromosomes join together to make pairs, and they exchange pieces of genetic material. The first stage of meiosis has a lengthy prophase that can be divided into five smaller phases. These are different stages in the cell process. The stages are called leptotene, zygotene, pachytene, diplotene, and diakinesis. In metaphase I, the bivalents line up in the middle part of the cell. This is followed by a step called anaphase I. During anaphase I, the pairs of chromosomes separate and move to opposite ends of the cell, with both of their parts called chromatids. Each pole gets half of the number of chromosomes from the parent cell. During telophase I, the protective layer around the nucleus and the structure responsible for making ribosomes show up again. Meiosis II is like mitosis. During anaphase II, the two copies of DNA called sister chromatids split apart from each other. So, after meiosis, four cells with half the usual number of chromosomes are created [3], [4].

The cell cycle is a step-by-step process in which a cell grows and divides to create two new cells. Cells going through cell division go through a series of growth stages, DNA replication, and division to make two identical cells. There are two main phases in the cell cycle: interphase and mitotic. During interphase, the cell gets bigger and the DNA is reused. During the mitotic phase, the DNA and substances inside the cell are copied and separated into two parts. Then, the cell splits into two separate cells. Mitosis makes two cells that have the same genes and are complete. Multicellular organisms grow through a process called mitosis. Cell growth changes the ratio between the nucleus and cytoplasm. Cells divide in order to maintain a balance between the nucleus and the rest of the cell. Mitosis helps the body fix damaged cells. The top layer cells on our skin, the cells lining our gut, and the cells in our blood are constantly being replaced. Mitotic differences in the growing tissues of plants (the tips and sides) lead to ongoing growth throughout their lives. In sexually reproductive organisms, meiosis helps to keep the same number of chromosomes each species has from one generation to the next. It also makes the genes of an organism population more diverse in each new generation. Differences are really important for how species change over time [5], [6].

DISCUSSION

The cell cycle is the process where cells get bigger, then split into two new cells. This can happen in two different ways either making identical cells (mitosis) or making special cells (meiosis). The cycle is split into four main stages: Gap 1, Synthesis, Gap 2, and either Mitosis or Meiosis. G1, S, and G2 are all part of the same phase called Interphase (Figure 1). The first part of interphase is a slow period called Gap 1 (G1). This is the beginning stage of interphase. This is the place where the cell does its usual tasks and gets bigger, especially after mitosis when the offspring cells are smaller than the original cell. This stage ends when the DNA synthesis phase starts, and each chromosome is copied. Even though the chromosomes are not tightly packed, they are copied as two identical halves joined together at the center during the S phase of interphase. After the replication in interphase, there is another waiting period called Gap 2 (G2). In stage 2, the cell keeps getting bigger and collects the proteins it needs to divide. There are different points, as seen in Figure 1, that are controlled by cyclins. Cyclins are proteins that help control how cells grow and divide. They work by activating specific enzymes that are needed for the cell cycle to happen. If there are

any issues with copying or getting the necessary proteins, the cell process will stop until it can repair the problem or die. The last step is called mitosis, which is when the cell splits into two. There are many different versions of this cell cycle. Cells going through meiosis typically skip the G2 phase. Cells, like hematopoietic stem cells, that are in the bone marrow and make all the other blood cells, will always go through these stages as they are always copying themselves. Other cells, like those found in the nervous system, will not divide anymore. These cells stay in a stage called G1 and never move on to divide. They enter a stage called G0 where they stop dividing permanently. However, certain cells, such as the larval tissues in Drosophila, go through multiple rounds of DNA synthesis without splitting into new cells, which is called endoreduplication.

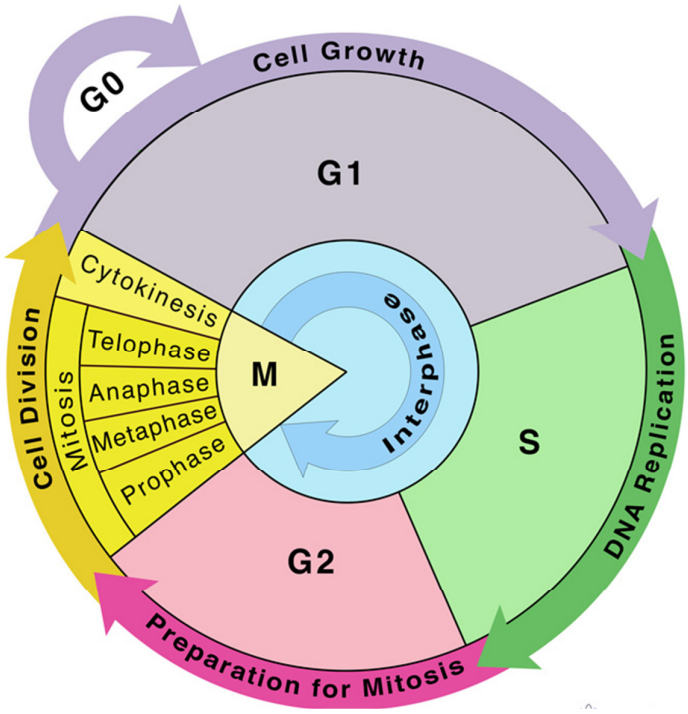

Figure 1: Representing the overview about cell cycle [Science Fact].

During the S-phase of interphase, the chromosomes make copies of themselves. This means that each chromosome now has two identical parts called sister chromatids that are joined together at a special area called the centromere. After the DNA synthesis phase and the gap phase 2, the cell goes into mitosis. The first step in mitosis is prophase. During this step, the center of the cell (the nucleus) breaks apart, and the copied chromosomes become tightly packed and visible. Next is metaphase. During metaphase, the microtubules connect to the kinetochore. The chromosomes line up in the middle of the cell. This middle part is called the metaphase plate. The kinetochore is the part of the chromosome where the microtubules attach. It has the centromere and proteins that assist the microtubules attach.

Afterward, in anaphase, the chromosomes' sister chromatids move toward opposite sides of the cell that is dividing. Finally in telophase, the identical sets of unreplicated chromosomes (single chromatids) are fully split into the two daughter cells, and a nucleus forms around each set of chromosomes. After the cell has finished dividing its nucleus (mitosis), the cytoplasm also divides (cytokinesis) to create two new cells that are the same as the original cell. The main stages of mitosis are remembered using the acronym iPMAT. Mitosis is a process where chromosomes are divided during cell division to create two new cells that are

exactly the same. Mitosis helps keep a certain group of living things stable by making copies of their chromosomes, which have their DNA [7], [8].

The amount of DNA inside a cell changes during fertilization, DNA synthesis, and mitosis. We use the letter c to show how much DNA is in a cell, and the letter n to show how many sets of chromosomes there are. single set of chromosomes), there is only one copy of each chromosome. This is in contrast to diploid cells, which have two copies of each chromosome. When a reproductive cell (sperm or egg) is formed, it contains half the amount of DNA compared to other cells (Figure 2). Additionally, the number of chromosomes in these cells is also half compared to other cells, which is represented by 1n. After the egg and sperm join together, the amount of DNA and the number of chromosomes in the resulting cell both doubles. After DNA replication, the amount of DNA doubles to 4c. However, the pair of sister chromatids are still stuck together at the centromere, so they are considered as one single chromosome, even though they have been copied. Therefore, the number of chromosomes stays the same at 2n. If the cell goes through mitosis, each daughter cell will become 2c and 2n again. This happens because it gets half of the DNA and one of each sister chromatid pair.

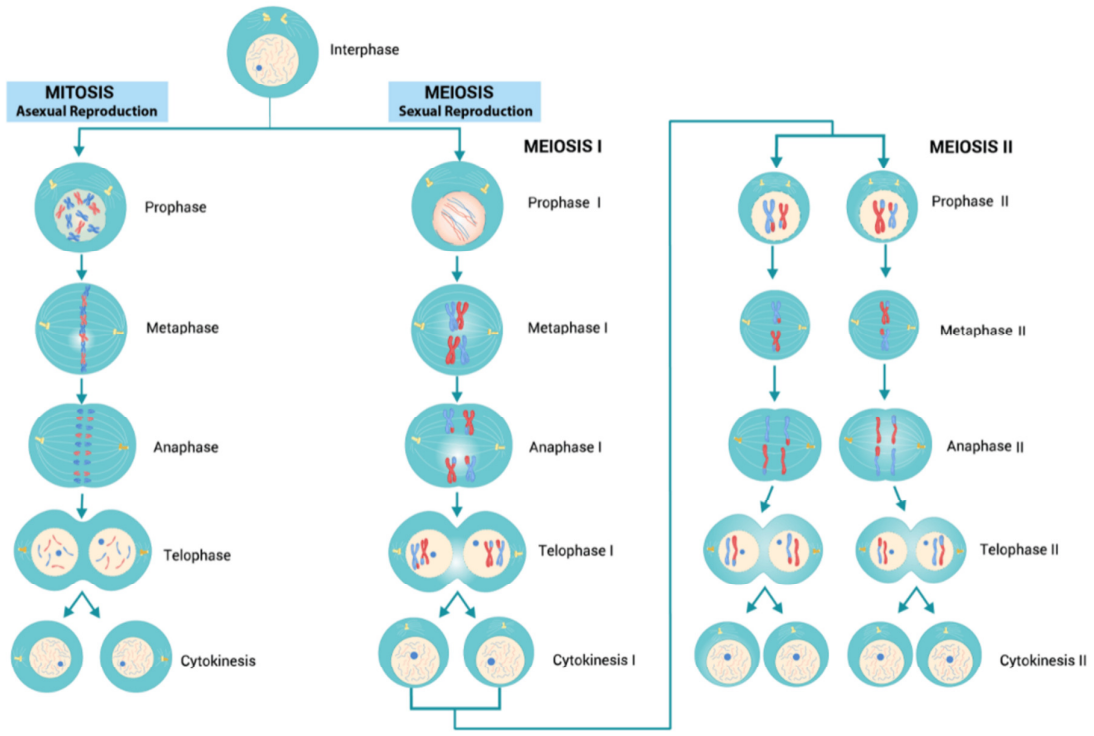

Figure 2: Representing the overview about mitosis and meiosis process [Mometrix Test Preparation].

When we mention the c-Value of the Nuclear Genome, we are referring to the total amount of DNA present in the nucleus of an organism. It is measured as the c-value, which can be in the form of the number of base pairs or picograms of DNA. the total amount of DNA in a cell) and the complexity of an organism. In general, more complex organisms have larger amounts of DNA in their cells compared to simpler organisms. The C-value is a measure of the genetic material (DNA) in an organism, while physical size or complexity refers to how big or complicated the organism is. Compare how big E is. Coli and humans are compared. But there are some exceptions to this comparison.

For example, while humans have 3. 2 billion DNA bases, wheat has 17 billion DNA bases, which is almost 6 times more. The Marbled Lungfish has a lot more DNA bases than a human, about 45 times more. It contains about 133 billion DNA bases. A fresh water amoeboid called Polychaos dubium has even more, about 200 times more than a human. It contains about 670 billion DNA bases. This confusing problem, called the C-value paradox, can be understood by knowing that not all DNA in the cell nucleus contains genes. In larger genomes, a significant amount of DNA does not code for genes. In many organisms, there are long stretches of DNA that don't have genes or any genetic information between them. A lot of the DNA that is not related to genes is made up of transposable elements. These elements are a type of DNA that can copy themselves. Other than genes, there are short pieces of DNA that repeat a lot in different ways. Junk DNA is a term used for nonfunctional DNA that is found together.

This test is about any suggested explanation for what non-coding (junk) DNA does. Can anyone explain why an onion needs about five times more non-coding DNA than a human even though humans have a smaller genome size. Why is there a range of genome sizes in organisms of similar complexity, such as onions. The term onion test was first used in April 2007 by T. Ryan Gregory is a biologist from Canada who studies evolution and genomes. We can make this statement simpler by saying 1c equals 3000 Mb. In this equation, c represents the amount of DNA in a gamete. When a sperm and egg come together, the resulting zygote is 2c, which is equal to 6000 Mb. Before the zygote can split into two cells, it needs to duplicate its DNA. This makes the amount of DNA double, reaching 12,000 megabytes. When the zygote splits, each new cell gets half of the DNA and becomes 2c which equals 6000 Mb. Then every cell will make copies of itself, so that there are now four cells that are the same. After that, each of these cells divides and becomes two new cells, making each cell half the size it was before. From now on, each cell in the embryo will have a size of 6000 Mb before a certain phase and 12,000 Mb after that phase. This is also true for the cells of unborn babies, young ones, and grown-ups. The cells used to make this chromosome spread were grown-up cells in metaphase, and each cell is four times the normal amount of DNA, which is equal to 12,000 Mb. Some things are not very common, like certain stages of cells that make germ cells and some unusual situations where liver cells have extra sets of chromosomes [9], [10].

Human gametes have 23 chromosomes. In simple terms, this statement means that the value of 'n' (the number of chromosomes in a gamete) is equal to 23. When one sperm with 23 chromosomes combines with one egg with 23 chromosomes, the resulting zygote will have a total of 46 chromosomes. However, the amount of DNA (c) changes when it replicates, but the number of chromosomes (n) stays the same. A replicated chromosome is just one chromosome that has been copied. So, after S phase, the zygote remains with 46 chromosomes. When the zygote splits into two cells, each cell has 46 chromosomes and is still considered as 2n = 46. Every cell in the early stages of development and throughout life has 46 chromosomes, except for a few special cases. In a normal human cell, a chromosome goes back and forth between being copied and not copied, and between being compact and not compact. The process of replication is simple to understand. If a cell decides to split into two new cells, it must first copy its DNA. This happens during the S phase. Before S phase, the chromosomes have one piece of DNA. After S phase, the chromosomes have two identical pieces of DNA. The condensation is a complicated process because genetic material in eukaryotic cells is always wrapped around proteins.

During the resting phase of a cell, a chromosome mostly exists as a 30 nm fiber. This helps it to fit in the nucleus and lets enzymes do activities on the DNA like making RNA, copying

DNA, and fixing DNA. The chromosome gets more compacted and certain processes stop at the beginning of mitosis. This is needed to make the chromosomes compact and able to move to the opposite ends of the cell. After mitosis finishes, the chromosome goes back to its original 30 nm fiber structure. Remember that each of our cells contains one chromosome from our mother and one from our father for chromosome 1. Here, we explain the ways mitosis and meiosis are different in humans, who have 46 chromosomes. Understanding the differences between these basic processes in cells is essential for your understanding of genetics throughout the course.

When genetic information is passed from one organism to another without involving sexual reproduction completed through a process called mitosis metaphase, anaphase, and telophase. In prophase, the chromosomes condense and the nuclear membrane breaks down. The spindle fibers begin to form. In metaphase, the chromosomes line up along the middle of the cell. In anaphase, the sister chromatids separate and move to opposite ends of the cell. In telophase, the chromosomes decondense and nuclear membranes start to form around each set of chromosomes. Finally, cytokinesis occurs, where the cell splits into two separate cells. Change the order and names of the words: Anaphase, Metaphase, and Telophase cell. This means that mitosis decreases the amount of cytoplasm in the cell, but does not affect the number of chromosomes. Daughter cells are new cells formed after a cell divides. Some of the DNA in an organism does not contain instructions for making genes. education institutions, there is a requirement for students to submit a written document called a thesis or dissertation.

CONCLUSION

Eukaryotes are organisms with complex cells, including plants, animals, and fungi. In these organisms, the majority of their DNA does not contain instructions for making proteins or other important molecules. Instead, it is thought to have little or no purpose. This DNA doesn't have a specific job and is known as junk DNA of coding DNA in a genome does not correlate with the complexity or size of an organism. In simpler terms, it means that the length of DNA sequences does not necessarily determine how complex or large an organism is size or amount of genetic material present in an organism. An organism is a living thing such as a plant, animal, or human. The c-value is the amount of DNA in a reproductive cell. Humans are a type of living beings 3000 Mb means 3000 megabytes. The n-value is the number of chromosomes in a reproductive cell. Humans have 23 pairs of chromosomes in their cells. A regular cell in your body has 6000 Mb and 46 units before replication. DNA replication is the process of making a copy of DNA. In this case, 4c means there are 12,000 million base pairs in the DNA and 2n represents that there are 46 chromosomes.

REFERENCES:

[1] R. A. Neumüller and J. A. Knoblich, “Dividing cellular asymmetry: Asymmetric cell division and its implications for stem cells and cancer,” *Genes and Development*. 2009. doi: 10.1101/gad.1850809.

[2] W. Shao and J. Dong, “Polarity in plant asymmetric cell division: Division orientation and cell fate differentiation,” *Developmental Biology*. 2016. doi: 10.1016/j.ydbio.2016.07.020.

[3] W. Margolin, “Themes and variations in prokaryotic cell division,” *FEMS Microbiology Reviews*. 2000. doi: 10.1016/S0168-6445(00)00038-3.

[4] M. Louveaux, J. D. Julien, V. Mirabet, A. Boudaoud, and O. Hamant, "Cell division plane orientation based on tensile stress in Arabidopsis thaliana," *Proc. Natl. Acad. Sci. U. S. A.*, 2016, doi: 10.1073/pnas.1600677113.

[5] L. Wang and Y. L. Ruan, "Regulation of cell division and expansion by sugar and auxin signaling," *Frontiers in Plant Science*. 2013. doi: 10.3389/fpls.2013.00163.

[6] A. Wodarz and W. B. Huttner, "Asymmetric cell division during neurogenesis in Drosophila and vertebrates," *Mechanisms of Development*. 2003. doi: 10.1016/j.mod.2003.06.003.

[7] M. K. Zhiponova *et al.*, "Brassinosteroid production and signaling differentially control cell division and expansion in the leaf," *New Phytol.*, 2013, doi: 10.1111/nph.12036.

[8] J. D. Lathia *et al.*, "Distribution of CD133 reveals glioma stem cells self-renew through symmetric and asymmetric cell divisions," *Cell Death Dis.*, 2011, doi: 10.1038/cddis.2011.80.

[9] A. L. Bottomley *et al.*, "Coordination of chromosome segregation and cell division in Staphylococcus aureus," *Front. Microbiol.*, 2017, doi: 10.3389/fmicb.2017.01575.

[10] A. Mashaghi and C. Dekker, "Systems and synthetic biology approaches to cell division," *Systems and Synthetic Biology*. 2014. doi: 10.1007/s11693-014-9132-z.

CHAPTER 3

MENDELISM AND NEO-MENDELISM: UNCOVERING THE NATURE OF GENE EXPRESSION

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ABSTRACT:

Mendelism is the study of genetics, particularly in traits that are controlled by a single gene. These principles were discovered by Gregor Mendel, a monk and biologist from Moravia, who established important laws that form the basis for classical genetics. Mendel lived in a monastery that focused on teaching and research. He had the freedom to study different subjects that interested him, such as math, plants, physics, and weather. He did careful experiments with pea plants in the monastery garden. From this, he figured out that the parts that determine traits are not mixes of parent traits, but are instead separate things that are passed down individually from one generation to the next. Mendel's discoveries were mentioned in a report in 1865 but people did not pay attention to it for a few years. Charles Darwin didn't read the Mendel's paper he got. Mendel is now known as the father of genetics because his work was rediscovered and seen as important. Mendelian inheritance is how parents give their genes and traits to their children. The different ways that genes can be passed down from parents to children are called autosomal dominant, autosomal recessive, X-linked dominant, and X-linked recessive. After Mendel's laws were found again, scientists studied various plants and animals and noticed many cases where the ratios predicted by Mendel's laws did not hold true. Based on these observations, scientists discovered different ways in which genes are passed down or work together. This can be explained by changing Mendel's law, so it is called post Mendelian genetics or Neo-Mendelian genetics.

KEYWORDS:

Dominant Epistasis, Genes, Plants, Traits.

INTRODUCTION

The main ideas of Gregor Mendel's model of how traits are passed down through generations have been accepted and proven true for more than 100 years. They can tell us about the traits that are passed down from parents to their children, in many different living things, including people. Traits that you inherit from your parents are determined by things in your body called genes. Genes are found in pairs, meaning they exist in two copies within an organism. Genes can have different forms, which are now called alleles. When a living thing has two different forms of a gene, one form will cover up the other form and decide how the living thing looks. When making eggs or sperm, each cell gets one of the two copies of a gene. The copy chosen for each cell is random. This is called the law of segregation. The genes for different traits are passed down separately from each other. These rules are the basis for how traits are passed down and how an organism's genetic makeup decides its observable characteristics. But there are some special cases, additions, and differences that need to be included in the explanation of how traits are inherited [1], [2].

Some of the differences in Mendel's rules have to do with individual genes. These are some examples of: Multiple alleles are multiple variations of a gene that can exist within a population. Mendel only studied two different versions of his pea genes, but in actual populations, there are usually more than two versions of a gene. Incomplete dominance is a genetic concept where neither allele is completely dominant or recessive. Instead, a blending or mixture of traits is observed in the offspring. Two versions of a gene may create a mixture of characteristics when both are present, instead of one completely controlling the characteristics. Codominance means that two different versions of a gene are equally expressed in an individual. This means that both versions of the gene are seen in the traits or characteristics of the individual and neither version is dominant or recessive over the other. Two versions of a gene can both have an effect on how an organism looks or behaves, instead of just one version being responsible for the whole outcome. Pleiotropy is a term used to describe a situation where one gene has multiple effects or influences on different traits or characteristics. Some genes influence multiple traits, rather than just one trait [3], [4].

Lethal alleles are specific genes that, when present in an organism's chromosomes, can cause severe or fatal health problems. Some genes have different versions called alleles. These alleles can cause problems and prevent survival when they are either the same or different. Sex linkage refers to the inheritance of certain traits or characteristics that are more commonly observed in one sex than the other. Genes on sex chromosomes, like the X chromosome in humans, have different ways of being passed down compared to genes on non-sex chromosomes. This article is about different types of genes and traits that affect how they are expressed. It explains multiple alleles, which are different versions of a gene that can determine different traits. It also talks about incomplete dominance, which happens when the two alleles mix together to create a new trait. Lastly, it discusses codominance, where both alleles are fully expressed and can be seen in the organism. Pleiotropy refers to when one gene affects multiple traits or characteristics in an organism. Lethal alleles are genes that, when present in certain forms, can cause the death of an organism. Different versions of multiple genes can affect certain traits or characteristics in an organism. Other variations of Mendel's rules involve how different genes can work together in pairs or in larger groups. Several traits are influenced by multiple genes. When two genes impact the same function, they can work together in various ways [4], [5].

Complementary genes are genes that work together to produce a certain trait or characteristic. They complement each other's functions to achieve a desired outcome. If you have two different genes with recessive alleles, they can both cause the same physical characteristics or traits to be expressed. Epistasis is a term used in genetics to describe a type of interaction between different genes. The types of a gene can hide or cover up the types of another gene. Furthermore, there are genes that are close to each other on a chromosome and are connected genetically, which means that they do not separate on their own. Polygenic inheritance refers to the inheritance of traits that are controlled by multiple genes, rather than just one gene. Environmental effects refer to the influence of the environment on the expression of these traits. Lots of things that matter to us every day like how tall we are, the color of our skin and eyes, and how likely we are to get sick with diabetes are influenced by many different factors working together. These things may be because of your genes, or because of where you live, or both of them together.

Polygenic inheritance refers to the inheritance of traits that are controlled by multiple genes. Some traits are controlled by many genes. In polygenic inheritance, traits usually vary in a range of ways instead of fitting into distinct groups. Environmental effects are the changes and impacts that occur in the natural world as a result of human activities or natural events.

Many traits are affected by both genes and the environment, which work together to determine how certain characteristics are shown in a person or organism. The genes we have and the things around us affect how traits show up. Sometimes, not everyone with a certain genetic makeup shows the same physical traits. Also, some people with a certain genetic makeup can have stronger or weaker versions of those traits [6], [7].

DISCUSSION

Sir Gregor Johann Mendel was an Austrian monk who did experiments on plant breeding using garden pea. He shared his findings in 1865. But other scientists, named Tschermak, Correns, and DeVries, found his work again in 1900, many years after Mendel had died. However, because Mendel was the one who first came up with ideas about how traits are passed down, he is considered the person who created genetics. Mendel conducted experiments by breeding tall pea plants with dwarf pea plants. The small parts from the flowers of tall plants were taken off and their sticky parts were sprinkled with pollen from flowers of short plants. The scientists also did another test. They took off the anthers from small plants and put pollen from tall plants on their stigmas. In the next spring, we collected seeds from the new plants and planted them. Mendel discovered that all the plants in the first generation, also known as F1, grew to be tall. He let them fertilize themselves. He gathered the seeds once more. The next year, after he planted the seeds, he discovered that most of the plants were tall and only a small portion were short. He did the experiment multiple times and discovered that there were three tall plants for every one dwarf plant.

He attempted to breed pea plants that were different in seven distinct characteristics or traits. These were the first ones. Plants with red flowers and plants with white flowers; Axillary flowered means that the flower grows where the leaf meets the stem. Terminal flowered means that the flower grows at the top of the stem. The comparison is between yellow seeds and green seeds. Round seeded means that the seeds have a circular shape, while wrinkled seeded means that the seeds have a crumpled or creased appearance. Green pod and yellow pod 6 are being compared. There are two kinds of plants: one type has big pods and the other type has smaller pods. Plants that are very tall compared to plants that are very short. Plants with opposite traits existed in different types that could pollinate themselves, so they consistently showed only one type of characteristic from one generation to the next. Crossbreeding plants that differed in just one characteristic is known as a monohybrid cross. Mendel also experimented with combining different characteristics, like tall and red flowers with short and white flowers. These crosses are called dihybrid crosses [8], [9].

Mendel's Principles of inheritance

According to what Mendel discovered in his experiments, he came up with the following rules about how traits are passed down from parents to children. The law of segregation or purity of gametes means that during reproduction, traits from parents are divided and only one trait is passed on to offspring. When gametes are formed, the two chromosomes in each pair separate and go into two different cells that become the gametes. This is a law that applies to all organisms that reproduce sexually. It means that when gametes are formed, the two parts of a pair are separated into different gametes. Every gamete gets one part of a pair of factors and the gametes are pure. Mendel's factors that he discovered were later called genes. Two The law of dominance means that one trait in an organism can be more dominant or stronger than another trait. characteristics or features), offspring receive a combination of genetic information from both of their parents. This genetic information determines the traits that an individual will possess.

The color of your eyes, color of a flower, and shape of a seed are determined by a set of genes working together. When both genes in a pair are the same type (for example, both are tall genes or both are short genes), they are called homozygous genes. When someone has two copies of the same gene for a trait, such as having brown eyes or a red flower, it is called homozygous. When two chromosomes have different genes (one for red flowers and one for white flowers) controlling the same feature (flower color), it's called heterozygous. The genes for red and white flower color are different versions of the same gene, which is the gene that determines flower color. Different versions of the same gene are called Alleles (Figure 1). The second law of inheritance says that when there are two different genes for certain traits, one gene is more powerful and will be expressed over the other gene. If a pea plant has two tallness genes (TT), it is homozygous. If it has one tallness gene and one dwarfness gene (Tt), it is heterozygous. Tall means a person or thing that is higher in height than others. A recessive gene is the opposite of a dominant gene. characteristic) is one that only appears when both parents pass down the same gene. The plant is only dwarf when both of its genes are in the same condition. The law of dominance was proven to be true in both monohybrid and dihybrid crosses for all seven traits that Mendel studied in garden peas [8], [10].

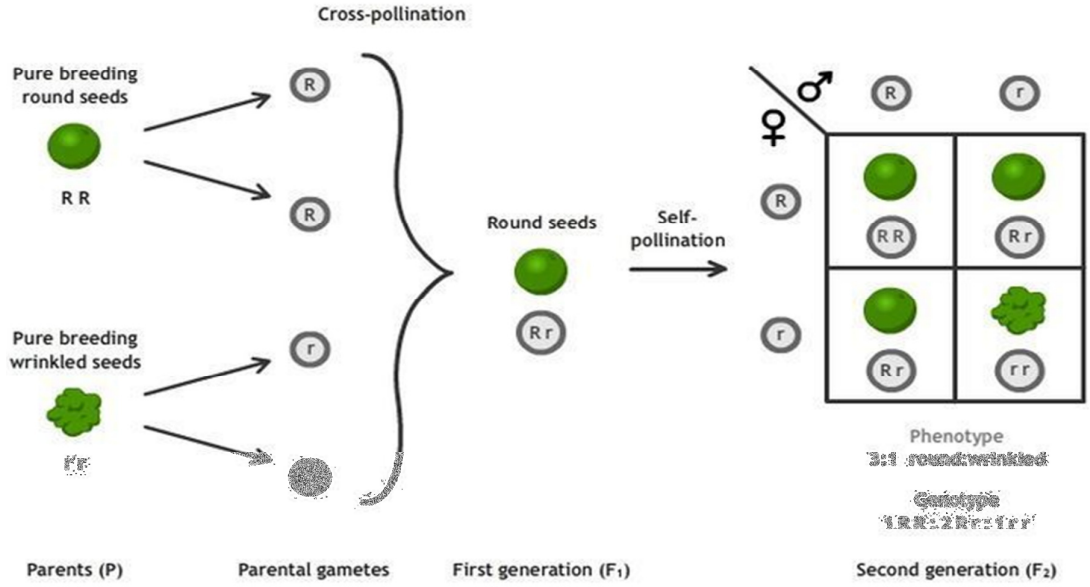

Figure 1: Representing the overview about Mendel's Principles of inheritance [Science Learing Hub].

Law of Dominance States

The law of dominance states that some traits or characteristics are stronger and will show up in offspring even if only one parent carries the trait. characteristics, features, qualities) from parents to offspring, there are some traits that have a greater likelihood of being passed down than others. This is due to genetic variations and the way these traits are passed on through genes. The color of our eyes, the color of flowers, and the shape of seeds are decided by a set of genes. When both genes in a pair are the same type (like both being blue or both being yellow), it is called homozygous. When both parents pass on the same gene for eye color or flower color, and the result is brown eyes or red flowers, it is called homozygous. When two chromosomes have different genes for a certain feature (like flower color), and one has a red flower gene while the other has a white flower gene, it is called heterozygous. The things that determine if a flower is red or white are different versions of the same thing called a gene.

Different versions of a gene are called alleles. The second law of inheritance states that when there are two different genes, one will have a stronger effect than the other. If both genes are for tallness (TT), the pea plants will be tall. If one gene is for tallness and another for dwarfness (Tt), the pea plants will still be tall. A gene that is not dominant is called recessive gene. characteristic or trait) is one that can only be expressed when there are two copies of the gene responsible for it. In other words, both parents must contribute a copy of the recessive gene in order for the feature to be seen in their offspring. The plant will only be small when both of the genes are the same (tt). The law of dominance was proven to be true when Mendel studied seven different traits in garden peas and looked at both crosses involving one trait and crosses involving two traits.

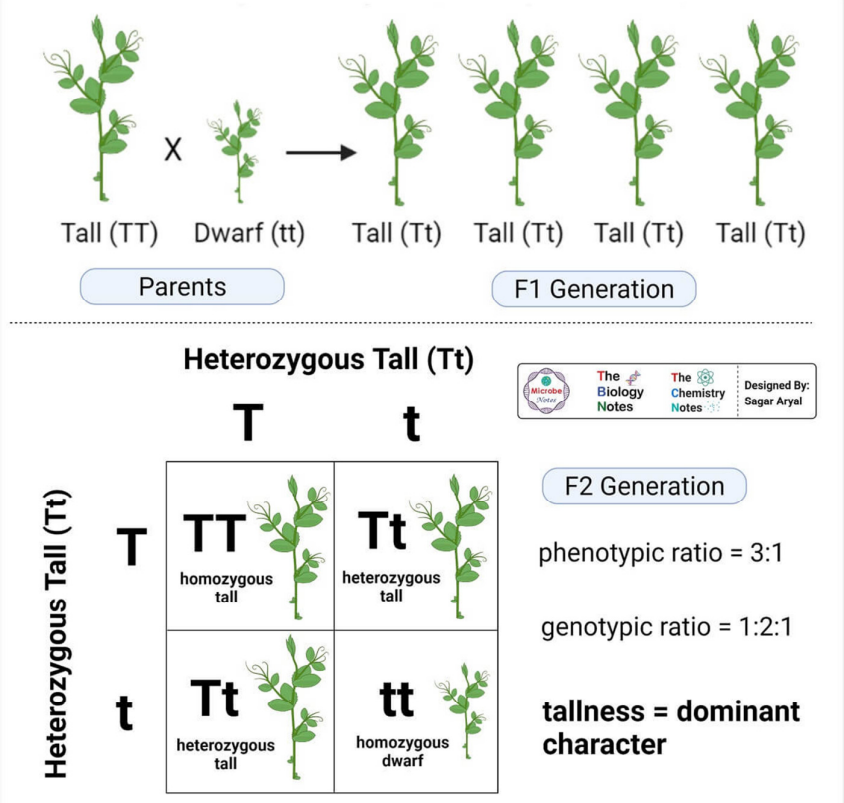

Figure 2: Represeneting the that two pairs of genes are not connected to each other and are inherited independently [Microbe Notes].

Figure 2 shows that two pairs of genes are not connected to each other and are inherited independently. Y represents the colour yellow in the seed, while y represents the colour green. R represents a round shape of the seed, while r represents a wrinkled shape. The combination of these genes determines the appearance of the seed, which is called the genotype. This appearance is what we can see and is called the phenotype. The proportion of offspring in the crosses is called the phenotypic ratio. However, when more scientists started doing genetic experiments, they realized that Mendel's laws are not always true.

Gene interaction

Gene interaction refers to the way genes work together to produce certain characteristics or traits in an organism. This interaction can involve multiple genes interacting with each other, or genes interacting with the environment. By understanding how genes interact, scientists can gain insights into how certain traits are inherited or expressed and how different genetic

variations can affect an organism's development and health. Mendelian genetics cannot explain all types of inheritance where the physical characteristics ratios are different from what Mendel's theories predict. For example, the phenotypic ratios can be different from 3:1 for monohybrid and 9:3:3:1 for dihybrid in the second generation (Figure 3). This happens because sometimes one gene can be more dominant than another, or because there are more than two genes involved, or because some genes can be harmful. These types of genetic interactions between alleles of one gene are called allelic interactions. Non-allelic interactions happen when multiple genes affect each other to control one trait. Therefore, the way a gene is expressed is not separate from other genes and relies on whether other genes are there or not. These differences from the idea that one gene determines one trait are called the Factor Hypothesis or Interaction of Genes.

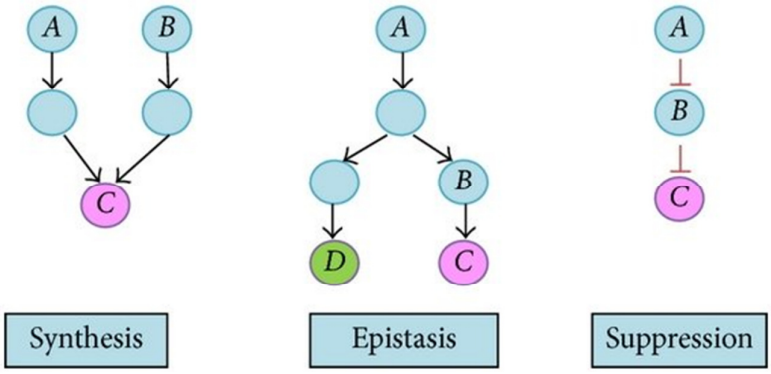

Figure 3: Representing the types of gene interactions [Research Gate. Net].

Lethal Factor (2:1)

A lethal factor is a gene that causes the individual carrying it to die. Recessive lethal traits are only shown when an organism has two copies of the trait. If an organism has only one copy, it does not show any effects. There are genes that have a strong visible effect but can cause death when only one copy is present, like the gene for yellow fur in mice. However, many genes have both a hidden effect and can cause death when two copies are present, like the gene that makes barley seedlings albino. Dominant lethal traits are removed from a population because they cause the death of an organism even if it is in a mixed state with a different trait. For example, the epiloia gene in humans is an example of this. Conditional lethals are lethal only under certain conditions. For example, a type of barley mutant will die when exposed to low temperatures. Balanced lethals are organisms that have both dominant and recessive lethal genes. Both dominant and recessive forms of the gene will cause the organism to die. One example is the balanced lethal system observed in Oenothera. Gametic lethals prevent the reproductive cells from joining together, for example, the segregation distorter gene in male fruit flies. Semi-lethal genes don't kill all organisms, like the xentha mutants in certain plants. Gene interactions occur when different genes work together to influence a trait. In duplicate recessive epistasis, recessive alleles at either of two locations can hide the effects of dominant alleles at those locations.

This is also called complementary epistasis. The sweet pea flower color is a good example of duplicate recessive epistasis (Figure 4). The purple color of a sweet pea flower is controlled by two strong genes called A and B. When these genes are in separate individuals with the genetic makeup of AAbb or aaBB, or in recessive individuals with the genetic makeup of aabb, they create white flowers. When a purple flower with the genes AABB and a white flower with the genes aabb are bred together, their offspring (F1) will have purple flowers.

The crossing of F1 plants resulted in F2 generation plants with 9 purple flowers and 7 white flowers.

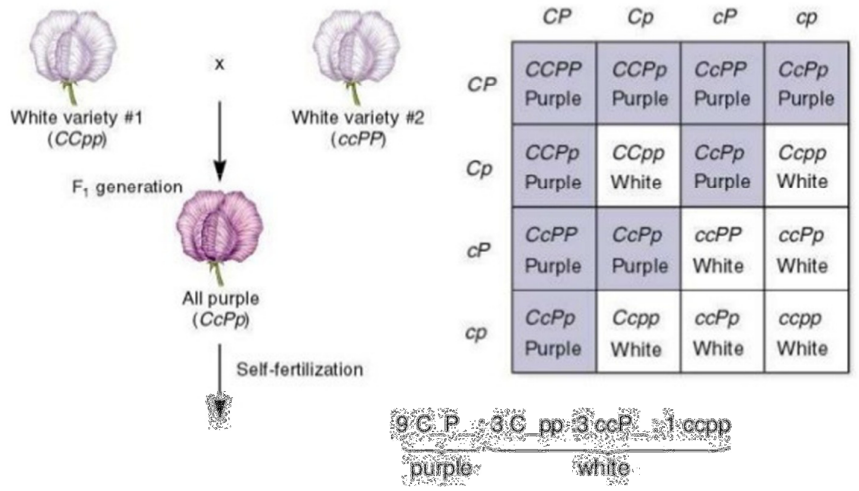

Figure 4: Representing the overview about the complementary epistasis [Toppr].

Here, a recessive gene is more important than the B/b genes and it hides their effects. Another type of gene, called b, can hide or overshadow the effects of the gene A/a. So, in the F2 generation, plants that have the genetic makeup A-B-(9/16) will have purple flowers. Plants with aaB-(3/16), A-bb-(3/16), and aabb (1/16) genotypes will produce white flowers. So, there are only two types of traits in this generation, purple and white. The usual ratio of traits is changed from 9 purple : 3 purple and white : 3 white to 9 purple : 7 white in the next generation.

Dominant Epistasis

Dominant Epistasis is a genetic pattern where one gene hides the effect of another gene. In this pattern, there is a ratio of 12 individuals with the dominant trait, 3 individuals with a hybrid trait, and 1 individual with the recessive trait. When one gene can hide the effects of other genes, it is called dominant epistasis.

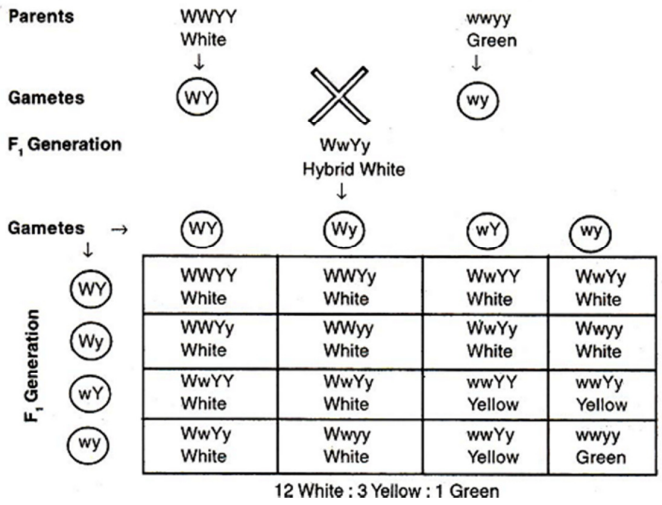

Figure 5: Representing the example of Dominant Epistasis [Biology Exam 4 U].

In simpler terms, when there is a dominant gene, it hides the effects of a different gene that may be dominant or recessive. This is also known as simple epistasis. One example of dominant epistasis is when the color of the fruit in summer squash is determined. This cucumber can be found in three different colors: white, yellow, and green. The color white is determined by the stronger gene W, and the color yellow is determined by the stronger gene G. White is more powerful than both yellow and green. The recessive condition (wwgg) produces green fruits. When a plant with white fruits and a plant with yellow fruits were bred together, their offspring (called F1) had white fruits. When the F1 plants were mated with each other, the resulting F2 plants had fruits that were white, yellow, and green in color. The ratio of these colored fruits was 12 white : 3 yellow : 1 green (Figure 5). In simple terms, the letter W is more powerful than w, and can block the effects of alleles G and g. So, it will hide the appearance of the G/g alleles. So, in this case, some plants will produce white fruits, some will produce yellow fruits, and some will produce green fruits. Therefore, the usual ratio of 9:3:3:1 when studying two different traits together changes to a ratio of 12:3:1 in the second generation. The same kind of gene interaction has been observed for the color of skin in mice and the color of the outer coat of seeds in barley.

Duplicate Dominant Epistasis

Duplicate dominant epistasis occurs when two dominant alleles at different loci both suppress the expression of a third gene. In this type of interaction, the presence of either dominant allele is sufficient to mask the effect of the third gene. The ratio of phenotypes resulting from duplicate dominant epistasis is typically 15:1, which means that for every 15 individuals expressing the dominant phenotype, there will be one individual expressing the recessive phenotype.

Parents — Awned Rice AABB × Awnless Rice aabb

F_1 — AaBb — Awned Rice

F_2

	AB	Ab	aB	ab
AB	AABB [A]	AABb [A]	AaBB [A]	AaBb [A]
Ab	AABb [A]	AAbb [A]	AaBb [A]	Aabb [A]
aB	AaBB [A]	AaBb [A]	aaBB [A]	aaBb [A]
ab	AaBb [A]	Aabb [A]	aaBb [A]	aabb [a]

A = Awned Rice, a = Awnless Rice

Figure 6: Representing the duplicate dominant epistasis character in rice [Toppr].

When a strong gene at either of two spots can hide the effects of weak genes at those spots, it is called duplicate dominant epistasis. This is also known as the action of duplicate genes. One example of duplicate dominant epistasis can be seen in the awn character of rice. The

growth of awn in rice is managed by two strong duplicate genes (A and B). Having either of these two alleles can create awn. The awnless trait only appears when both of these genes are in the same hidden state (aabb). When a certain type of plants with hairy ends (awned) and another type without hairy ends (awnless) are bred together, their offspring (F1) will have hairy ends. The mating of two different F1 plants resulted in the production of plants with and without awns in a ratio of 15:1 in the F2 generation (Figure 6). This can be described in the following way. The allele A controls whether or not plants develop awn, and it is more important than the B/b alleles. Another strong genetic trait B can override the effects of the alleles A/a. People who have this gene will also develop the trait of having an awn. So, in F2, plants with certain gene combinations will grow awns. The awnless condition will only happen if both parents pass on the recessive awnless gene to their offspring. This is a rare occurrence, happening in only 1 out of every 16 cases. This means that only two types of plants are created instead of the usual four types that are expected. We have discovered that the way genes work is similar for both the process of nodulation in peanuts and the non-floating characteristic in rice.

CONCLUSION

The idea of heredity has been around for a long time, just as long as civilization. It was not by chance that animals and plants had babies that looked a lot like them, and it was also normal for them to only reproduce with other similar animals or plants. In the past, people noticed that when parents had children, the children were never exactly the same as their parents. There were always small differences. It seemed like both parents had a part in this. A long time ago, even during the Babylonian era, farmers knew that they could control certain traits by choosing which animals or plants should mate and reproduce. Ancient Egyptians improved their crops by mixing plants together, as shown by their old writings. However, even though the ancients knew that hereditary manipulation had practical benefits, there are no records before the Greeks that show their ideas about how heredity works.

REFERENCES:

[1] G. E. Allen, “Eugenics as an International Movement,” in *International Encyclopedia of the Social & Behavioral Sciences: Second Edition*, 2015. doi: 10.1016/B978-0-08-097086-8.82001-2.

[2] J. C. Loehlin, “The Emperor’s New Clothes: Biological Theories of Race at the Millenium,” *Intelligence*, 2002, doi: 10.1016/s0160-2896(01)00089-7.

[3] K. G. Valente, “‘A finite universe ?’ Riemannian geometry and the Modernist theology of Ernest William Barnes,” *British Journal for the History of Science*. 2005. doi: 10.1017/S0007087405006722.

[4] P. J. Bowler, “Alternatives to darwinism in the earlytwentieth century,” in *The Darwinian Tradition in Context: Research Programs in Evolutionary Biology*, 2017. doi: 10.1007/978-3-319-69123-7_9.

[5] P. G. ’espinasse, “On the logical geography of neo-mendelism,” *Mind*. 1995. doi: 10.1093/mind/104.416.827.

[6] J. P. Gaudillière, “Mendelism and medicine: Controlling human inheritance in local contexts, 1920-1960,” *Comptes Rendus l’Academie des Sci. - Ser. III*, 2000, doi: 10.1016/S0764-4469(00)01268-3.

[7] A. Lazcano, "Alexandr I. Oparin and the Origin of Life: A Historical Reassessment of the Heterotrophic Theory," *J. Mol. Evol.*, 2016, doi: 10.1007/s00239-016-9773-5.

[8] N. Serpente, "More than a Mentor: Leonard Darwin's Contribution to the Assimilation of Mendelism into Eugenics and Darwinism," *J. Hist. Biol.*, 2016, doi: 10.1007/s10739-015-9423-6.

[9] M. Morrison, "Modelling populations: Pearson and Fisher on Mendelism and biometry," *Br. J. Philos. Sci.*, 2002, doi: 10.1093/bjps/53.1.39.

[10] R. A. Ankeny, "Marvelling at the marvel: The supposed conversion of A.D. Darbishire to Mendelism," *J. Hist. Biol.*, 2000, doi: 10.1023/A:1004750216919.

CHAPTER 4

MULTIPLE ALLELES: EXPLORING THE DIVERSITY OF PLANT POPULATIONS

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ABSTRACT:

Multiple alleles in a population mean there are many different forms of a gene. In organisms that have two copies of each gene, called diploid organisms, each organism can show two different forms of a gene at once. They can be the same type of gene, which is called a homozygous gene combination. On the other hand, the genotype can have different types of alleles together, which is called a heterozygous genotype. Haploid organisms and cells have only one version of a gene, but the population can still have many different versions of that gene. In both simple and complex organisms, new traits are created by unplanned changes in the genes. These changes can happen in many different ways, but they cause a different order of building blocks in the DNA. The genetic code is like a language that uses groups of three nucleic acid bases to represent different amino acids. A mutation is when the order of amino acids changes, which can happen in a small or big way. Small changes in a few amino acids can create many variations in a group of organisms. These variations all work in a similar manner, but to varying extents. Different changes in the protein caused by mutations can make it completely unable to work. Some changes in genes create new protein forms that help living things develop new ways of working, shaping, and doing things.

KEYWORDS:

Alleles, Genes, Mutltiple Alleles, Traits.

INTRODUCTION

The word allele is a general term that describes the different forms of a gene. These contrasting forms of a gene are called alleles. Bateson used to think of these alleles as imaginary companions in Mendelian segregation. In Mendelian inheritance, a spot on a chromosome had two types of genes: a regular gene and its unusual recessive gene for wrinkled seed shape. But it is possible that the regular gene in peas might have many other changes besides the one for wrinkledness. In simple words, multiple alleles means that there are different versions of a gene located in the same spot on a chromosome. It can be a normal version and two or more different mutated versions of the same gene. To put it simply, multiple alleles refers to several different types of a gene. The idea of multiple alleles is explained using the term "multiple allelism. Dawson and Whitehouse in England came up with the term panallele to describe all the different gene mutations that can happen in one specific part of a chromosome. These are different from multiple factors because multiple factors are found in different places, while alleles are found in the same place. When there are three or more different types of genes that exist in the same place in the DNA, they are called multiple alleles. These multiple alleles have specific characteristics.

The study of multiple alleles can be conducted in a group of individuals. There are many different forms of a gene that can be found on matching chromosomes in the same position. Calculate the difference between 12 and 7. 5 is the result. The members of multiple alleles do not mix or swap with each other. Crossing over happens between two different genes but not within a single gene. FourMultiple alleles affect a specific trait [1], [2].

Different forms of a gene cannot work together to produce a functioning trait. The complementation test is useful for distinguishing between genes that are allelic located on the same gene and those that are not allelic. When a living thing has both a regular trait and two abnormal traits caused by different genes, it's called a complementation test. The usual allele is mostly stronger than other strange alleles, but those strange alleles may also be stronger or have a medium effect on how things look. Could you please provide the text that needs to be rewritten in simple words. When two different versions of a gene are combined, the physical appearance of an organism is different than the usual type. 8Rewrite this text using simpler language: Additionally, the offspring from these crosses in the second generation (F2) exhibit the usual ratio for a single trait. Use traditional genetics method by doing an allelism test. This test checks if two alleles are behaving independently, connected, or located in the same spot (which is called allelic). Imagine there are two alleles that are dominant. Each allele is responsible for creating a unique outcome, and when both alleles are present, their effects add up together. If the genes are on different chromosomes, when a parent with AAbb genes is crossed with a parent with aaBB genes, the expected ratio of traits in their offspring (F2) is 9:3:3:1 [3], [4].

This means that for every 9 offspring, 3 will have the traits A_3 will have the traits A_bb, 3 will have the traits aaB_, and 1 will have the traits aabb. If the genes are at the same location (locus), when a parent with A1A1 genes is crossed with a parent with A2A2 genes, the expected ratio of traits in their offspring is 1:2:1. This means that for every 4 offspring, 1 will have the traits A1A1, 2 will have the traits A1A2, and 1 will have the traits A2A2. Similar calculations can be done for other genetic models and crosses. another model to see which one performs better. Can you please provide the text that you would like me to rewrite in simple words. If you have a connection, the ratio may be unclear if you have a noisy quantitative trait. The importance of having multiple alleles is that it helps us learn more about how traits are passed down from parents to offspring. TH's perspective suggests that Morgan gained a lot of understanding about genes from multiple alleles. These alleles mean that a gene can change in various ways and have different outcomes. Multiple allelism suggests that varying amounts of heterochromatin can affect genes in different ways or to varying extents. Pseudo alleles are different versions of a gene that are found in the same spot or location. Sometimes, scientists have discovered genes that are not related to each other, but are located close to each other. These genes can influence the same trait or characteristic in the same way, as if they were different versions of the same gene. They are called fake alleles. These fake forms of genes that are very close together can mix and match during the process of crossing over, which is different from the normal genes [5], [6].

These different versions of a gene will have the same physical effect when there are two copies of the same version, but there will be a slight difference in effect when there are two different versions. In the fruit fly, the gene for red eye color is stronger and will be seen over the gene for white eye color. The red gene makes flies dark red in color when they have two copies of it. But when combined with a white gene, the red gene makes flies dark red in color.These are called isoalleles. Self-sterility in Nicotiana species is caused by different forms of a gene called alleles. These alleles are labeled as s1, s2, s3, and s4, and so on. The hybrids S1/S2 or S1/S3 or S3/S4 cannot have babies by themselves because their pollen

grains do not grow fully. However, the pollen from S1/S2 can successfully fertilize S3/S4 and create offspring. The genes that make plants unable to reproduce themselves probably work by controlling how fast the pollen tubes grow. In flowers where the pollen tube matches well, it grows faster towards the ovule. However, in flowers where there is a poor match, the pollen tube grows slower and the flower dies before fertilization can happen [7], [8].

Pseudoalleles are genes that are closely connected and have a similar function. A group of fake genes is called a fake gene series or a complicated gene location or a complicated area. The main qualities of pseudoalleles are listed below. Different versions of the same trait are controlled by false genes called pseudoalleles. Put simply, they work together. Pseudoalleles are special genes that are found in a particular area of a gene that has different sub-sections. So, they are in different places, but on the same area.. They do not often swap genes through crossing over. Simply put, crossing over happens between fake versions of genes, but it doesn't happen very often. They show a position effect where the molecules are in either the same or different positions. In individuals with Trans heterozygotes, these mutants create a mutated appearance. However, in individuals with cis-heterozygotes, they have a normal appearance. Pseudoalleles are like fake versions of alleles. There are many examples of pseudoalleles. Some famous examples include the lozenge gene and the star asteroid in a fruit fly called Drosophila.

DISCUSSION

Gregor Johann Mendel studied pea plants and found two important rules in genetics: the law of segregation and the law of independent assortment. He looked at seven different characteristics with two different types and found that there were only two types of genes for each characteristic. An allele is like a different version of a gene, and a locus is where that allele is found in an organism's genetic material. The idea that each gene only has two options has changed because we have discovered that genes can have more than two options. There is no limit to how many options a gene can have in a population. When a group of individuals has more than two options for a gene, it is called multiple alleles, or allelic series. The idea of having different forms of a gene applies to a group of organisms, not to each organism individually.

A certain organism can have up to two different versions of a gene on each pair of matching chromosomes. However, different members of a species can have various alternative forms of a gene. Multiple alleles have the same way of being passed down as two alleles but multiple alleles can have many different combinations of genes and physical traits. The formula to find the number of genotypes is $n(n + 1)/2$. Here, n represents the number of alleles. The number of homozygotes is n and the number of heterozygotes is $n(n-1)/2$. Now let's use a hypothetical example to explain the idea of multiple alleles at the molecular level. An organism has two sets of chromosomes, one set from the male parent and one set from the female parent [9], [10].

The genes are found on the chromosomes and each gene has a specific position on the chromosome called a locus/loci. On the other hand, a gene is a string of molecules that does particular jobs. A mutation is when there is a change in the DNA of a gene, and this can create a different variation of the gene called an allele or mutant. Multiple alleles are different versions or variations of a gene that happen because of changes in the DNA sequence that result from mutations at a specific location in the gene. In this example, the normal allele A changed into three different alleles called a1, a2, and a3. Mendel said that for each gene, there are only two versions called alleles.

One allele is dominant and the other is recessive. We now understand that this is a way of making things seem too simple. Although each person and all organisms with two sets of chromosomes can only have two versions of a gene, there can be various versions of the gene in a population, leading to many different combinations of two versions in different individuals. When there are different versions of a gene, the most common one in wild animals is called the wild type. It is seen as the normal or standard version. Other types or forms are seen as variations of this normal type, which means they are different from the natural form. The different form can either be weaker or stronger than the normal form of a gene. An example of having many options for coat color is seen in rabbits (Figure 1). Here, there are four different forms of the c gene. The normal version, C+C+, is shown as brown fur. The chinchilla look, called cchcch, appears as white fur with black tips. The Himalayan phenotype, chch, has black fur on the body parts far away from the center and white fur on the rest of the body. The albino phenotype is when an organism has white fur instead of colored fur. In situations where there are different versions of a gene, some versions may be stronger or more dominant than others. In this situation, the normal allele is stronger than all the others.

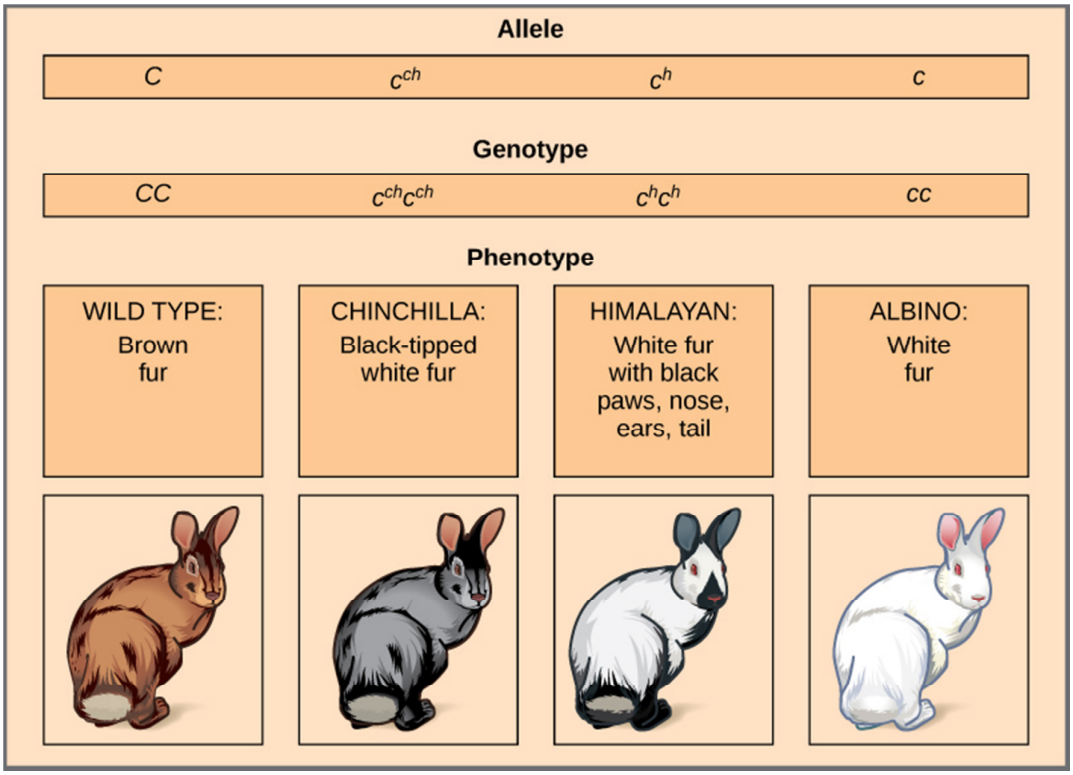

Figure 1: There are four different versions of the rabbit coat color gene (C) [Lumen Learning].

Chinchilla is kind of strong against Himalayan and albino, but not completely. Himalayan is stronger than albino. This hierarchy, or series of different forms of a gene, was discovered by looking at the physical characteristics of the offspring that have one copy of each different form of the gene. The wild-type version of a trait is usually stronger than any mutant versions because it has the right amount of a specific gene product. The mutant versions do not have enough of this gene product. In rabbits, there are different kinds of alleles. The normal allele gives rabbits a certain amount of fur color, but the mutant alleles give them less or no fur color at all. The Himalayan phenotype in rabbits happens because of a gene that only

produces color in the cooler parts of the rabbit's body. Instead, one gene mutation can be more powerful than all the other variations, including the normal type. This can happen when the changed gene affects the genetic instructions in a way that even if a person has one normal gene copy, they still show the changed trait. One instance of this is the Antennapedia mutation in a type of fruit fly called Drosophila. In this situation, the abnormal gene causes the gene product to spread more than usual. As a result, the Antennapedia heterozygote grows legs on its head instead of antennae.

Female parent (Stigma spot)	**Male parent (Pollen source)**		
	S_1S_2	S_2S_3	S_3S_4
S_1S_2	Self Sterile	S_3S_2 S_3S_1	S_3S_1 S_3S_2 S_4S_1 S_4S_2
S_2S_3	S_1S_2 S_1S_3	Self Sterile	S_4S_2 S_4S_3
S_3S_4	S_1S_3 S_1S_4 S_2S_3 S_2S_4	S_2S_3 S_2S_4	Self Sterile

Figure 2: Representing the different combinations of progeny in self- incompatibility [Brain Kart.Com].

Multiple alleles are different forms or variations of a particular gene that can exist within a population. These different forms can lead to different traits or characteristics in individuals. Different versions of a gene always take up the same position on corresponding chromosomes. So, there is no mixing of traits in the alleles of a group. The regular forms of a group of genes show dominant traits, while the changed forms may affect dominance or have a mix of traits. In plants, there are different versions of particular genes that are linked to the inability to reproduce or the inability to mate with oneself. Self-sterility means that a plant's pollen cannot grow on its own stigma and cannot fertilize its own ovules. In 1925, East discovered that Nicotiana plants have different versions of certain genes that cause them to be unable to mate with themselves. This is known as self-incompatibility or self-sterility. The gene for self-incompatibility is called S. There are different versions of this gene called S1, S2, S3, S4, and S5. The tobacco plants that were pollinated by each other did not always have the same genes. However, all the plants had different genes from each other.

When plants with different characteristics were crossbred, the long tube that carries the male reproductive cells did not grow as it should. However, we noticed that the pollen tube developed well when we crossed with types other than S1S2, like S3S4 (Figure 2). When we made crosses between seed parents with S1S2 and pollen parents with S2S3, we noticed that there were two different types of pollen tubes. The pollen grains with S2 did not work well,

but the pollen grains with S3 were able to make plants become fertilized. So, when the flowers from one area are mixed with the flowers from another area, the mixture of pollen from the two areas produces four different kinds of plants.

Usually, one gene determines one trait. But some genes can affect multiple traits, these genes are called pleiotropic genes and the condition is called pleiotropy. A gene called the s gene can cause sickle cell anemia in people who have two copies of it. When a gene affects multiple traits that are not connected to each other, it is called a pleiotropic gene. Please write this text using simple words: For instance, In cotton, there is a gene called Punjab hairy lintless. This gene makes seeds without any lint. This gene also makes the leaf have incomplete cuts, makes the boll smaller, and affects fertility. In a plant, a gene can make different parts like flowers, stem, and leaves turn red. However, this gene is not considered pleiotropic because it only affects one thing, which is the production of pigment. A gene that controls the growth of wings may be an old gene that is not used much anymore. This gene can also be called a bristle gene or a fecundity gene. Several other genes that are not dominant can have significant and often harmful effects on human beings. They are called syndromes. Penetrance refers to how likely it is for a gene to have the same physical traits in everyone who has it in the right genetic makeup.

For instance, when pea seeds have two copies of the w gene (ww), they all have a wrinkled shape. The seeds with WW or Ww genes are all round. Complete expressivity refers to when a gene makes all the individuals with that gene have the same traits. Instead of being consistent, many genes produce different characteristics in individuals who have the right combination of genes. In simple words, genes show themselves in all individuals with the right genetic makeup, which is called complete penetrance. However, not all individuals with specific genes will show the expected physical traits. This means that not everyone who has a certain trait shows it. When a gene is in the right genetic code, the percentage of individuals in which it can show itself is called its penetrance. So, if lima beans have a gene that causes a lack of chlorophyll, it will show up or affect 10% of the time. Most of the genes that don't fully show their effects also don't fully show their traits. Incomplete penetrance means that some individuals have a gene but it doesn't show up as a noticeable trait. Isoalleles are alleles that appear similar but are actually different when tested. A person with blood group A can have three slightly different types called IA1, IA2, and IA3. These types may seem similar, but they are actually different when tested. Pseudoalleles are genes that are very close together and can only be separated by rare genetic crossover events. These genes are referred to as pseudoalleles.

Usually, the connection between different forms of a gene can be understood in two ways: based on their structure and based on how they work. We call these connections "structural allelism" and "functional allelism," respectively. In structural allelism, if there are two or more changes in the same spot of the DNA, and these changes affect the same characteristic, scientists can use a test to determine if these changes can combine or not at the DNA level. If two mutations can mix together and create normal genes, they are not related to each other. However, if the mutations cannot mix and do not make normal DNA, they are related to each other. Changes in different positions of DNA can undergo a process called recombination, which we call structurally non-allelic. Changes in the same position of DNA cannot recombine, which we call structurally allelic.

Functional allelism is used to figure out if two mutations that control the same trait are found in the same gene or in different genes. This is tested by seeing if it complements the product. If two people have changes in the same gene, they are called functionally allelic and they won't have normal traits if they have children together. But if two people have changes in

different genes, their children will have normal traits. These people are called functionally non-allelic. So, if two changes are very similar in their structure and how they work, they are homoalleles. But if two changes work in a similar way but are different in structure, they are heteroalleles. Pseudoalleles are two genes that are related and have similar effects. They are found close to each other on a chromosome. When two genes are genetically linked, they usually get passed down together and might act as one gene. Pseudoalleles are not the same as pseudogenes.

Pseudogenes are nonfunctional copies of working genes, and they can be created through duplication or by making a copy of RNA. Pseudoalleles, on the other hand, are genes that are connected to each other at a specific location. The pseudoalleles are different from multiple alleles. Pseudoalleles are located near each other, while multiple alleles are different forms of a single gene. To tell the difference between fake versions of alleles and multiple versions, some clues have been found in the past. For example, we tried crossing different mutants together to see if they would mix, but it didn't work. We also noticed that when an organism had two different mutant genes, its appearance was in between the appearances of the two pure versions, instead of looking like the regular version. There have been some cases where a test for allelism shows positive results for genes that are not actually alleles, because of something called the position effect. This situation is called positional pseudoallelism, where two genes are found in different places and can be affected by their position. In positional pseudoallelism, when the genes are paired in a specific way, the coupling phase heterozygote has a normal or almost normal appearance, while the repulsion phase heterozygote has abnormal appearances.

CONCLUSION

Most people think that a potato's shape is always the same, but its roundness or length can be seen at a lower level. This is the first time anyone has found proof that there are different allele systems in a potato tuber. It can be compared to a similar study that was done on maize. The tuber shape gene that is less common is called a null or near-null gene. We can measure how much dominant alleles are different from each other. The concept of a near-null or null allele for a recessive gene is supported by the way we explain the effects of different gene variations in a specific location. When we separate additional measurements into specific characteristics inherited from both parents in experiments, we can determine which traits are more significant in describing the variation of genetic characteristics. Alleles are pairs of genes that are found in the same spot on a chromosome. In a diploid organism, each gene usually has only two versions. Multiple allelism is when a gene has more than two different versions. Allelism means that a gene can have different versions. These differences in genes, which are often caused by changes in DNA, create traits that can be passed down through families.

REFERENCES:

[1] L. Liu, D. K. Pearl, R. T. Brumfield, and S. V. Edwards, “Estimating species trees using multiple-allele DNA sequence data,” *Evolution (N. Y).*, 2008, doi: 10.1111/j.1558-5646.2008.00414.x.

[2] S. W. Guo and E. A. Thompson, “Performing the Exact Test of Hardy-Weinberg Proportion for Multiple Alleles,” *Biometrics*, 1992, doi: 10.2307/2532296.

[3] R. Thirumurugan, M. Kathirvel, K. Vallayyachari, K. Surendar, A. V. Samrot, and M.

Muthaiah, "Molecular analysis of rpoB gene mutations in rifampicin resistant Mycobacterium tuberculosis isolates by multiple allele specific polymerase chain reaction in Puducherry, South India," *J. Infect. Public Health*, 2015, doi: 10.1016/j.jiph.2015.05.003.

[4] D. V. Zaykin, A. Pudovkin, and B. S. Weir, "Correlation-based inference for linkage disequilibrium with multiple alleles," *Genetics*, 2008, doi: 10.1534/genetics.108.089409.

[5] Y. Han, J. N. Cameron, L. Wang, and W. D. Beavis, "The predicted cross value for genetic introgression of multiple alleles," *Genetics*, 2017, doi: 10.1534/genetics.116.197095.

[6] A. J. Roche, J. P. McFadden, and P. Owen, "Antigen 43, the major phase-variable protein of the Escherichia coli outer membrane, can exist as a family of proteins encoded by multiple alleles," *Microbiology*, 2001, doi: 10.1099/00221287-147-1-161.

[7] A. G. Clark, "Mutation-selection balance with multiple alleles," *Genetica*, 1998, doi: 10.1023/a:1017074523395.

[8] S. Aoki, "Network algorithm for the exact test of Hardy-Weinberg proportion for multiple alleles," *Biometrical J.*, 2003, doi: 10.1002/bimj.200390026.

[9] M. E. M. Peluso *et al.*, "DNA adducts and combinations of multiple lung cancer at-risk alleles in environmentally exposed and smoking subjects," *Environ. Mol. Mutagen.*, 2013, doi: 10.1002/em.21788.

[10] J. A. Muniz, M. S. Camargo, D. F. Ferreira, and R. D. Veiga, "Methods of estimation of the inbreeding coefficient in a diploid population with multiple alleles," *Cienc. e Agrotecnologia*, 2008, doi: 10.1590/s1413-70542008000100014.

CHAPTER 5

ANALYZING THE LINKAGE AND CROSSING-OVER: METHODS OF GENE ALTERNATIONS

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ABSTRACT:

Each living thing has its own traits that it passes on to its offspring. These traits are determined by many genes found on the chromosomes. During meiosis, the chromosomes move into the gametes as a complete set. All the genes on a specific chromosome will be grouped together and passed on from one generation to the next. The fact that genes on the same chromosome tend to stick together when passed down through generations is called linkage. Genes that are on the same chromosome are known as linked genes. Bateson and Punnet found a way that things in plants can be connected to each other, back in 1906. They discovered this in the sweat pea plant called Lathyrus odoratus. However, the idea of linkage was introduced by Thomas Hunt Morgan in 1910 after he conducted an experiment on Drosophila melanogaster. Morgan and Castle created a theory that explains how chromosomes are linked together. Genes are lined up in a straight line in the chromosomes. Genes that show linkage are found together on the same chromosome. Genes usually stay together with the genes from the parents, except when crossing over happens. The distance between genes on a chromosome decides how closely they are connected. Genes that are close together are more strongly linked because they are less likely to mix with other genes during crossing over. A linkage group is a group of genes that are all located on the same chromosome. Because genes on a specific chromosome have their alleles on the matching chromosome, they are called genes on homologous chromosomes. Therefore, the number of linkage groups is the same as the number of single sets of chromosomes in a species.

KEYWORDS:

Chromosomes, Crossing Over, Genes, Genetic, Linkage.

INTRODUCTION

Linkage and Crossing Over are two similar but distinct events that happen in complex organisms with a nucleus. Genetic linkage means that genes like to stick together on a chromosome. Crossing over is when genetic information is swapped between cells that make eggs or sperm. Both of these are important in passing traits from parents to children. Both events have some connection to each other. However, linkage is when genes in a chromosome often pass down together. During crossing over, the genes in an organism separate and move into different reproductive cells called gametes. Genetic linkage is when certain genes tend to be inherited together because they are located close to each other on a chromosome. Genetic Linkage means that genes or DNA sequences on a chromosome often get passed down together during the process of sexual reproduction. Linked genes are genes that are found on the same chromosome. For instance, genes that determine the color of hair and eyes. That's why some people get the same hair and eye colors from their parents.

People can have black hair and brown eyes or brown hair and blue eyes if they get these traits from their parents. Crossing over means when traits from one parent are exchanged with

traits from the other parent during reproduction. Crossing over is when chromosomes exchange parts with each other to create gametes [1], [2].

Crossing Over is when the genes from the mother and father mix together and are carried in the gametes. It shuffles the alleles on the chromosomes. In easier words, it is when genes are exchanged in the reproductive cells. During meiosis, which is the process of creating egg and sperm cells, pairs of chromosomes line up. This allows similar pieces of DNA from the paired chromosomes to come together. This thing explains why there are differences in genes in organisms that reproduce sexually. It is also very important for how chromosomes are separated correctly. Linkage and crossing over are two different processes that happen during genetic inheritance. Linkage refers to the tendency of certain genes to be inherited together because they are located close to each other on a chromosome. When genes are linked, they are less likely to be separated and recombined during the process of genetic recombination. On the other hand, crossing over is a specific event that can occur during genetic recombination. During crossing over, corresponding chromosomes from a pair exchange segments of their genetic material. This can result in the mixing and shuffling of alleles between the chromosomes, leading to genetic diversity in offspring. In simpler terms, linkage means genes being inherited together because they are nearby on a chromosome, while crossing over is when chromosomes exchange parts of their genetic material [3], [4].

Crossing over is when genes are separated and mixed to create different gametes. Linkage means that genes that are passed down together are found on the same chromosome. Linkage is when two genes are near each other on the same chromosome. On the other hand, Crossing Over happens when two genes are positioned far away from each other on the same chromosome. Crossing over can interfere with the gene combinations created by linkage. Crossing over only happens during a specific stage of cell division called meiosis I. It results in new combinations of genes, unlike linkage where genes stay together. Linkage and Crossing Over are two things that happen in living things with cells that have a nucleus. They are similar, but also have some differences. Genetic Linkage means that genes like to stick together on a chromosome, while Crossing Over is when genetic information is exchanged during reproduction. Both of these things are important for passing on traits from parents to offspring. Both events have some connection to each other. However, linkage is when genes in a chromosome tend to be inherited together. During Crossing Over, the genes split up and go into different reproductive cells [5], [6].

DISCUSSION

Linkage is a concept that refers to how certain traits and characteristics are passed down from parents to their offspring over several generations, like F1, F2, F3, and so on. Without any changes, they are called linked characters and this is called linkage. This is different from Mendel's principle of independent assortment. Mendel's law of independent assortment applies to genes that are on different chromosomes. When genes for different traits are on the same chromosome, they are connected and are called linked (Figure 1). The traits are passed down together to the children and will not be separated or mixed up. The fact that two or more genes on the same chromosome tend to stay together during inheritance is called linkage. Bateson and Punnet (1906) studied sweet peas and noticed that the color of the flowers and the shape of the pollen usually go together and don't separate like Mendel's law of independent assortment suggests. Morgan said that there are two things related to linkage called coupling and repulsion when he was studying Drosophila. He said that linkage is when genes that are on the same chromosome usually stay together and move together when they are passed on. Genes on the same chromosome that are inherited together are called linked genes, and the traits controlled by them are called linked traits. The likelihood of their genes

mixing together is always less than 50%. All the genes that are on the same chromosome are part of one group. The number of chromosome pairs in an organism is equal to the total number of linkage groups. For instance, men have 23 linkage groups, sweet peas have 7, and Drosophila melanogaster have The Chromosome Theory of Linkage was created by Morgan and Castle. Genes that are linked are found on the same chromosome.

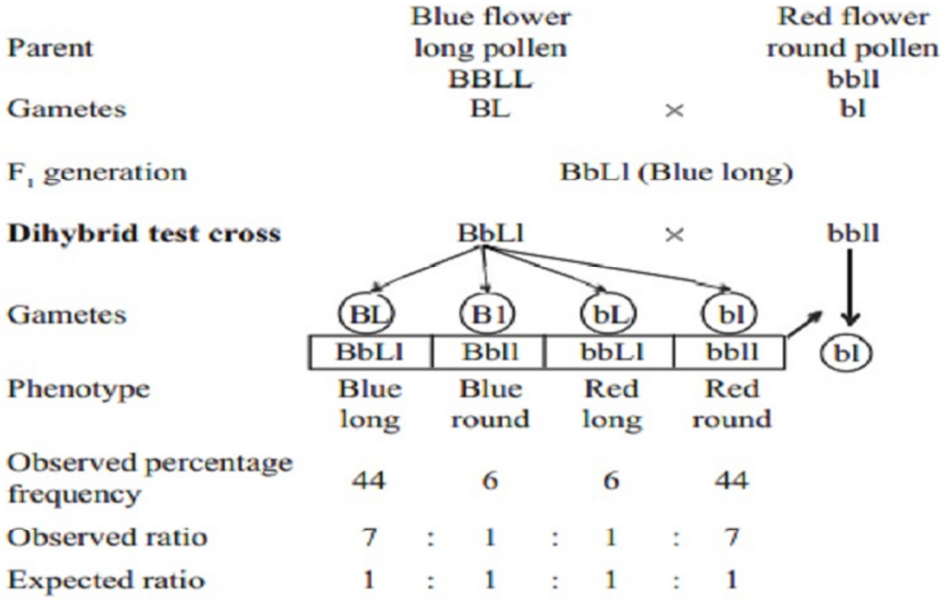

Figure 1: Representing the overview about the mechanism of Linkage [Brain Kart.Com].

Genes are organized in a straight line on the chromosome, meaning they are linked in a linear way. The distance between linked genes is smaller when the strength of linkage is higher. Genes that are located close together show strong linkage, while genes that are far apart have a higher chance of getting separated through crossing over (weak linkage). The genes that are connected have two different ways of being positioned on the chromosome. When the strong forms of multiple genes are on one chromosome and the weak forms are on the other chromosome, it is called cis-arrangement. However, if one gene has the dominant trait and the other gene has the recessive trait on one chromosome, while the other chromosome has the recessive trait for the first gene and the dominant trait for the second gene, then this is called a trans arrangement Linkage in maize (Hutchinson's test cross) Examples of Linkage:In MAIZE. Maize shows how characteristics are connected together. Hutchinson mixed two different types of corn. One type had colorful and full seeds, while the other type had colorless and small seeds. The gene for color (C) is stronger than the gene for no color. The gene for full seeds (S) is stronger than the gene for shrunken seeds (s). All the plants from the first generation produced seeds that were colored and complete. But during a test, when female plants that have a mix of traits are pollinated with pollen from a plant that has no color and smaller seeds, four different types of seeds are made [7], [8].

Based on the result mentioned above, it is evident that there are more variations of parents (96. 4%) compared to the new combination (3. 6%) This means that the parents are connected. The genes are on the same chromosome and only in 3. 6% of people, these genes are separated through crossing over. This is an example of a situation where things are not connected properly. 42 Coupling and Repulsion hypothesis: When two types of sweet pea plants were bred together - one with red flowers and round pollen, and the other with blue

flowers and long pollen - the resulting plants in the first generation (F1) had blue flowers and long pollen. This shows that the blue and long traits were stronger than the red and round traits. When the blue long hybrids were crossed with red and round individuals, they didn't have the expected ratio in the next generation. These created four groups of objects in different proportions. The groups consisted of blue long objects, blue round objects, red long objects, and red round objects. The ratio of blue long objects to blue round objects to red long objects to red round objects was 7 to 1 to 1 to 7. The test results show that there are seven times more combinations of traits from the parents (blue, long and red, round) than combinations that don't come from the parents. Bateson and Punnet said that when genes from one parent (like B and L) are together (BBLL × bbll), they are more likely to go into the same reproductive cell and be passed down together (coupling). In the same way, the genes (B and 1) from two different parents (like BBLL x bbll) usually go into different reproductive cells and get passed on separately and independently [9], [10].

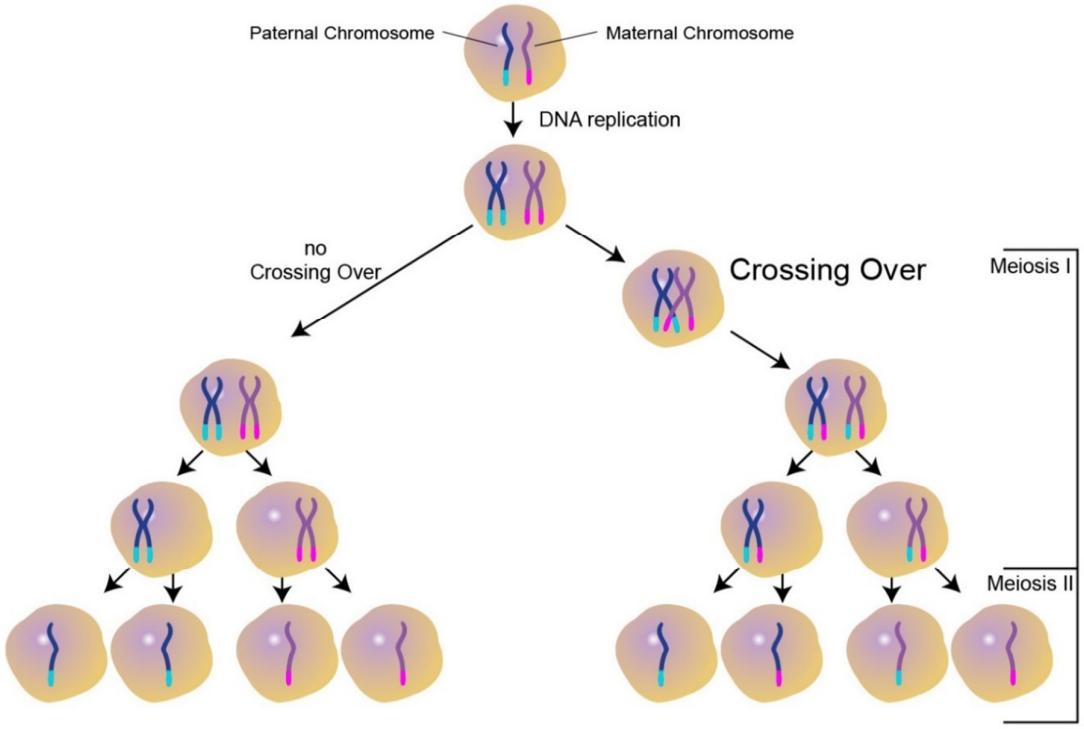

Figure 2: Representing the overview about the crossing over mechanisms [Qeios].

There are two types of linkage complete linkage and incomplete linkage. Complete linkage happens when certain traits are always inherited together and are found in multiple generations in their original combinations. Incomplete linkage occurs when new combinations of traits are sometimes present in offspring. These genes do not create new combinations. Genes that are closely located in the same chromosome are completely linked. The genes that determine a grey body and long wings in male Drosophila are always found together and never separate. Incomplete linkage refers to genes that create a certain amount of combinations that do not match those of the parent generation. These genes are far away on the chromosome. This happens when parts of chromosomes accidentally break during crossing over. The importance of linkage is that it helps determine the extent and type of breeding and selection programs. Linkage prevents genes from separating and keeps the traits of the parents together. So, it helps living things to keep their traits from their parents, their

ethnic group, and other important qualities. Because of this, it is hard for plant and animal breeders to mix different traits together.

Crossing Over

The process of crossing over happens when linked genes separate during meiosis, although this doesn't happen often. Morgan called the process of genes swapping parts between chromosomes crossing over. Crossing over is when parts of chromosomes swap between similar chromosomes during a specific stage of cell division called Meiosis Prophase I. This swapping allows for the combination of genes. Crossing over is when segments swap between the inner chromatids of similar chromosomes. Crossing over is when matching segments are swapped between similar chromosomes. This process helps genes that are linked together recombine. This happens during diplotene after the matching chromosomes have gone through the four-strand stage. This creates a cross-shaped figure where the chromatid segments exchange. Crossing over can happen at multiple points within one tetrad, creating several chiasmata (Figure 2). The number of chiasmata depends on how long the chromosomes are. We should understand that chiasma is the outcome, not the reason for crossing over. Many scientists have studied the formation of chiasma, but we still do not fully understand all the details of this process. The reason for the chromatids breaking and joining together is not known. According to recent research, chromosomes can break and come back together, and this could happen because of enzymes in our bodies. The enzyme endonuclease causes something to break apart, and the enzyme ligase is responsible for putting it back together.

Mechanism of Crossing Over

Classical Theory

This idea was suggested and developed by Morgan and Sharp in 1934. Commonly referred to as the two-plane theory, it proposes that nearby loops would be situated in two distinct planes that are oriented at right angles to each other. According to this theory, chiasma formation happens when two parts of a chromosome connect with each other. Normally, the two chromosomes in a bivalent stay connected to each other through their sister chromatids during synapsis. But in many parts of the cell, the sister chromatids move away from each other and connect with other chromatids that are not related, creating something called chiasma. During diplotene, when the matching chromosomes start to separate, the parts involved in chiasma formation feel physical tension or strain. This happens because of the separation process that happens during equational separation and reductional separation. This might cause the two chromatids to break. If the broken segments of the chromatids come back together, it could result in crossing over or recombination of genes that are connected.

According to the classical theory, a chiasma is created when non-sister chromatids from similar chromosomes join together during synapsis. Chiasma formation causes crossing over, it is not the result of it. Each chiasma does not cause recombination or crossing over. The results of the experiments we have do not support this idea, and it only matters in our understanding of history. This idea, on the other hand, is no longer accepted. The prevalence of obesity has been increasing globally, with a significant rise in both adults and children. This is a pressing issue as obesity is associated with various health problems, such as heart disease, diabetes, and high blood pressure. Efforts are being made to address this problem through promoting healthier lifestyles, encouraging physical activity, and providing access to nutritious food options. It is important for individuals to be aware of the potential risks of obesity and take steps to prevent and manage it. The theory of Chiasma Type was first suggested by Janssens in 1909 and further developed by him in 1924. Additionally, Belling

and Darlington have fully developed it. This theory is also called the one plane theory because it suggests that when there is a chiasma, the chromatids separate and reduce on both sides. According to this idea, when parts of different chromosomes break and then come back together, it's called crossing over. When the chromosomes that are similar begin to move away from each other during diplotene, chiasmata are formed at the places where crossing over has happened. So, a chiasma is what happens when two things cross over each other.

Sister chromatids are connected to each other in the bivalent, while non-sister chromatids come together to create chiasmata. Each chiasma is a result of crossing over. As a result, there should be an equal number of chiasma and crossing over events. Most of the evidence we have supports the chiasma type theory. Beadle, Brown, and Zohary have been strong supporters of this theory. They have found that there is a direct relationship between chiasmata and genetic crossing over, which helps explain the structure of gene maps and frequencies. However, Kaufmann (1934) and Cooper (1949) disagree with this theory because chiasmata are also created in certain male Drosophila tissues. Because male Drosophila do not have genetic crossing over, the chiasmata cannot be explained using chiasma type theory. Therefore, it seems like everyone agrees that chiasma type theory is correct. Copy-Choice Theory is an idea put forth by J. According to the Ledeberg hypothesis in 1955, the copying and combining of genetic material happen at the same time. Put simply, the chromatids copy themselves by creating new genes. Then, new connections between these genes are created. So, the changes in genes happen because new genes are made. There are two main issues with this. The first issue is that only two out of the four chromatids are involved in crossing over. So, two original strands stay the same and two new strands change during recombination. The second theory says that copying of DNA should happen during the later part of a specific stage in cell division, but new evidence shows that DNA replication happens even before that stage.

Types of crossing over: single crossing over and double crossing over.

There are different types of crossing over called single and multiple. The type depends on how many chiasmata are in the chromosomes. Single crossing over means that there is only one point in a pair of chromosomes where they exchange genetic material. The gametes produced by crossing over are called single cross-over gametes. Sometimes, crossing over can happen at two places on the same pair of chromosomes. This is called double crossing over.

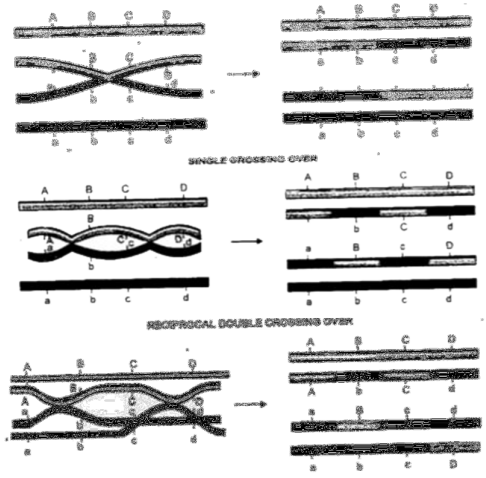

Figure 3: Representing the various types of crossing over [Doubtnut].

The special gametes made when crossing over happens are called double cross-overs. It happens less often than single crossing over (Figure 3). Multiple crossing over refers to when there are three, four, or more points where genetic material is exchanged between chromosomes. It is also called triple crossing over, quadruple crossing over, or multiple crossing over.

Various factors can influence how often crossing over occurs between two points. These factors can be related to genetics, the body's processes, or the environment. Some factors encourage more crossing over, while others decrease the chance of it happening. Please rewrite the text so that it is easier to understand. Sex is the physical activity between two individuals. Mutation is a process in which the genetic material of an organism changes. X radiation is a type of energy that is produced by a machine called an X-ray machine. The area near the centromere is very important in genetics for crossing over. Crossing over, a common occurrence, shows proof that genes are arranged in a straight line. Building chromosome maps and tracking linkage groups have been made easier thanks to information gathered from studying crossing over. It makes more changes happen more often, which is important for evolution. It creates many different combinations that can be chosen by nature to survive and reproduce. Understanding the way genes are organized and arranged helps us to learn more about their characteristics and how they work.

Three Point Test Cross Chromosome Maps

The chromosome maps show a picture of how closely genes are located on a chromosome. It uses a percentage to show the likelihood of genes being swapped during reproduction, and it focuses on one group of chromosomes. From the examples we studied about how traits are inherited, we can say that sometimes genes are linked together and don't follow Mendel's second law. The characteristics of an organism are determined by genes found in the chromosomes. Additionally, scientists believe that genes can be grouped together in different clusters called linkage groups. The linkage groups represent the same number as the chromosomes. The Drosophila melanogaster fly has four sets of chromosomes, and these are divided into four groups called linkage groups. Morgan's idea of crossing over assumes that the genes are arranged in a straight line on the chromosome. It was also believed that the distance between the two genes on the chromosome is connected to the amount of crossing over seen in two corresponding alleles. The more distant the alleles are on the chromosomes, the higher the percentage of crossing over. These two facts can be shown on a map where the genes are shown in a straight line along the chromosome. The percentage of crossover between two genes increases when they are farther apart. Therefore, a chromosome map is a line that represents different groups of genes. These maps are sometimes called cross-over maps because they show the amount of crossing over between genes. We can figure out the percentage of crossing over by doing test crosses. When mapping genes, a unit of measurement called distance is used. This is a small part called a Map unit or Morgan, which represents one percent of crossing over.

The exchange of genes between genes that are located close to each other can be very small, sometimes only 1/10 of 1%, or it can be as high as 50%, depending on the types of genes. In 1911, Sturtevant and Bridges created the first two maps of chromosomes. In 1920, Morgan and his team spent a lot of time studying Drosophila and made maps of its chromosomes. Then these maps were created using corn, baby chickens, tomatoes, etc. Finding the location of genes on a chromosome map involves determining how often a gene "crosses over" with another gene. We can explain how to make a chromosome map by using a three point test cross. A three point test cross is a breeding experiment where the offspring from a cross involving three genes that are close to each other on a chromosome are bred back with

individuals that have all three genes in a recessive form. In Drosophila, there are three genes called scute, echinus, and crossveinless that are linked to sex. Scute is a condition where some hairs on the body are missing, echinus means rough eyes and crossveinless means there are no cross veins on the wings. These conditions are caused by recessive mutations. When females with these mutations mate with wild type males, their offspring, called F1 females, look like the wild type. This happens because females get one sex chromosome from their mother. When the F1 females are bred with triple recessive males, there are eight different results.

When creating the chromosome map, the distance between two genes that are connected is measured by the frequency of crossing over, which is shown as a percentage. Since scute and echinus have a cross-over frequency of 7. 6%, it means that these two genes are 7. 6% distance away from each other. The cross-over frequency between echinus and crossveinless is 10. 1%, which means these two genes are 10. 1 units apart To figure out the order of the three genes, we need to know how often scute and crossveinless genes swap places. The scute and crossveinless genes, along with the normal genetic traits, were passed down to the offspring by one parent. However, in 352 cases, only one of these genes was present in the offspring, without the other. We can also include the two double cross-overs in these numbers. So, there were 354 cross-overs between scute and crossveinless, which is equal to 17. 7% of the total. This is the total and not the distance between 7. 6% and 101 So, the gene "crossveinless" is located after the gene "echinus". This means that the sequence is sc, ec, and cv.

CONCLUSION

During the creation of sex cells, genes can be mixed and matched independently. This is an important process in genetics. There are times when certain genes do not sort independently. These genes are inherited together as a group because they are on the same chromosome. Linked genes are genes that are found on the same chromosome and are passed down together. These genes are part of a group called a linkage group. Linked genes cannot freely separate and distribute independently during meiosis because it is the chromosome, not the gene, that is passed down. How can we figure out which genes on a chromosome are connected. We use test crosses to find out. This information about linked genes and linkage groups is used to make a map showing how chromosomes are connected. The data gathered from genetic mapping is valuable in various areas of genetic research. For instance, chromosome maps can show us if specific genes that create a certain trait are found together on the same chromosome. This information can help us understand how genes work in an organism, the DNA sequences related to specific genes, and how it can be applied to DNA research and technology. In some cases, genes that are connected to each other might not be passed on together as expected. This happens because when similar chromosomes pair up during the first step of cell division, they swap segments with each other. This process is called crossing-over. Crossingover is what causes genes that are connected to not be passed together, and it creates new combinations of genes. In this unit, you will learn about the idea of linkage, the proof that shows parts of chromosomes can swap places which leads to recombination, and how to create a map of a chromosome's genes.

REFERENCES:

[1] J. Stapley, P. G. D. Feulner, S. E. Johnston, A. W. Santure, and C. M. Smadja, “Recombination: The good, the bad and the variable,” *Philos. Trans. R. Soc. B Biol. Sci.*, 2017, doi: 10.1098/rstb.2017.0279.

[2] V. Plagnol, B. Padhukasahasram, J. D. Wall, P. Marjoram, and M. Nordborg, "Relative influences of crossing over and gene conversion on the pattern of linkage disequilibrium in Arabidopsis thaliana," *Genetics*, 2006, doi: 10.1534/genetics.104.040311.

[3] C. H. Langley, B. P. Lazzaro, W. Phillips, E. Heikkinen, and J. M. Braverman, "Linkage disequilibria and the site frequency spectra in the su(s) and su(wa) regions of the Drosophila melanogaster X chromosome," *Genetics*, 2000, doi: 10.1093/genetics/156.4.1837.

[4] C. J. Bidau and D. A. Martí, "110 Years of orthopteran cytogenetics, the chromosomal evolutionary viewpoint, and Michael White's signal contributions to the field," *Journal of Orthoptera Research*. 2010. doi: 10.1665/034.019.0202.

[5] S. W. Baxter *et al.*, "Linkage mapping and comparative genomics using next-generation rad sequencing of a non-model organism," *PLoS One*, 2011, doi: 10.1371/journal.pone.0019315.

[6] J. L. Lissemore, "My Favorite Sites: Linkage of genetics and ethics: more crossing over is needed," *Biol. Cell*, 2005, doi: 10.1042/bc20040130.

[7] L. K. Anderson *et al.*, "High-Resolution Crossover Maps for Each Bivalent of Zea mays Using Recombination Nodules," *Genetics*, 2003, doi: 10.1093/genetics/165.2.849.

[8] D. Rubinsztein, "Understanding Genetics: A Molecular Approach," *J. Med. Genet.*, 1994, doi: 10.1136/jmg.31.11.902.

[9] X. Sun and L. Liang, "A genetic linkage map of common carp (Cyprinus carpio L.) and mapping of a locus associated with cold tolerance," *Aquaculture*, 2004, doi: 10.1016/S0044-8486(03)00445-9.

[10] R. J. Kulathinal, S. M. Bennett, C. L. Fitzpatrick, and M. A. F. Noor, "Fine-scale mapping of recombination rate in Drosophila refines its correlation to diversity and divergence," *Proc. Natl. Acad. Sci. U. S. A.*, 2008, doi: 10.1073/pnas.0801848105.

CHAPTER 6

CHROMOSOMAL ABERRATION: MECHANISM OF GENETIC INFORMATION EXCHANGE

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ABSTRACT:

Chromosomal problems can be grouped into two types: those that involve the number of chromosomes and those that involve the structure of chromosomes. Numerical errors in chromosomes are called aneuploidies. The most common types of aneuploidy are when there is a missing chromosome or an extra one. For example, monosomies happen when there is only one chromosome instead of a pair, and trisomies happen when there are three chromosomes instead of a pair. There are four main types of problems that can happen in chromosomes. These problems can be called deletion, duplication, inversion, and translocation. Deletions happen when a part of the chromosome is removed, which can make the chromosome work less well. For instance, if a piece of the short arm in chromosome 5 is missing, it can cause Cri-du-chat syndrome. Babies with this syndrome usually have a smaller head size and cry with a high-pitched sound. In duplication, a section of the chromosome is copied, which leads to extra genetic material. This happens in Charcot-Marie-Tooth disease type I, where there is a copy of a section of chromosome 17. This causes weakness in the muscles. Inversion of a chromosome occurs when the genetic material is turned around or flipped in the opposite way. Inversions rarely cause illness and usually impact chromosome 2 the most. Translocations happen when a part of one chromosome breaks off and sticks to another chromosome. A Robertosonian translocation happens when two chromosomes with unbalanced arms lose their short arms and join their long arms together. Robertsonian translocations can sometimes lead to trisomies.

KEYWORDS:

Crossing Over, Cell Divisions, Chromosomes, Gene, Genetic Material.

INTRODUCTION

Chromosomal abnormalities can be divided into two main groups those related to the number of chromosomes and those related to the structure of chromosomes. There are more numerical disorders than structural disorders. Numerical disorders happen when there is a change from the normal number of chromosomes in a species. This category can be split into two groups. One group has individual chromosomes that are either missing or duplicated, and the other group has whole sets of chromosomes that are added or lost. On the other hand, structural abnormalities involve the rearrangement of one or more chromosomes. Most structural problems come from chromosomes not being exchanged properly or when two chromosomes are repaired incorrectly. Some examples of this issue are when parts of chromosomes are missing or moved, duplicated, or when chromosomes form into a ring shape or have two identical sides [1], [2].

Chromosomal problems can be grouped as either inherited or acquired. Constitutional chromosomal abnormalities can occur when eggs or embryos are forming and can affect a large or small part of an organism's cells. These abnormalities are estimated to happen in

20% to 50% of human conceptions and there have been over a thousand different abnormalities documented in babies that survive birth. When a person becomes an adult, they can develop abnormalities in their chromosomes. These abnormalities only affect a specific group of cells in the body and are not found in other cells. These changes are involved in the development of many tumors, but this review will not focus on that. As we mentioned before, the normal human cell usually has 46 chromosomes. However, some types of cells like red blood cells, platelets, eggs, and sperm have only 23 chromosomes. Sometimes, there can be abnormalities in the number of chromosomes, either in multiples of 23 or in other cases. Aneuploidy means having an abnormal number of chromosomes, with either too many or too few chromosomes. It is a common condition in humans, affecting 5 to 10% of pregnancies. It is a major cause of miscarriage and birth defects. Most cases of aneuploidy are not survivable, but there are a few rare syndromes that can be viable. In addition, it is not uncommon for human embryos to have more than two sets of all chromosomes, which is called polyploidy. For instance, about 3% of all human conceptions may have three sets of chromosomes [3].

However, all types of having too many sets of chromosomes monosomy, triploidy, tetraploidy, pentaploidy, hexaploidy, and so on are not suitable for humans. Usually, an aneuploid chromosome set is a little different from the normal set, and this difference is often just one extra or missing chromosome. Trisomy is when a person has three copies of a specific chromosome, which is the most common genetic abnormality in humans. However, most trisomies are lethal and result in miscarriages. Monosomy, which is when a person has only one copy of a chromosome, is very rare in humans and usually only occurs with chromosomes 21 or X. These findings suggest that the effects of having extra sets of chromosomes may result from an imbalance of important genes found on small chromosomes with few genes. It's important to understand that abnormalities in sex chromosomes have different effects than abnormalities in other chromosomes. For example, having an extra or missing sex chromosome is more common and usually has less severe effects than having an extra or missing autosome [4], [5].

When it comes to issues with the structure of chromosomes, there is one big problem called Robertsonian translocation that is very important to understand. In this situation, two chromosomes with a certain shape break in the middle, losing their short parts and creating a new chromosome with a different structure. This new chromosome has one middle part and the longer parts of the original chromosomes. Some people might have a specific genetic mutation but still look normal because there is only a small amount of genetic material on certain parts of their chromosomes. Therefore, the two arms that are still there can make up for the missing arm. But sometimes, it can cause problems with genes and make babies with extra or missing chromosomes, which can have serious effects on their appearance and abilities, like Down syndrome. A Robertsonian translocation is a type of genetic mutation. The estimated number of people in the general population who have this mutation is 1 out of 1000 [4], [6].

DISCUSSION

Chromosomal Aberration, is a certain kind of issue found in the chromosomes. Chromosomes have many genes on them. The genes are organized in a straight line. The genes on a chromosome are always in the same order and location. When a chromosome breaks apart, the pieces can come back together, sometimes leaving parts that don't have a center. These parts without a center eventually go away. The fixed parts will make a chromosome that is missing certain genes, especially the ones from the missing section. This means that if a certain part of a chromosome is lost, it can result in the loss of one gene or a group of genes.

When a section of genetic material is lost from a chromosome, it is called deficiency or deletion. There are a few ways that deletions can happen. One way is when parts of the chromosomes are lost. Another way is when parts of the chromosomes are removed from the middle. This is shown in the diagram. In simpler terms, deletions can happen when parts of a chromosome are lost. This loss can occur at the ends or in the middle of the chromosome. At a molecular level, deletions can be so small that they result in missing nucleotides in the DNA molecule, which can cause mutations. If there is a big lack, the cell might die. But if the lack is small, the cells can survive. If a fertilized egg is like this, it will produce something that is very harmful and can cause death. When both copies of a certain gene are missing, it usually causes death, but when only one copy is missing, it is typically seen as a normal change in the gene.

Deficiency can be identified by two main characteristics: genetic effects and cytological effects. These primarily happen because of the loss of genetic information, and secondarily because of changes in the genetic composition and balance. Problems have been helpful in figuring out the exact places of genes on the chromosomes. One of the genetic consequences of not having enough is called pseudo-dominance. Pseudodominance happens when a chromosome with a missing dominant gene pairs up with a normal chromosome that has a recessive gene instead. If there are no strong alleles, then the weaker alleles would show up in the way that strong ones normally would. This is known as pseudo-dominance. If certain genes are harmful when both copies are present, they can still be harmful when only one copy is present. When a part of the chromosomes is missing, it cannot go back to the way it was before. We can show that there are big missing parts in the chromosomes of eukaryotes by looking at two things When cells divide, we can see that there are pieces of chromosomes that are missing or not in the right place. During a specific phase of cell division, we can see that certain parts of the chromosomes are not joined together as they should be. If the cell has one copy of a gene deleted, i. eit is not the same on both copies. In simpler words: It has a regular chromosome and a chromosome that is missing some parts. When they come together during synapsis, the genes match up perfectly in the regular parts, but not in the missing parts. The regular chromosome doesn't pair up at all in the missing parts. So, a bend will happen in the normal homologue where there's a lack of space in between. That's why it's called the buckling effect [7], [8].

This setup is called a deficiency loop or compensation loop and can be seen using a microscope. Chromosomal Abnormailty: The second type. Duplications are when genes in a specific part of a chromosome in both simple and complex organisms double. In simpler terms, when there is an extra part or copied gene sequence added to a chromosome, it is called duplication. If part of one chromosome is added to another similar chromosome, it's called intrachromosomal duplication. If a segment of a chromosome is duplicated and stays in the same chromosome, it's called intrachromosomal duplication. However, if the duplicated segment ends up in a different chromosome or exists as a small piece in the set of chromosomes, it's called interchromosomal duplication. The duplicate parts on a chromosome can be in different arrangements depending on where they are and how the genes are duplicated. In direct tandem duplication, a gene is copied and placed right next to the original gene on the chromosome. The order of genes in the duplicated segment is the same as the order in the normal section of the chromosome. The gene sequences in the normal and duplicated chromosomes show that there is a reverse tandem duplication. This means that the duplicated section with the opposite gene sequence is right next to the normal sequence.

Displaced direct duplication means that the duplicated part is not right next to the original part. Instead, there are other parts in between them. The same genes can be duplicated in either the same arm of a chromosome or in different arms. In a Displaced Reverse duplication, the duplicated genes are separated from the normal genes by another section of genes. In a Transposed duplication, the duplicated genes are attached to a different position on another chromosome. The size of the copied part can be very different. The smallest duplicates that can be looked at under a microscope are single bands on a type of chromosome called polytene chromosomes. However, all parts of the chromosomes may be copied, resulting in the creation of an isochromosome. When all the groups of connections or chromosomes in a single set of chromosomes are copied, it is called genome mutation. Duplication can happen in a few different ways. A very common way is unequal crossing over, where one chromosome gets extra genes and another is missing some genes. This is the main change in the structure of a chromosome. The diagram in Figure shows how gene duplication can happen on a chromosome when there is an unbalanced exchange of genetic material during crossing over [9], [10].

Duplications can happen when certain parts of genes swap places or are flipped in opposite directions. Mutations in the structure of chromosomes can be divided into two types: duplications and deficiencies. Generally, duplications are more likely to survive and be successful compared to deficiencies. A heterozygous duplication looks like a deletion. Sometimes, duplications can be seen as strong changes in genes. By copying a gene variant, it can be copied once, twice, or multiple times. Therefore, duplications can be used to study the impact of different amounts of certain gene variants. One famous example of duplication that had a big effect on genie theory is the Bar-eye mutation in Drosophila. The researchers found that in this case, extra parts of a chromosome were attached to a normal X-chromosome and it duplicated and tripled a specific section of the chromosome. The Bar-eye is a genetic mutation that causes the eyes of a fruit fly to become rod-shaped with fewer facets. It suddenly appeared in a group of fruit flies with normal round eyes. The Bar-eyed mutants bred with similar types created flies with normal eyes and flies with even smaller eyes at about the same rate.

These observations showed that something was not consistent about the Bar locus, but it was difficult to understand why there were the same amount of normal and double bar flies. Additionally, when female flies with the Bar mutation were bred with male flies without the mutation, their offspring showed two different physical characteristics. However, when male flies with the Bar mutation were bred with female flies without the mutation, their offspring did not show the same physical characteristics. Many factors that make copies are known in corn and peas. The duplications and deletions can be really tiny, almost at the molecular level. Changing the genie balance could cause strange changes in the body's appearance or features. Duplications can help create new genes by making copies that can change and do different things. Like not having enough of something, duplications have effects that can be observed through a microscope and have to do with genes. Cytological Effects: When eukaryotes have small copies of their chromosomes, it can be seen through changes in the way the chromosomes pair up during certain stages of cell division. This can happen during the first stage of meiotic cell division or when cells pair up in specific tissues like the salivary glands of insects in the Diptera group. Crossing over happening backwards in a duplication sequence can cause a chromosome to have two centromeres, which is called a dicentric chromosome [9], [11].

This can often be noticed in corn. When the two center parts of a chromosome move apart, a bridge is formed between them. This bridge can later break at any place. The pieces that are

broken are stuck together, and when they are copied, they can create two pieces that are joined because they have sticky ends. This means that the whole chromosome arm can be copied, creating isochromosomes. Isochromosomes are rare in the tissues of plants. Genetic effects of duplications depend on the information they contain and how they alter the balance of genes. In both homozygous and heterozygous states, these traits can either make the carriers stronger or weaker, and in severe cases, they can be deadly. Duplications are important for evolution because they provide more genetic material and change the genetic balance. This happens at both individual and population levels. In simple terms, sometimes there can be extra copies of genes because of changes over time. These extra copies can change in different ways without causing any problems for the organism. When a gene pairs affect the same trait, it can happen when genes duplicate. This can result in multiple factors or complementary factors affecting the trait. This means that when highly evolved organisms have repeated DNA sequences, it is a clear sign. The third type. Translocation: Chromosomes can break into pieces, and each piece may have an unprocessed end. These parts can come together again, and when they do, they can either fuse pieces from the same chromosome or pieces from different chromosomes. Ionizing radiations like X-rays and gamma-rays are often used to break chromosomes in order to create changes in their structure.

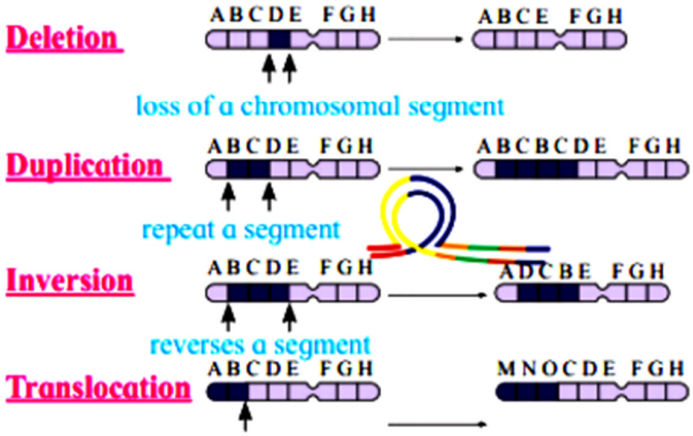

Figure 1: Representing the types of chromosomal Aberration [Sarthaks e Coneect].

In this process, a piece of a chromosome is moved to a different chromosome that is not related within the group of chromosomes. In simpler terms, a translocation is when a piece of one chromosome moves to a different chromosome. This can happen in two ways the piece moves from one chromosome to another, and pieces swap places between chromosomes that aren't a matching pair. When translocation happens, genes change position but the number of genes remains the same. Simple non-reciprocal translocations or transpositions occur when a piece of one chromosome is moved to a different chromosome that is not its pair. There are two types of translocation: one is called simple or terminal transposition, and the other is called shift or interstitial transposition. Simple Translocation: When a chromosome breaks into two parts because of stress from outside or inside the body, one of the broken segments may stick to the end of the closest chromosome, even if it's not a matching pair (Figure 1). This is a basic transfer of something. Let's say we have two sets of chromosomes, one set labeled A B C D E F G and the other set labeled T U V W X Y Z. If we take a part of the first chromosome (called F G) and move it to the second chromosome, a new chromosome named TUVWXYZFG would be created, as shown in the picture. This type of non-standard chromosome is called a translocation chromosome, and it's considered to be in a simple

translocation state. Usually, terminal transpositions are not common because the ends of unbroken chromosomes are not sticky.

In simpler terms, intrachromosomal shift translocation or interstitial transposition is when a segment inside a chromosome breaks and moves to another broken chromosome that is not a match. It is sometimes called intercalation or insertion. If the order of genes in the moved part of the chromosome is the same as in the original part of the chromosome, it's called encentric translocation. But if the order of gene segments in a translocated chromosome is flipped, it is called a dyscentric translocation. Two people were arguing loudly in the street. Reciprocal translocation happens when a break occurs in two chromosomes that are not a matching pair. The broken ends of the chromosomes can join together, with the second chromosome's broken end attaching to the first chromosome's broken end. Let's say we have 6 chromosomes named A, B, C, D, E, F, and 6 chromosomes named G, H, I, J, K, L. After the exchange of genetic material between chromosomes, two new chromosomes can be formed. These chromosomes are labeled A, B, C, J, K, L, G, H, I, D, E, and F, as seen in the picture.

The reciprocal translocation is the most common kind of translocation. If two breaks happen in two different chromosomes that are not related, there is a chance that the intercalary segments may switch places. However, this doesn't happen very often. Reciprocal translocation is a process similar to crossing over, but it involves swapping of segments between two chromosomes that are not the same. It is sometimes called "wrong crossing over". The reason why translocations happen can be explained by either the breakage-reunion model or the exchange model. Translocation can happen in different parts of a cell. It can occur in the chromosome or in a single part called chromatid. Translocation points are the places where translocations occur. The reciprocal translocation can be either imbalanced or balanced. The unequal movement of chromosomes results in two different types of chromosomes: one with two centromeres and one with no centromere. It is possible for a chromosome bridge to form when the two centromeres of a specific type of genetic mutation are separated during cell division. This occurs during a specific stage of cell division called anaphase. In symmetrical translocation, the resulting products are single-centered.

When chromosomes undergo homozygous translocations, they typically act like normal chromosomes but create new groups that are linked together. If they continue, they can cause new types of chromosomes in the group of organisms. When someone has a symmetrical reciprocal translocation, they have two chromosomes with the translocation and two normal chromosomes. These chromosomes share some similarity, but none of them are exactly the same. When gene pairs come together, it's called synapsis. This happens in homologous regions, which are spread out over four chromosomes. In a simple reciprocal translocation heterozygote, all four chromosomes will join together. This connection will cause a cross shape at a specific point in the cell division process called pachytene. It means that four chromosomes will come together - two normal ones and two that have changed places. Having different versions of a chromosome (heterozygosity) and having a rearranged chromosome (translocation) lowers the amount of genetic exchange that happens during reproduction. The crossing over can happen in any of the four parts that look like a cross when there is a mix of two different translocations.

However, the outcome will be different depending on where the crossing over happens in relation to the middle part and the points where the translocation happened. If the crossing over happens in the regions between the centromeres and break points, it will lead to chromosomes that have extra or missing parts, regardless of how they are distributed next to each other or in different patterns. This could be a big reason why translocation heterozygotes are unable to have children. But it seems like the crossing over is limited in the areas between

the chromosomes because they don't connect very well. If the crossing over happens outside the areas in between genes, it doesn't change how the new gene combinations are separated because one section of the chromosome is swapped for another. There is less crossing over in the translocated part, especially near where the exchange happens. This happens because the chromosomes don't match up correctly in the swapping area, or because they have trouble aligning during cell division. What happens next with this cross pattern depends on how often and where the chiasmata and the centromere are. If chiasma forms in all four parts where chromosomes pair up, it makes a ring of four chromosomes. Scientists have noticed that quadrilateral rings can be found in certain plants and animals during a specific stage called metaphase.

These plants include Datura, peas, wheat, and Tradescantia. If the process of chiasma formation doesn't happen properly in one of the segments where chromosomes pair up, a chain of four chromosomes is produced. If parts of chromosomes come together in pairs next to each other or in a pattern that alternates, there will be either a group of three chromosomes and one independent chromosome, or two pairs of chromosomes. Rings and chains have chromosomes that are either normal or changed, and they alternate throughout the sequence. Now, how the four chromosomes are arranged in a ring or chain shape during anaphase I of meiosis is decided by how the centromeres are positioned. There are two common ways things can be arranged: next to each other or in a repeating pattern Twenty-two point one four. In adjacent distribution, the chromosomes that are paired together are separated. One normal chromosome and one translocated chromosome go to one side, while their matching chromosomes go to the other side. In this situation, both of the products made by meiosis are copied. In adjacent distribution, there are two events. When the centromeres of nearby chromosomes that are not similar separate and go to the same side. When similar centromeres move to the same side, but this almost never happens. In alternate distribution, during a specific phase, two regular chromosomes go to one end and two translocated chromosomes go to the other end. This happens when the cells divide to create gametes, resulting in two types of gametes some with regular chromosomes and some with translocated chromosomes.

Translocations have two main genetic effects. Firstly, they can change the structure or grouping of chromosomes. Secondly, they can change the order of genes in chromosomes, which can cause abnormalities in the body's characteristics. This is the way that where something is placed can have an impact on its effect or outcome. Semi-sterility means having some level of infertility or being partly unable to reproduce. The translocation heterozygotes are usually partially unable to reproduce because they make reproductive cells that have extra or missing chromosomes due to the way the chromosomes pair up, exchange genetic material, and separate. Moving from one place to another is very important for both individuals and species. The biggest problem caused by a reciprocal translocation is that it can make a person partially unable to have children. It also changes how things normally grow and develop. In the evening primrose flower, there are different types of changes called translocations. This was the plant that had different characteristics, which inspired De Vries to develop his well-known mutation theory. Chromosomal Aberration: Fourth Type. Inversion is a type of abnormality in which a segment of a chromosome and its gene sequence are reversed compared to the usual arrangement in a chromosome or group of genes. It happens in a part of the chromosome called the intercalary segment. There is no proof that terminal segments of chromosomes can flip inwards. According to the breakage reunion model, when a chromosome breaks in two places, the middle part between the breaks (called inversion points) is flipped around, and then the three parts come back together at the break sites.

Inverted parts can sometimes get inverted again. This is called flipping the order. So, in the example mentioned earlier, if the section GFEDC in the upside-down chromosome goes through another inversion, the chromosome will go back to its original gene order ABCDEFGHIJ. There are different types of inversions. They are divided based on how many segments are flipped within the chromosome and where the flipping points are in relation to each other. Rewrite this text in simple words: 1. Please simplify the text below: The government has implemented a series of policies in order to improve economic growth rates and decrease unemployment. These measures aim to boost investment and productivity, increase consumer spending, and promote job creation in various sectors. The government believes that by implementing these policies, they will be able to stimulate the economy and provide opportunities for more individuals to find employment. Single inversions happen when a chromosome has one segment that is backwards. Single inversions are categorized based on whether or not the flipped part of the chromosome contains the centromere. Paracentric inversion means that a part of a chromosome breaks off and reattaches at a different location on the same chromosome, but in the opposite orientation. This is the most common kind of reversal that only occurs in one arm of a chromosome. In this case, the chromosome segment that is flipped does not have the centromere. It can also be referred to as an off-center or unbalanced or irregular or lopsided reverse.

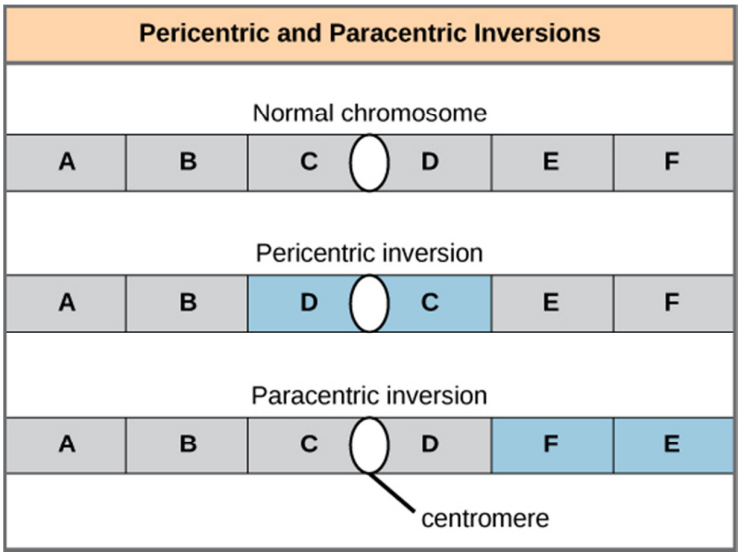

Figure 2: Representing the overview about the chromosomes inversions [Luman Learning].

This type of inversion can result in genetic abnormalities, as it can disrupt the normal functioning of genes located within the inverted segment. It can also be referred to as transcentric, eucentric, transkinetic, or symmetrical inversion. If the two points where the chromosome breaks and reforms in a certain way are the same distance from the center, the resulting chromosome will look similar to a normal one. However, if the places where the chromosomes break are not evenly spaced from the center, the center of the chromosome may move from one end to the middle or vice versa. This can cause a noticeable change in how the chromosome looks. This shows that pericentric inversions may have been important in the development of new chromosome arrangements. Complex inversions occur when there is more than one flipped section on a chromosome. The different types of inversions can be sorted into two categories: homozygous or heterozygous. When a person has homozygous inversions, it means their two copies of a chromosome have the same inversions. They behave normally during cell division processes called meiosis. Heterozygous inversions

occur when one chromosome has an inversion and the other chromosome does not have an inversion. In other words, one chromosome is normal while the other chromosome has a rearranged section. In individuals with inversion heterozygosity, important changes in cells and genes can be seen. Inversion heterozygotes are perfectly viable because there is no gain or loss of genetic material.

The way an inverted chromosome pairs with a regular or non-inverted chromosome depends on how long the inversion is and how the segments of the inverted and non-inverted chromosomes are arranged (Figure 2). If the upside-down part of the chromosome is big, it creates a loop in the normal matching chromosome. The upside-down chromosome then attaches to the normal chromosome in a way that the matching parts pair up. The place where the inverted segment is located can be identified by looking at the presence of a loop in the paired chromosomes during cell division. If the upside-down part is very small, it can't create a loop and it either stays alone or pairs with a different part of the normal chromosome. The loop's size depends on how big the inversion is. If the inversion is bigger, the loop will be bigger too. The crossing over and chiasma formation inside and outside an inversion loop lead to changes in the structure of the genetic material, such as duplication or deletion. These changes depend on the type of inversion (paracentric or pericentric), the number and location of chiasmata. These changes in structure lead to meiotic products with an uneven number of chromosomes.

When there is a single crossing over in a chromosome that has a change in its center (called a pericentric heterozygous inversion), it produces two normal cells and two abnormal cells. The abnormal cells either have extra copies or are missing certain genes. I'm sorry, but there is no text provided to rewrite. Can you please provide the text you would like me to simplify. In plants, if the gametes have too many or too few copies of their genetic material, they usually cannot survive. So, people with pericentric inversion of chromosomes have a condition where their ability to produce offspring is reduced, but more than 50% of them can still have children. In animals, when the cells responsible for reproduction have extra or missing chromosomes, they usually work fine. However, when these cells combine to form a fertilized egg, it often cannot develop and survive.

When a crossover happens in a specific area of a chromosome, it can cause some complicated effects. It makes one chromosome have two centromeres and one chromosome with no centromer. During a specific part of cell division called anaphase 1, the dicentric chromosome is pulled between two poles, while the acentric chromosome floats around randomly and eventually gets lost. The dicentric bridge can break anywhere, which can cause copies and missing parts in the meiotic products. Interestingly, in fruit flies and plant egg cells, the dicentric bridge can stay together for a long time even after anaphase I. So, the two daughter nuclei will either be joined together by a special bridge or they will have the pieces of the bridge if it breaks. The part linked to the bridge causes a problem and the size of the problem determines how much less fertile something is. In simpler words, if a gamete is missing genes because a part of its chromosome is lost, it will probably not be able to survive and reproduce. Usually, females who have one copy of a certain genetic change called paracentric inversion do not have problems with being able to have children. This is because during the process of making reproductive cells, the chromosomes are arranged in a certain way that helps get rid of any abnormal chromosomes that could cause infertility. When two crisscrosses happen in a loop, the outcome will rely on how many strands are involved. When two strands of DNA crossover, they form four chromatids.

Out of these four, two will have participated in the crossover process, while the other two will not have. We can only detect this condition if there are specific genetic markers in the area

where crossing over happens. Three-strand double crossing over results in the production of one chromatid without any crossing over, one chromatid with a crossing over, and two fragments that are not connected to a centromere. If there is a double cross-over with four strands of DNA, it would create two abnormal chromosomes with two centromeres and two smaller pieces without centromeres. If there are any missing or extra pieces of DNA, it would likely lead to the cells or reproductive cells not being able to survive or the death of a fertilized egg. So, it is clear that when parts of a chromosome are flipped, it can have a big impact on how the chromatids recover during crossing over. Another important impact of inversions is that they prevent the mixing and shuffling of genes on a chromosome through the process of crossing over. This helps to keep a particular section of the chromosome unchanged within a population.

When a genetic crossover happens in an inversion, the resulting genetic material usually doesn't pass on to the next generation. This is because the cells or embryos that carry the crossed over chromosomes are not able to develop properly in plants or animals. Having a recessive lethal gene within an inverted section can actually be helpful in keeping diversity in the structure of genes. This is because organisms carrying both the normal and lethal versions of the gene can survive, while those with two lethal versions cannot survive. In CIB stock of Drosophila, there is a special segment of the chromosome called C factor. It helps suppress cross-overs. This segment is inverted, meaning it is flipped around. This C factor also prevents the chromosome from being the same on both sides, and there is also the B gene that is responsible for bar eye. However, having two copies of the CIB chromosome is harmful and leads to death. In CIB, the C factor is located between two marker genes 1 and B. In 1928, Muller discovered a way to find harmful genes linked to the X chromosome in flies by studying how gene crossovers are affected by genetic changes caused by X-ray exposure. The genetic evidence of inversion can show two things.

It reduces the exchange of genetic material during reproduction, and It might cause mutations due to changes in gene positioning. As mentioned before, inversion and translocation do not cause any increase or decrease in the genes. They only move some genes, but the genes themselves do not change in these cases. When genes move around in chromosomes, it can have important effects. This is because when certain genes are grouped together, they are involved in the completion of specific steps in biochemical reactions that happen over and over again. When genes change, it can affect how they work. These changes are called "position effects". Inversion homozygotes can be identified using genetic and cytological methods. The first method is by studying the genetic linkage and observing any changes in the relationship between different genes. The second method is by examining the changes in the structure of chromosomes during a specific stage of cell division called mitotic metaphase. By looking at the differences in the patterns on chromosomes. Inversion heterozygotes can be found by observing the formation of an inversion loop during a specific stage of cell division called prophase I of meiosis. During anaphase I, the cell can sometimes form a structure called a dicentric chromosomal bridge. This occurs when two chromosomes attach to each other in the middle, creating a bridge-like shape. Additionally, there can be fragments of chromosomes that are missing a centromere, known as acentric fragments. Developing unusual meiotic products that can be identified using tetrad analysis. The ability to have babies is reduced because of the production of unbalanced genetic material during a specific process in reproduction called crossing over. Lowered frequency of genetic recombination.

CONCLUSION

A mare that is of breeding age and cannot have babies because of underdeveloped reproductive organs might have a genetic problem with their chromosomes. The most frequently observed problem with chromosomes in horses is called 63,X gonadal dysgenesis. This means that there is only one sex chromosome instead of the usual pair. Horses with gonadal dysgenesis who don't have a Y chromosome develop as girls in their physical characteristics. Horses that are affected have small ovaries on both sides, as well as a small and weak uterus, and underdeveloped endometrial glands. Many other issues with chromosomes have been found. A sure way to identify a chromosomal abnormality is by examining the chromosomes or their arrangement using a technique called karyotyping. There is no cure for this condition. Around half of early spontaneous abortions have abnormal karyotypes. The most common abnormalities include having an extra chromosome (trisomies), having multiple sets of chromosomes (polyploidy), and having only one X chromosome (monosomy X). Trisomy 16 makes up about one-third of all trisomies. Abnormal karyotypes, such as empty sacs, disordered embryo growth, or focal defects, happen in 60% to 80% of cases. Therefore, if someone discovers a strange-looking embryo or abnormal features of embryonic death in the placenta, they can likely expect that there is an abnormal genetic makeup. Some pregnancies with abnormal chromosomes still go on until the second and third trimesters, and we can identify how the baby looks even with these abnormalities. Macerated fetuses, even if they don't look deformed, often have genetic problems. Besides triploidy, it is usually not possible to determine the exact chromosomes present in a spontaneous abortion just by looking at the structure of the placenta. However, finding certain things like fetal red blood cells without a nucleus, issues with the umbilical cord, infarcts, minimal fluid buildup, and long-term inflammation has been linked to normal chromosomes.

REFERENCES:

[1] Q. Song, M. Peng, Y. Chu, and S. Huang, “Techniques for detecting chromosomal aberrations in Myelodysplastic syndromes,” *Oncotarget*. 2017. doi: 10.18632/oncotarget.17698.

[2] E. Schatorjé *et al.*, “Primary immunodeficiency associated with chromosomal aberration - An ESID survey,” *Orphanet J. Rare Dis.*, 2016, doi: 10.1186/s13023-016-0492-1.

[3] W. P. Kloosterman and R. Hochstenbach, “Deciphering the pathogenic consequences of chromosomal aberrations in human genetic disease,” *Molecular Cytogenetics*. 2014. doi: 10.1186/s13039-014-0100-9.

[4] T. D. Kim *et al.*, “Impact of additional chromosomal aberrations and BCR-ABL kinase domain mutations on the response to nilotinib in Philadelphia chromosome-positive chronic myeloid leukemia,” *Haematologica*, 2010, doi: 10.3324/haematol.2009.014712.

[5] S. Anand, B. Nath, and R. Saraswathy, “Diabetes-increased risk for cancers through chromosomal aberrations?,” *Asian Pacific J. Cancer Prev.*, 2014, doi: 10.7314/APJCP.2014.15.11.4571.

[6] L. Kroeldrup, L. A. Larsen, C. Fagerberg, J. M. Hertz, and K. Christensen, “Chromosomal aberrations in monozygotic and dizygotic twins versus singletons in

Denmark during 1968-2009," *Twin Research and Human Genetics*. 2017. doi: 10.1017/thg.2017.17.

[7] B. D. Gane, S. Nandhakumar, V. Bhat, and R. Rao, "Effect of therapeutic hypothermia on chromosomal aberration in perinatal asphyxia," *J. Pediatr. Neurosci.*, 2016, doi: 10.4103/1817-1745.181269.

[8] A. D. Bolzán and M. S. Bianchi, "Telomeres, interstitial telomeric repeat sequences, and chromosomal aberrations," *Mutation Research - Reviews in Mutation Research*. 2006. doi: 10.1016/j.mrrev.2005.12.003.

[9] A. K. Jain, D. Singh, K. Dubey, R. Maurya, and A. K. Pandey, "Chromosomal aberrations," in *Mutagenicity: Assays and Applications*, 2017. doi: 10.1016/B978-0-12-809252-1.00004-3.

[10] U. Ben-David *et al.*, "The landscape of chromosomal aberrations in breast cancer mouse models reveals driver-specific routes to tumorigenesis," *Nat. Commun.*, 2016, doi: 10.1038/ncomms12160.

[11] R. Lee *et al.*, "Chromosomal aberrations in lymphocytes of lung cancer patients treated with carbon ions," *J. Radiat. Res.*, 2004, doi: 10.1269/jrr.45.195.

CHAPTER 7

A COMPREHENSIVE OVERVIEW ON CONCEPTS OF POPULATION GENETICS

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ABSTRACT:

Population genetics chapter focus on simple scenarios of viability selection that do not consider the population size or genetic makeup. Unlike before, the textbooks now use "viabilities" to mean the same thing as fitnesses, so newer population geneticists associate fitness with viability. The limited range of discussion in population genetics lets scientists focus on keeping track of the effects of different genotypes on fitness, as well as the rules for mating and passing on genes. Another benefit of focusing on simplified life histories is that it allows us to examine fitness in a quick and specific way. One can think about models where fitness changes over time, in different places, or based on the gene frequencies or number of individuals in a population. Population genetics is about understanding how different traits are passed down within populations over time. This field has specific ways of studying these patterns and a language that scientists use to communicate about it. Population genetics has been used for studying organisms in several important ways. Firstly, it has helped us find and classify species that look very similar but are actually different genetically. Secondly, it has allowed us to measure and understand the amount of genetic diversity within species and how it is spread out in different areas. Thirdly, it has helped us study how genetic traits change over time. Fourthly, it has allowed us to analyze genetic information from past collections to learn more about the history of plant species. Lastly, it has provided us with long-term perspectives by studying the DNA sequences of species and comparing them with information about the ancient oceans.

KEYWORDS:

Drift, Genes, Genetics, Populations, Traits.

INTRODUCTION

Population genetics is a field of study within genetics that looks at how traits are passed on in entire populations, rather than just within families. Population genetics is a field of study that tries to explain how the genetic makeup of a population changes over time, which is a key part of Darwinian evolution. The genetic makeup of a population is determined by how often different versions of a gene or genetic marker appear in samples of that population. This can be seen through the physical traits or characteristics of organisms, like their color or patterns on their macromolecules when separated by electricity. The specific combinations of genes that individuals have can be figured out by looking at their physical traits. These genes are located at specific spots on a chromosome, which is called a locus. Genes are important for passing down traits, like making proteins or controlling how our bodies work. But sometimes parts of our genes don't really do anything, and scientists use these parts to identify different groups of people. Most of the zygotes in bigger organisms have two copies of each gene, which they get from their parents.

A diploid genotype is a combination of two sets of genes. If these genes are the same in how they look or where they come from, it is called homozygous. A genotype with two different alleles is called heterozygous. The amount of mixed genotypes in a population, which is called heterozygosity, is a significant way to measure how diverse the genetic makeup is. When a population has more than one variation of a particular trait or marker that is determined by genes, and these variations occur more often than what would be expected from random changes in genes, we call it polymorphic. On the other hand, a monomorphic or fixed genetic trait or marker is shown by only one physical characteristic in a group of individuals who all have the same gene [1], [2].

The four main things that cause genetic changes over time. Mutation happening again and again - Natural or human-made selection Genes moving between different groups within a larger population. Random changes that occur when studying a small number of organisms Without these forces, the genes in a population that mates randomly will remain the same over time. This is known as the Hardy-Weinberg principle. This basic principle states that the frequencies of different versions of a gene at a specific location in the DNA can be calculated using a mathematical formula. The formula is different depending on whether there are two versions of the gene or more than two versions. Let's say there is a group of living organisms. In this group, there are two different versions of a certain gene. These versions are called alleles. One version is called A and the other version is called a. Both versions have the same level of importance. In the group, the allele A is more common and the allele a is less common. Different possible combinations of genes produced by multiple alleles in organisms with two sets of chromosomes. There are three different ways to study population genetics. Empirical studies are done to understand how genes vary in different groups of living things and to figure out why this variation happens. Scientists study the main influences on genetic variation in populations and how the species' biology affects this variation. Experimental studies try to examine the outcomes of inbreeding and crossbreeding, both natural and artificial selection, and random genetic changes caused by population bottlenecks or small population size. They also aim to determine how often spontaneous mutations happen and their impact on the overall fitness [3], [4].

DISCUSSION

To know how population genetics started, and understand why it is important, we need to look back at the history of biology for a bit. In 1859, Darwin wrote a book called Origin of Species. In this book, he had two main ideas. The first idea was that all the different species we have now come from the same ancestors. The second idea was that the main way species change and evolve is through a process called natural selection. The scientific community accepted the first idea easily, but they didn't accept the second one. A lot of people had a hard time believing that natural selection could explain what Darwin's theory required it to explain. This situation continued until the 20th century. People accepted that evolution had occurred but had doubts about Darwin's explanation of why it happened. People who disagreed with natural selection had a valid point. Even though Darwin's theory was convincing, it didn't explain how traits get passed on to offspring. In order for evolution by natural selection to happen, parents need to look similar to their children. If they don't, traits that make a person stronger and better won't spread in a group of living beings. In the beginning, Darwin based his argument on the idea that children often look like their parents. He called this the strong principle of inheritance. However, he admitted that he didn't know why this happens. Darwin later tried to come up with a theory about how traits are passed down, using made-up things called 'gemmules', but it turned out to not be true [5], [6].

Darwin was confused and worried because he didn't fully understand how traits are passed down from parents to offspring. This made it difficult for him to respond to a strong argument against his theory. In order for a population to change through natural selection, the individuals in the population need to be different from each other. If all organisms are exactly the same, there won't be any selection happening. In order for a population to change slowly over a long time, like Darwin suggested, it is necessary to constantly have new differences among individuals. Fleeming Jenkin said that the amount of variation that is available will be used too quickly. In simple words, he believed that a child's traits are a mixture of its parents' traits. If a short and tall organism have a baby, the baby's height will be in the middle. Jenkin said that if traits blend together, a population will become more similar in just a few generations. This happens much quicker than the time it takes for natural selection to create complex changes.

Thankfully, Darwin's theory is not supported by the way Jenkin believed inheritance works. The kind of inheritance called 'Mendelian' means that offspring receive separate pieces of hereditary information from their parents. This is different from a 'blending' type of inheritance. It also means that sexual reproduction does not reduce the variation that can be inherited in a population. The understanding of this concept took a while for two reasons. At first, scientists didn't pay attention to Mendel's work for forty years. Secondly, even after scientists found Mendel's work again around the year 1900, many people thought that Darwin's theory of evolution and Mendel's ideas about inheritance could not both be true at the same time. The people who researched early genetics did not think that natural selection was important in the development of species over time. Because of this, they did not realize that Mendel's discoveries actually supported Darwin's theory of evolution. The combination of Darwin's ideas and Mendel's ideas led to the creation of population genetics. However, it took a long and difficult journey to bring these two theories together [7], [8].

The main ideas of Mendel's theory of inheritance are easy to understand. In his experiments with pea plants, Mendel noticed something strange. He started with two types of plants that always had either round or wrinkled seeds. After that, he combined these to make the first group of children (the F1 generation). All of the F1 plants had round seeds and there were no more wrinkled seeds. Mendel then bred the F1 plants together to create the F2 generation. Surprisingly, around 25% of the F2 plants had seeds that were not smooth. So, the trait of having wrinkles returned, but it skipped one generation. Mendel explained these and other similar observations in a way that was easy to understand. He believed that every plant has two things that work together to decide its physical characteristics, like the shape of its seeds. A plant gets one trait from each of its parents. Imagine if there is only one thing that determines whether a seed is round or not. In simple terms, this means that something is in a ratio of about one part, to two parts, to one part. Mendel's First Law, also known as the Law of Segregation, states that during the formation of reproductive cells, the pairs of genes inherited from each parent separate or segregate randomly.

In the pea plant example, the different forms of a gene are called alleles. The law of segregation means that when sex cells are formed, they only receive one of each chromosome pair from their parent organism. Other parts of Mendel's theory have been changed because new information was found later on. Mendel believed that most physical traits were controlled by only one pair of factors. For example, he studied pea plants and found that seed shape was determined by a single pair of genes. However, it has been discovered that most traits are actually influenced by multiple pairs of genes, rather than just one. Mendel thought that the factors responsible for different traits separate from each other independently. However, we have since learned that this may not always be true. Notwithstanding these

factors, Mendel's theory is a pivotal moment in how we comprehend inheritance. When Mendel's work was rediscovered in 1900, the scientific community did not immediately start believing in Mendelism. At that time, the main way of studying inheritance was biometry. This method, led by Karl Pearson in London, involved using math to analyze how traits vary in different groups of living things. Biometricians were mostly focused on traits that change gradually, like height. They weren't as interested in traits that have clear categories, like seed shape. They were also supporters of Darwin's theory of gradual evolution. The Mendelians, led by William Bateson, believed in distinct variations and thought that significant changes could happen through individual genetic mutations instead of gradual natural processes proposed by Darwin. They disagreed with the biometricians. A big argument started between two groups of scientists called the biometricians and the Mendelians. As a result, people started linking Mendelian inheritance with a view of evolution that goes against Darwin's ideas.

Population genetics started because scientists wanted to explain how the ideas of Mendel and Darwin fit together. They felt a strong need to do this as they kept finding more and more evidence that supported Mendel's ideas about how traits are inherited. A big achievement was R. A'sFisher's 1918 paper, 'The Correlation between Relatives on the Supposition of Mendelian Inheritance', explained how biometrical and Mendelian research methods can work together. Fisher showed that if a trait like height is influenced by many small factors, it will have a normal distribution in a population. In simpler words, proving that the way traits are inherited is compatible with both Darwin's theory and Mendel's theory was a crucial step in bringing these two ideas together. This was because it was commonly thought that Darwin's theory worked best for traits that changed gradually over time.

In the 1920s and early 1930s, Fisher, Haldane, and Wright used math to fully resolve the disagreement. Fisher's work was published in 1930, Haldane's work was published between 1930 and 1932, and Wright's work was published in 1931. These researchers created models to study how natural selection and other evolutionary factors like mutation and random changes would change the genes of a population over time. This work was a big leap forward in the study of how organisms have evolved over time. It allowed scientists to measure and analyze the effects of different ideas about evolution, instead of just describing them. People stopped arguing with words about what natural selection can do or not do, and about the different patterns of genetic variation it causes. Instead, they started using clear and direct mathematical arguments. The way scientists create formal models to understand how evolution works is still the most common approach in population genetics today. However, nowadays scientists have a lot more real-world data to compare their predictions with, unlike in the 1930s.

Modern population genetics is conducted in a different scientific environment than what Fisher, Haldane, and Wright experienced. They were working during a time when molecular biology was not yet understood. During this time, scientists used the concept of a "gene" to explain how certain traits were passed down, even though they didn't know the physical details of what a gene actually looked like or how it was made up. Genetic variation can only be seen indirectly through the changes in physical traits that it may cause. This means that there wasn't enough real-world data to test population genetic models, so the study stayed mostly theoretical. In the past hundred years, scientists have learned a lot about genes. They went from being a concept to being something that we can study and understand how it works. Since the 1980s, the process of determining the order of nucleotide bases in a DNA strand, called gene sequencing, has become faster and less expensive. This has made it possible for scientists to study the different genes found in natural populations by looking at a

small group of individuals and examining a specific gene (or sometimes all the genes). Population genetics has become a science that has lots of data, which is very different from when it first started and had very little data [9], [10].

It is important to note that no matter what the initial genetic proportions are, when animals mate randomly, their offspring will naturally have genetic proportions that follow the Hardy-Weinberg principles. If the parents die once they have children and don't overlap with future generations, only one random mating is required to reach Hardy-Weinberg proportions in the entire population. However, if parents can have children while still being alive, more than one random mating session is needed. If the population continues to mate randomly and is not affected by any changes caused by evolution, the proportions of Hardy-Weinberg will be maintained in future generations. This means that the population is stable because the frequencies of different gene combinations stay the same over time. The Hardy-Weinberg principle is important because it solves the problem of blending inheritance that Darwin was worried about. Jenkin believes that sexual reproduction will quickly decrease the differences among individuals in a group. However, the Hardy-Weinberg principle proves this to be incorrect. Sexual reproduction does not naturally decrease the variety of genetic characteristics in a population because the proportions of different genetic traits stay the same from one generation to the next, as long as certain assumptions hold true. It is true that natural selection can reduce variations, making things more similar. But this is not the same thing. The 'blending' objection said that when different organisms have babies together, their traits would become the same over time, even without any specific selection. But the Hardy-Weinberg principle proves that this is not true [11], [12].

Another advantage of the Hardy-Weinberg principle is that it makes it much easier to model how evolution happens. When a population is in Hardy-Weinberg equilibrium, you can keep tabs on the types of genes in the population by measuring how often different versions of the genes appear. It's obvious that this is true because if we know how often different forms of a gene appear in a population and we know that the population is in a stable state, we can easily calculate the distribution of different combinations of those genes. If the population is not in Hardy-Weinberg equilibrium, we would have to keep a close eye on the frequencies of different genotypes, which is more complex. Many models of population genetics assume that Hardy-Weinberg equilibrium is true. This means that it assumes mating is random regardless of genotype. But can we prove that this assumption is true. The answer is sometimes yes, but other times no. In the human population, when it comes to blood types, mating is mostly random. This means that the genes that determine blood type are found in similar proportions in many populations. However, when it comes to height, people don't randomly choose their partners. Instead, they often pick someone who is similar in height to themselves. So, if we have a specific genetic factor that affects how tall someone is, then when it comes to choosing a mate, people won't choose randomly. Johnson stated that the size of a population, as well as other factors, can affect the genetic diversity of that population. Ewens said that the Hardy-Weinberg principle is not always easy to understand even though it is the most important theorem in its subject.

Genetic drift is when allele frequencies in a population change randomly without any benefit to the population. When a population is smaller, it is more prone to genetic changes due to limited genetic diversity (Figure 1). This means that certain gene variations are more likely to either disappear completely or become very common throughout the whole population. Random events that change the frequencies of certain genes will have a bigger impact when there are fewer genes in a population. Genetic drift and natural selection often happen together in groups of organisms, but it's usually hard to tell why the frequency of certain traits

changes. Differential pollen and seed production means that there is a random selection of genes from the parent population. When the sample is smaller, the offspring deviate more from the expected allelic ratios in the Hardy-Weinberg theory. If there are not many individuals in the population, random changes in gene frequencies will happen every generation. Drift effects can continue for a long time even after the population recovers in size, if the original genetic diversity is not restored through the movement of genes, for example, after a major decline in population or if only a few individuals establish in a new environment. For instance, many temperate species still show a decrease in alleles after the ice age, even though genes have been exchanged for many generations.

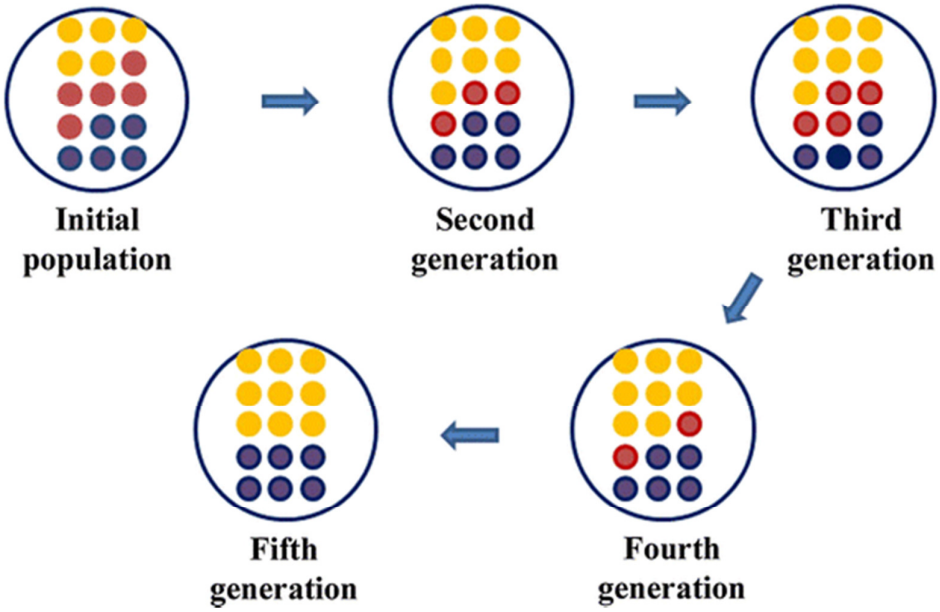

Figure 1: Representing the overview about the genetic drift [Research Gate.Net].

It is now obvious that the number of alleles in a population will depend on how many individuals are there in that population. But when there are only a few members in a population, there is another factor called genetic drift that affects the genetics of organisms. Genetic drift is when the frequency of certain traits randomly change in a group of organisms over time. Genetic drift is a random occurrence that can cause big changes in populations in a short time. Random drift happens when there aren't many individuals in a population, when the population size greatly decreases, or when a new population starts from a small number of individuals. Genetic drift causes certain traits or combinations of genes to become dominant in a group of organisms. Drift makes inbreeding worse and increases homozygosity by getting rid of different gene versions. Drift is likely to happen in populations that experience regular cycles of dying out and re-establishing. This could be really important in nature when plants and pathogens are spread out and exist in small groups.

Genetic drift is when allele frequencies change randomly. We call it "random drift" or "random genetic drift". There are three ways that sampling error can happen. We will look at these things in relation to the number of harmful organisms in plant diseases. A small recurring population size happens when there are not a lot of plants for the virus to infect, or when the conditions are not good for the virus to spread killed or removed and only a few individuals are left. This leads to a decrease in the diversity ofgenes within the population. When farmers collect their crops, or when the environment changes to stop the plant from getting sick or to kill the germ directly Hot and dry weather or extremely cold weather. A

founder effect happens when a few individuals start a new population with only a small amount of the variety of genes that the whole species has. A founder event happens when a couple of sick plants get into an area where they shouldn't be and bring a disease with them. This disease wasn't there before in that area.

The likelihood of fixing a gene variation because of a random change in a population's genes depends on how many individuals are in the population and the distribution of different gene variations. To "fix" an allele means that everyone in a population has the same gene at a specific spot. When there are many individuals in a population and the frequencies of different traits are spread out equally, it is less likely that one trait will completely take over the population. Genetic drift is when some alleles (different versions of a gene) become more or less common in a population by chance. If an allele is rare, it is usually at a disadvantage in genetic drift. Rare alleles are more likely to vanish from a group than alleles that are more common. In a situation where only genetic drift is happening, the chance of an allele becoming permanent in a population is based on how common it was at the beginning. If the starting amount of a gene is 0. 01, then there is a 1 in 100 chance that this gene will become the only one in the population. If we consider things from a different perspective, if 100 different groups had an initial allele frequency of 0. 01, then we would anticipate that around 1 of those groups would eventually have only this allele after many generations of random genetic changes. There are many results of genetic drift. It causes unpredictable changes in the types of genes in a population. Drift happens when certain traits become more common in a population because some alleles or gene combinations are lost. Drift can cause some genes to become more common or disappear completely in organisms that reproduce asexually. Drift makes it more likely for organisms with the same genetic traits to reproduce, which can result in increased inbreeding. When populations don't exchange genes, drift makes them more genetically different from each other.

Genetic drift has two important long-term effects on evolution. Genetic drift can help create new species by allowing the gathering of mutations that do not help with survival, but can separate populations. Drift helps a group of organisms move from being less fit to being more fit, as explained by Sewall Wright's shifting balance theory. The number of groups of people is likely to increase because some traits are randomly lost in different groups. Plus, there can be random changes in the frequencies of certain genes in different groups of organisms, and these random changes often lead to those groups becoming different from each other. In simple terms, when there are not many individuals in a population, the chances of close relatives mating with each other go up. This causes more inbreeding and a decrease in genetic diversity. In farm fields, the number of pathogens usually get very big because the host plants have similar genes. This means that genetic drift doesn't have a big effect on how the pathogens evolve. Not many tests have been done to check this idea. However, there is a lot of proof that supports the idea of founder effects in farming environments, particularly in Australia. This is because Australia was the most recently settled continent by Europeans, who brought their crops and diseases with them. In nature, genetic drift can have a bigger impact on the development of diseases because the animals or plants that get sick have different genetics and are spread out, so there aren't as many disease-causing organisms and they often go through periods where their population shrinks. We will talk about this idea again after we explain what metapopulations are.

Mycosphaerella graminicola is a type of fungus that causes a disease called Septoria tritici leaf blotch on wheat plants. McDonald and his team used markers called restriction fragment length polymorphism (RFLP) to study the genes of this harmful organism around the world. They discovered that most populations they examined from different places shared similar

common genes, except for those from Australia and Mexico. The Australian and Mexican populations had less genetic variety and fewer variations at each location. Some specific genetic patterns were found in these populations, and the gene frequencies were noticeably different from populations in other places. In Australia, there weren't many people who came to this continent when modern farming started, so this could be the reason why. The population of Patzcuaro, Mexico was selected from a breeding nursery that CIMMYT uses to test for resistance to a specific disease. This nursery is found in a place far from where wheat is grown. Because of this, it doesn't get many natural wheat diseases. Additionally, only a few types of diseases were purposely introduced to the nursery. This situation demonstrates how the genes of the diseases can change over time because there were only a small number of diseases at the beginning and they were kept isolated. On the other hand, the people in Israel had the greatest variety of genes. This supports the idea that the Middle East is where this harmful organism comes from.

A really big example of genetic changes caused by a small population is when the Phytophthora infestans pathogen, which makes potatoes sick, went through a bottleneck. It escaped from Mexico and went to North America, then arrived in Europe and caused the Irish potato famine. After that, it spread to different parts of the world because of human trade. Stripe rust of wheat in Australia can be traced back to one single occurrence or event. Please rewrite this text in simpler terms. researchers found only one type of This suggests that Europe was where it originally came from. Since the beginning, mutations have caused new varieties in the single genetic background that was introduced. The disease called chestnut blight in North America has fewer variations in its genes compared to the disease in Asia, which suggests that it started from a small group of individuals. It seems that Japan is the main place where there is a lot of different species and where they might have originally come from.

CONCLUSION

Population genetics is a branch of biology that focuses on studying how certain traits, called alleles, are distributed and how they may change in a group of organisms. Population genetics is the study of how different characteristics in a group of organisms change over time. It helps us understand how evolution happens. Since 1966, the field of population genetics has made a lot of progress. This includes creating a lot of math theories and tools, techniques for the lab, molecular markers, and a ton of information about genetic variations in databases. The most important idea in population genetics is the Hardy-Weinberg theorem. This important theory says that if there are lots of individuals in a population and they mate randomly, and if there isn't much mutation, selection, or migration happening, then the frequencies of different versions of genes will stay the same from one generation to the next. If not, the types and combinations of genes will be different in each new generation. These changes can directly impact how well a population can adapt to its environment. By studying the genetic differences in populations, we can gather important information for practical studies and decision-making.

REFERENCES:

[1] S. Casillas and A. Barbadilla, “Molecular population genetics,” *Genetics*. 2017. doi: 10.1534/genetics.116.196493.

[2] B. Haubold, “Alignment-free phylogenetics and population genetics,” *Brief. Bioinform.*, 2014, doi: 10.1093/bib/bbt083.

[3] J. L. Davey and M. W. Blaxter, "RADseq: Next-generation population genetics," *Brief. Funct. Genomics*, 2010, doi: 10.1093/bfgp/elq031.

[4] K. S. Kim and T. W. Sappington, "Microsatellite data analysis for population genetics," *Methods Mol. Biol.*, 2013, doi: 10.1007/978-1-62703-389-3_19.

[5] M. Stephens and P. Donnelly, "Inference in molecular population genetics," *J. R. Stat. Soc. Ser. B Stat. Methodol.*, 2000, doi: 10.1111/1467-9868.00254.

[6] S. Joost *et al.*, "Uncovering the genetic basis of adaptive change: On the intersection of landscape genomics and theoretical population genetics," *Molecular Ecology*. 2013. doi: 10.1111/mec.12352.

[7] A. Estoup, P. Jarne, and J. M. Cornuet, "Homoplasy and mutation model at microsatellite loci and their consequences for population genetics analysis," *Molecular Ecology*. 2002. doi: 10.1046/j.1365-294X.2002.01576.x.

[8] C. O. Wilke, "Quasispecies theory in the context of population genetics," *BMC Evol. Biol.*, 2005, doi: 10.1186/1471-2148-5-44.

[9] A. Gilabert and J. D. Wasmuth, "Unravelling parasitic nematode natural history using population genetics," *Trends in Parasitology*. 2013. doi: 10.1016/j.pt.2013.07.006.

[10] J. M. Housset, C. Carcaillet, M. P. Girardin, H. Xu, F. Tremblay, and Y. Bergeron, "In situ comparison of tree-ring responses to climate and population genetics: The need to control for local climate and site variables," *Front. Ecol. Evol.*, 2016, doi: 10.3389/fevo.2016.00123.

[11] L. Hosking *et al.*, "Detection of genotyping errors by Hardy-Weinberg equilibrium testing," *Eur. J. Hum. Genet.*, 2004, doi: 10.1038/sj.ejhg.5201164.

[12] J. E. Wigginton, D. J. Cutler, and G. R. Abecasis, "A note on exact tests of Hardy-Weinberg equilibrium," *Am. J. Hum. Genet.*, 2005, doi: 10.1086/429864.

CHAPTER 8

QUANTITATIVE GENETICS: UNDERSTANDING THE BEHAVIOUR OF GENE INHERITENCE

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ABSTRACT:

Quantitative genetics is a field of genetics that focuses on understanding how traits are inherited and measured. Classical quantitative genetics is another term for statistical genetics or biometrical genetics. This field focuses on using statistical analysis to identify potential genetic patterns in a specific group of organisms. In 1918, Fisher created a genetic model for quantitative traits that separates the gene effect and genetic variance into additive and dominance parts. His basic genetic model was expanded to include different interactions between genes and a set of methods were developed in the 1940s-1960s to identify and confirm the genetic model and its effects. The idea of a single major gene plus polygene mixed inheritance model was developed to explain how individual genes in the genetic system can have different effects. This concept and analytical procedure was established in the 1970s-1990s and includes the hypothesis of a generalized major gene plus polygene mixed inheritance model along with segregation analysis procedures. QTL mapping, using molecular markers, made studying how traits are passed down much easier and broader. The study of molecular quantitative genetics has given us a way to understand and analyze a trait that is influenced by multiple genes. Now that we have improved methods for analyzing and understanding biological information, we can create networks that show how genes work together in a coordinated way. This means we might be able to prove the idea that genes influence complex traits through a network of interactions. This would require conducting experiments in the future.

KEYWORDS:

Breeding, Genetics, Quantitative, Selections, Traits.

INTRODUCTION

Quantitative genetics is the study of how genes affect the differences in traits among individuals. Quantitative traits are traits that are controlled by many genes. They can also be influenced by the environment, which can change how an individual looks or behaves. Quantitative traits can change in a smooth and gradual way. Some examples are producing milk, how tall you are, how much you weigh, and how long you live. So, the study of genetics for traits that have a wide range of values is called quantitative genetics. Instead of looking at changes in specific genes, quantitative genetics measures changes in the distribution of traits that cannot be easily sorted into categories. Heritability (H2) in simple words means how much of a trait is influenced by genes. It is the proportion of the variation in a characteristic that is caused by genetic factors. It measures how much genes affect the differences in traits. Heritability (h2) in "narrow sense" is how much of a person's traits are influenced by their genes. It is necessary to understand how a group of living things reacts to natural or man-made selection. Understanding why certain traits vary in animals is important for predicting how they will respond to selective breeding and for studying evolution in

natural populations. In the past, researchers studied how traits vary using statistics without knowing which genes were responsible for these variations. They would break down the different factors that contribute to differences in physical characteristics. However, continued advancements in genomic technology are making it increasingly possible to find specific genes that cause differences in traits that can be measured and quantified [1], [2].

Molecular genetics studies how genetic material works and looks on a very tiny level. The field of study is about combining different parts of biology to learn more about it. The study of cells, how traits are passed down from parents, how molecules interact in cells, the chemistry of living things, and the use of technology in biology. The molecule that carries our genetic information and is responsible for passing traits from one generation to the next is called DNA. The finding of the DNA structure by Watson, Crick, Franklin, and Wilkins was a big achievement for genetic studies. The process of separating a restriction endonuclease in Escherichia coli (E. coli) bacteria into its own individual component. coli by Arber and Linn in 1969 started the field of genetic engineering. Restriction enzymes are tools that are used to precisely cut DNA into smaller pieces according to specific patterns. These smaller pieces can then be separated and studied using a technique called electrophoresis. In 1971, Berg used special enzymes to make the first mix of DNA from different sources. He also made the first small DNA piece that can replicate itself. In 1972, Cohen and Boyer made the first organism with altered DNA by putting new DNA into E. Coli, which is now called bacterial transformation, opened the door for molecular cloning. In the late 1970s, scientists made important discoveries about DNA sequencing. This allowed researchers to study how specific sequences in our genes are related to physical traits [3], [4].

These discoveries were a big step forward in genetic research. Polymerase chain reaction (PCR) is a scientific technique invented by Mullis in 1985. It uses an enzyme called Taq polymerase to make many copies of a specific DNA sequence. These copies can be used for experiments or to separate DNA using agarose gel. Ten years later, scientists sequenced the entire set of genes in a bacterium called Haemophilus influenzae. After that, in 2001, they successfully sequenced the full set of genes in a human as part of the Human Genome Project. The differences in important traits among animals can be understood by using two models: the infinitesimal model and the finite loci model. The very small model is used in quantitative genetics, while the limited gene model is used in molecular genetics. The infinitesimal model suggests that traits are influenced by many different genetic factors, each with a very tiny impact. These factors are not connected to each other. This model is useful for breeding animals and is the foundation for estimating the value of breeding. The finite loci model supposes that there is only a limited amount of genetic material that gets passed down from one generation to the next. Several pieces of evidence have shown that the way these genes affect our traits can be grouped into a few genes that have a strong impact and many genes that have a weaker impact [5], [6].

DISCUSSION

Population genetics and quantitative genetics are two types of biology that study how genes impact the differences in how individuals look and function within a group of living things. Population genetics is about studying how often different versions of genes are found in a population. Quantitative genetics is about studying how certain traits are linked to the genes that cause them so that researchers can better understand how these traits change in a population over time. This helps us predict how these traits will respond to natural selection based on information about how they look and how genetically related individuals are to each other in the population. In the past, quantitative genetics started with math ideas about genetic effects. This was explained by Karl Pearson and Ronald Fisher in the early 1900s. This is the

traditional perspective on quantitative genetics. The new way scientists look at genetics is using tools like genomics, bioinformatics, and computational biology to find connections between genes and complex traits. Genes that control measurable characteristics are called quantitative trait loci (QTLs). Scientists are using a method called molecular-based QTL analyses to study how different parts of our DNA are related to certain physical or behavioral characteristics. This helps them understand the genetic makeup that contributes to these traits. There are signs of a big change in the field of studying genetics using numbers. In this chapter, we talk about both traditional and modern ways of studying genetics [7], [8].

In this section, we will be talking about classical quantitative genetics. We will discuss things like the differences in genetics and environment, the connections between genetics and diversity, and problems related to genetics in populations. Most traits in plant breeding are inherited in a quantitative manner. Many genes determine traits, with each gene having a small impact on how the trait is expressed. The difference in how much a trait is expressed does not have any jumps or breaks, but is instead a smooth and constant change. Continuous variations are traits that can be measured and observed in different degrees or amounts. These traits can also be referred to as metric traits. Trying to put these traits into different groups is just a random decision. For instance, height can be measured with numbers. If we divide plants into two groups based on height (tall and short), we can still find tall plants in the short group, and short plants in the tall group. Qualitative genetics and quantitative genetics differ in terms of the type of traits they study. Qualitative genetics involves studying traits that follow Mendelian inheritance patterns and can be clearly categorized and described according to their characteristics. Qualitative genetic traits create distinct differences in physical traits, while quantitative genetic traits create a wide range of gradual differences. How many genes are there. In simple terms, in qualitative genetics, the effects of individual genes are easily noticeable, but in quantitative genetics, the effects of individual genes cannot be seen [9], [10].

Instead, traits are controlled by many genes, each with a small and undetectable effect. Mating pattern refers to how animals or organisms choose their partners to reproduce. Qualitative genetics focuses on the pairings of individuals and their offspring. Quantitative genetics focuses on studying a group of individuals that have different ways of reproducing. Data analysis using mathematical and computational techniques. Qualitative genetic analysis is easy to understand. It involves looking at numbers and comparing them. On the other hand, quantitative analysis gives us numbers that estimate certain characteristics of a whole group, based on information from a smaller sample within that group. 423 The environment and quantitative variation: All the genes in an organism are influenced by the environment, leading to variations in physical characteristics which are a combination of the genetic information and the impact of the environment. But, numerical characteristics are more affected than descriptive characteristics. Under very strong environmental influences, traits that are determined by only a small number of genes can show a pattern of inheritance that is more continuous rather than discrete. When the environment has a strong effect, the different groups start to blend together.

Quantitative traits are traits that are influenced by more than one gene. Polygenes are genes that have very tiny effects and cannot be identified individually (Figure 1). They are also known as minor genes. In polygenic inheritance, the trait is influenced by many different genes and each gene can be located at different spots on the chromosomes. The way traits show up in plants can be changed a lot by different environmental factors that the plants are exposed to. Polygenic variation cannot be divided into clear categories. Instead, it is a continuous range of variation. This is because there are many separate areas in our genes that

contribute to traits, but each one only has a small impact. So, it is difficult to see how each gene affects specific traits. However, traits that are influenced by many genes show a wide range of variations. The amount of genes. In genetics, we can easily see the impact of individual genes in qualitative genetics. However, in quantitative genetics, it is difficult to see the effects of individual genes. Instead, traits are controlled by many genes that have small, unnoticeable effects. The way animals reproduce. Qualitative genetics focuses on how individual organisms mate and the traits that are inherited by their offspring. Quantitative genetics studies a group of individuals that can have different ways of reproducing. Statistical analysis is when you use numbers and data to study and understand things. Genetic analysis based on counts and ratios is easy to understand. However, quantitative analysis gives us an idea of certain characteristics of a large group of people.

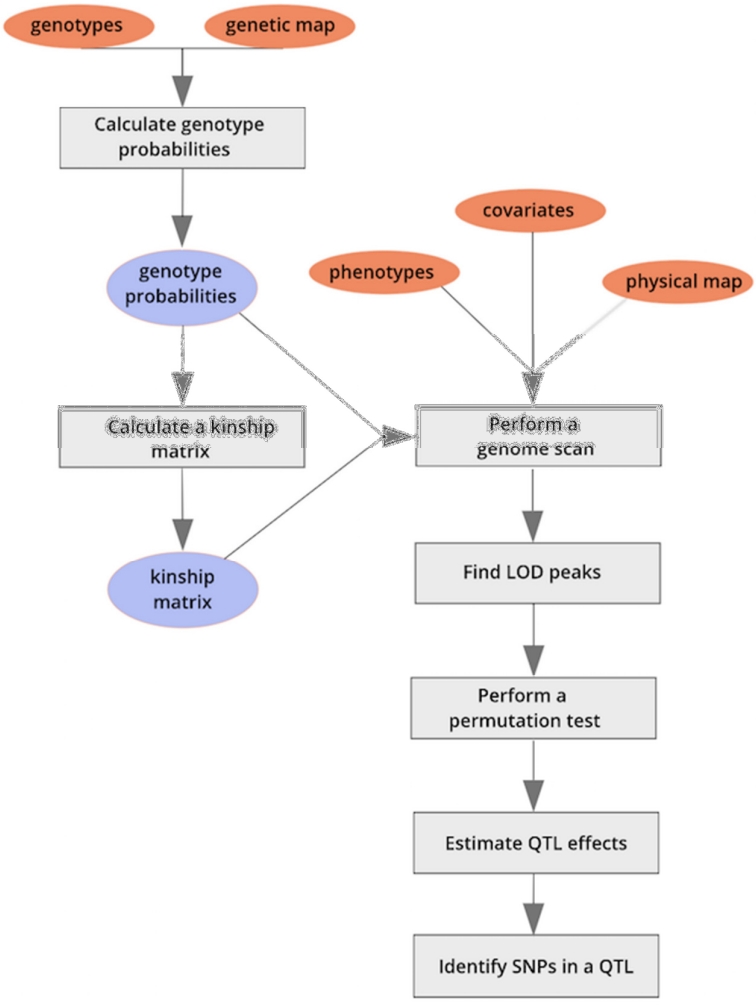

Figure 1: Representing the overview about the Quantitative traits determination [Smcclatchy].

The environment and quantitative variation All genes are shown in an environment appearance is determined by a combination of genes and the environment's impact. However, characteristics that can be measured tend to be affected more than characteristics that are more subjective. When the environment has a big impact, certain characteristics that are controlled by just a few important genes can show patterns of inheritance that involve a range of values. A strong environmental influence makes the different classes come together.

Polygenes and polygenic inheritance in simple words mean that quantitative traits are controlled by more than one gene. Polygenes are a type of genes that have very tiny effects and cannot be easily identified on their own. Sometimes, people refer to them as minor genes. In polygenic inheritance, the trait is influenced by many genes located at different places in the DNA. The way plants look and behave can be influenced a lot by the environment they grow in. Polygenic variation cannot be sorted into separate groups because it is continuous. This is because there are many separate locations in our genes that determine traits, but each location has a very tiny effect. It is impossible to figure out the specific effects of each gene in this process. However, this research has helped connect our knowledge of traits that can be measured and traits that cannot be measured. Polygenic inheritance means that many genes contribute to a trait. These genes do not have a dominant form and their effects are added together.

This section is about how organisms react to selection, specifically in terms of genetic improvement. After creating different variations, the breeder's next important task is to move the population forward through selection. Selection means choosing certain individuals from a group with different genes to create the next generation. This means that different types of genes in the population reproduce at different rates, which changes the frequencies of those genes. As a result, the characteristics of individuals, both in terms of their genes and physical traits, are also affected. Although artificial selection has a direction, the idea of completely or purely selecting artificially is not possible because natural selection already influences the plants before breeders can choose the ones they want. The person who breeds animals or plants wants to choose the best ones from a group of different individuals. They hope that these chosen ones, who have strong genetic traits, will improve the average quality of the population in the next generation. The person who breeds animals needs to have a clear goal or purpose in mind. The characteristic that needs to get better must be clearly described. Characteristics that are influenced by important genes are usually not difficult to choose. But, traits that are determined by multiple genes and are complicated genetically and biologically, are difficult for breeders to work with. The response to selection (R) is the difference between the average characteristics of the children of the chosen parents and all the parents before they were selected. The response to selection is just the change in the average traits of a population over generations after selection. Similarly, the selection differential (S) is the average value of the chosen individuals' physical traits compared to the average value of all individuals in the previous generation. The way organisms respond to selection is influenced by their inherited traits. This can be calculated using the equation: $R = h2S$. It means that the response to selection (R) is equal to the heritability (h2) multiplied by the selection pressure (S). The goal of selection is to achieve genetic improvement in one generation.

The amount of variation in the population that will be selected from. The target trait's heritability. This means the plant breeder decides how many plants from the current generation will be chosen for the next generation. A big difference in physical traits would give the breeder many options to choose from. Even if the trait is highly influenced by genetics, there won't be much improvement unless there is a lot of variation in how the trait appears. When the heritability is high, choosing and improving only a few of the best performers is likely to result in a bigger genetic improvement compared to choosing many performers who are okay but not exceptional. However, if there is a lot of pressure to choose certain traits, it can cause a decrease in the amount of differences among individuals. When a trait is not strongly influenced by genetics, the breeder should choose animals with less strict criteria to try to breed as many individuals with high potential as possible. Basically, when predicting how something will respond, it is only accurate for one group that is chosen. This is because how much a trait is passed down from parents to offspring affects how it responds

to being selected for. In order to guess how future generations will turn out, we need to figure out the heritabilities in each generation. The amounts of traits inherited can vary from one generation to the next. This is because if a trait changes, it will also cause changes in the genes that determine inheritance. Additionally, choosing specific parents lowers the variability and inheritability, particularly in the beginning generations. It's important to mention that heritability changes are usually not very big. If heritability is unity (VA = VP; no environmental variance), a breeding program would have perfect progress and the average traits of the offspring would be the same as the average traits of the parents chosen for breeding.

Plant breeders have to look at a lot of different plant qualities when they're trying to create new plants. While breeders may choose specific traits to focus on during breeding, they are also interested in how well the overall cultivar performs. During selection, breeders imagine what they want the final product to be like and consider the good qualities and minor flaws before making their final decision in the selection process. Explicit indices are difficult and time-consuming because they require the breeder to keep detailed records and perform complex statistical analysis. Most breeders use a mix of cutting off certain traits and making decisions based on intuition when selecting traits for their breeding programs. General worth means that each crop has different qualities that make it desirable to both farmers and consumers. These qualities, when looked at as a whole, determine how much the crop is valued. These characteristics can vary from about ten to several dozen, depending on the type of plant, and they are the main set of characteristics that plant breeders focus on improving. These characteristics are not all equally important and they can be easier or harder to change through breeding. Plant breeders usually focus on one or a few specific characteristics to improve in their breeding program. In other words, the breeder creates a list of characteristics to meet the requirements of the objectives. Almost everyone wants to get the most economic product when breeding plants. Disease resistance is more about the specific needs of a particular place. What might be economically significant in one location may not matter as much in a different area.

Although a plant breeder may concentrate on a few characteristics, their ultimate goal is to enhance all the important qualities that make a crop more desirable or valuable overall. Simply put, breeders take a well-rounded approach when choosing which animals to include in their breeding program. The last decisions are based on a fair assessment of the main characteristic of the plant. Some things can be easily checked just by looking at them, like shape, color, and size. Other things need to be tested in a lab or measured with special equipment, like the amount of oil in something or the characteristics of cotton fibers. Special needs like a greenhouse or isolation area might be necessary for disease breeding. It is more dependable to assess yield by observing it across different seasons and locations in the field. Besides selecting the desired characteristics, the breeder needs to determine how much of each trait should be present. If the level falls below a certain point, the plant material will be deemed useless. The level at which a trait is considered acceptable can be either very strict or not so strict.

In crops grown for industry, like cotton, the quality of the product can be very specific, for example, it needs to have a certain weight and be a certain length. In breeding for disease resistance, choosing plants with just a little resistance may be just as good as choosing plants with very strong resistance. On the other hand, when it comes to improving the quality of the food we breed, there might be rules about how much of harmful substances are allowed. Early generation testing is a way for breeders to test lines or families of plants that have different genes. This testing happens earlier than usual. For instance, we can use recurrent

selection with testers to check materials in the beginning stages. One important thing the breeder thinks about when choosing how to breed animals is to get as much genetic improvement as possible each year. Testing early means trying things out at the beginning, and if it works well, it helps to find and choose good plants from strong families in the beginning stages of the breeding program. The early generation selection method has been compared to other methods like pedigree selection, single seed descent, and bulk breeding, and people like it more. People often wonder when the test will start. Which family should it be in, F1, F2, or F3. When deciding on the generation for selection, factors to think about are the trait being improved and if there are nurseries available to produce more generations per year instead of selecting early.

Over time, scientists who work with plants have tried to find ways to make plant breeding easier by choosing the best parents for mating, selecting the best plants from a group for further breeding, and predicting how plants will respond to breeding efforts. Measuring the influence of genetics on plant breeding involves using statistical methods to determine different factors and calculate their levels of variability. Because the estimates of the variation in genetic traits are not strong or precise, the usefulness of statistical genetics for breeders has been restricted. This method can be used for creating new corn varieties. The process called combining ability involves evaluating crosses between chosen parents to determine how much variation among crosses is due to the traits passed down from the parents, and how much is due to other factors. When you draw multiple lines across one line, it adds up to the average performance of that line in all the intersections. This means how well a line performs compared to the average of all crosses. This is what Sprague and Tatum called the general combining ability (GCA) of the lines. The GCA is found by adding up all F1s that have this specific line as one parent, and then dividing by the total number of crosses. The result is a measure that shows how different these F1s are from the average of all crosses. Every intersection has a predicted value, which is the combined value of the two parental lines. However, each combination may differ from what was expected. This difference is called the specific combining ability (SCA) of the two lines that were combined. The reasons for the differences in GCA are because of the way certain things add up together in the population. The variations in SCA are caused by differences in genes that are not passed down from parents. Additionally, as inbreeding becomes more common in the population, the SCA is predicted to increase faster and have a wider range of variation. GCA means how well a plant performs when it is crossed with different tester lines, while SCA measures how well a plant performs in a particular combination compared to other combinations of crosses.

Genomic selection is a method used to choose plants or animals with desirable traits by analyzing their DNA. This analysis looks at all the genes in the organism to determine which ones are associated with certain traits. Using this information, breeders can then select individuals with the desired traits more accurately and efficiently. In regular plant breeding, selection is usually based on estimates of breeding values using mixed models that consider the ancestry of plants. However, these models cannot consider Mendelian segregation, and without inbreeding, they can only explain half of the genetic differences. Molecular markers can help trace the genetic traits of an organism at different parts of their DNA. This helps us get more precise estimates of the organism's genetic values and improve their breeding selection. Although marker-assisted selection (MAS) has had some success, it is difficult to use for improving traits that are measured on a continuum due to different obstacles. The biparental mating methods used to find genes affecting quantitative traits and the statistical methods used are not good for traits controlled by multiple genes. Marker-assisted selection (MAS) uses molecular markers that are connected to the genes of interest. Genomic selection,

also known as genome-wide selection, is suggested as a better way to improve traits related to quantity. It uses all the available markers in the genes to estimate genetic or breeding values. There are many markers in the genes. We can use high-density markers and high throughput genotyping to make a prediction model. This helps us avoid having biased estimates for the effects of markers and allows us to capture more of the variation caused by the small-effect genes. The genomic selection method uses two types of data: a training set and a validation set. This is used in a group of organisms that is different from the one used to estimate the effects of the markers.

It can help identify the way genes are passed down in different parts of an organism's DNA, which makes it easier to measure the genetic value and make improvements through breeding. Even though using marker-assisted selection (MAS) has shown some successes, it is challenging to use it to improve traits that have a lot of variation and are difficult to measure accurately. The way we typically study traits that are controlled by many different genes is not very effective. We use a method called biparental mating, where two parents are selected to mate and their offspring are studied to understand the genetic factors influencing a trait. However, this method is not well suited for traits that are controlled by many genes. In these cases, we use a different method called marker-assisted selection (MAS), which looks for specific markers in the DNA that are linked to genes controlling the trait. The idea of using genomic selection is suggested as a better way to improve traits that can be measured or quantified. This technique uses all the tiny markers in a living organism's genes to figure out important genetic information. These markers are found all over the genes. By studying them, scientists can estimate how good an organism is for breeding purposes. By using accurate marker scores and a fast genotyping method, genomic selection can predict traits more accurately and capture a larger portion of the genetic variation caused by small genetic factors. The genomic selection method relies on two different groups of data - a training group and a validation group. It is used in a group of people that is different from the original group used to estimate the marker effects.

CONCLUSION

Quantitative genetics is the study of traits that are not controlled by just a few genes. It is based on math models and methods, but with some strong assumptions. Although these methods may not be considered realistic, they are still effective. Studying genes with lots of information is giving us more knowledge about how traits are structured. However, we still don't know everything, as there are many genes that don't explain all the differences in traits, even if there are many of them. So, new ways of predicting success in breeding programs rely on using numbers and genetic information. The results of lab experiments show that we can make long-lasting improvements in genetics. There is a lot of variation in genes in natural populations, but we need more accurate information about how different traits are related to each other and how they affect a living thing's survival and reproduction. New approaches that use summary statistics and predictions, instead of looking at individual genes, are probably going to be more popular for a while longer.

REFERENCES:

[1] P. Gienapp, S. Fior, F. Guillaume, J. R. Lasky, V. L. Sork, and K. Csilléry, “Genomic Quantitative Genetics to Study Evolution in the Wild,” *Trends in Ecology and Evolution*. 2017. doi: 10.1016/j.tree.2017.09.004.

[2] M. Zhu, M. Yu, and S. Zhao, "Understanding quantitative genetics in the systems biology era," *International Journal of Biological Sciences*. 2009. doi: 10.7150/ijbs.5.161.

[3] D. A. Roff, "A centennial celebration for quantitative genetics," *Evolution*. 2007. doi: 10.1111/j.1558-5646.2007.00100.x.

[4] P. Bijma, "The quantitative genetics of indirect genetic effects: A selective review of modelling issues," *Heredity*. 2014. doi: 10.1038/hdy.2013.15.

[5] R. M. Nelson, M. E. Pettersson, and Ö. Carlborg, "A century after Fisher: Time for a new paradigm in quantitative genetics," *Trends in Genetics*. 2013. doi: 10.1016/j.tig.2013.09.006.

[6] N. A. Dochtermann and D. A. Roff, "Applying a quantitative genetics framework to behavioural syndrome research," *Philosophical Transactions of the Royal Society B: Biological Sciences*. 2010. doi: 10.1098/rstb.2010.0129.

[7] C. P. Klingenberg, "Quantitative genetics of geometric shape: Heritability and the pitfalls of the univariate approach," *Evolution*. 2003. doi: 10.1111/j.0014-3820.2003.tb00230.x.

[8] R. De Vlaming and P. J. F. Groenen, "The current and future use of ridge regression for prediction in quantitative genetics," *BioMed Research International*. 2015. doi: 10.1155/2015/143712.

[9] F. Pelletier, D. Réale, J. Watters, E. H. Boakes, and D. Garant, "Value of captive populations for quantitative genetics research," *Trends in Ecology and Evolution*. 2009. doi: 10.1016/j.tree.2008.11.013.

[10] S. M. Leal, "Genetics and Analysis of Quantitative Traits," *Am. J. Hum. Genet.*, 2001, doi: 10.1086/318209.

CHAPTER 9

GENETIC MAPPING: PRINCIPLE, METHODS AND FUTURE DEVELOPMENT

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ABSTRACT:

Gene mapping is the process of determining the order in which genes are located on a chromosome. Genetic maps are unique for each species and are made up of genetic markers or genes, showing the distance between each marker. The distances are figured out by how often chromosome crossovers happen when cells divide, rather than where they are placed on the chromosome. There are existing detailed maps of genetic markers for plant genes. New technologies are helping us make similar maps for other species. Genetic maps are important for finding disease genes or trait locations, which is also called linkage mapping. Combining genetic mapping and gene mapping with advanced sequencing techniques has been a successful approach in plants genetic research. Studying the genetic makeup of complex traits is a continuous challenge for scientists who specialize in genetics. There are two ways to find out about genes, linkage mapping and association mapping. These approaches have been used successfully to understand complex traits in many crops. Both of these methods find specific genetic traits by looking for connections between markers and traits. The main difference between the two methods is the type of group being studied, which affects how detailed and accurate the mapping is. In this review, we will talk about the benefits and drawbacks of family-based mapping and natural population-based mapping. We will compare these methods to linkage mapping and association mapping. After that, we explain ways to improve the ability to detect things and make computers work faster using statistics. We also mention new areas of research, such as studying the genetics of crops on a big scale. In the age of new sequencing technology, it is important to have good planning for studying groups of people, using advanced statistical methods, and accurately observing traits to make the most of efficient genetic testing.

KEYWORDS:

Genes, Genetic, Markers, Mapping, Specific Gene, Traits.

INTRODUCTION

The goal of genetic mapping is to find specific areas of DNA that cause differences in how living things look or behave naturally. There are two main methods used for genetic mapping in plants: linkage mapping and association or linkage disequilibrium mapping. Linkage mapping is a way to map genes that relies on how they get reshuffled during the creation of a group of plants or animals for the purpose of mapping. In the last 20 years, scientists have often used linkage mapping to study different plants, and they have identified and studied many specific genetic regions called QTL. However, linkage mapping has some drawbacks. It has a lower ability to accurately determine the location of genes, fewer variations in genetic material, and it takes longer to carry out. Association mapping is a way to find out how different traits or characteristics in plants or animals are passed down from one generation to the next. It looks at the recombination of genes that happened in the past and has been

building up over many generations. This helps us see these traits or characteristics more clearly and allows us to find out how many different variations of genes are present. After scientists learned how to study diseases in people, they started using the same method to study diseases in crops. Since it was first used on plants, association mapping has become more popular in genetic research. Because sequencing technologies have become cheaper, researchers have been able to conduct association mapping in various plants, including popular crops like rice, maize, wheat, soybean, barley, sorghum, potato, and tomato [1], [2].

The main difference between association and linkage mapping is whether the genes are studied in a group of individuals or in families. However, these two methods have a consistent plan for finding molecular markers connected to QTL. As we enter the time of completely sequencing genomes, the distinction between the two methods will go away. Genetic mapping can be divided into two types: family-based mapping and natural population-based mapping. Family-based mapping is done by studying the offspring of crosses between two or more parents. Natural population-based mapping is done in populations where the relationships between individuals are not known. In this review, we discuss how scientists study complex traits in families and groups of individuals. We also explain the statistical methods used for genetic mapping and discuss the current trends and future directions in crop genetics. Family-based mapping is a method used to identify and locate specific genes or genetic traits within a family. It involves studying the inheritance patterns of these traits across multiple generations of relatives. This helps researchers understand how these genes are passed down and can aid in identifying the specific gene responsible for a certain trait or disease [3], [4].

The main thing to do in family-based mapping is to make groups of family members, like parents and children, to use for experiments. These groups can be made in different ways, like by having two parents and their children, or by breeding back to one of the parents. Some other ways to make these groups are by using special plant cells or by making plants with mixed-up genes. The typical process of biparental mapping involves gathering parents that have different traits, choosing markers that can tell the difference between the parents, creating a group with a mix of the parents' DNA, studying the DNA and physical characteristics of the group, finding regions of the DNA that are related to the traits using a suitable statistical method. The ability to detect QTL (quantitative trait loci) is influenced by the strength of the QTL, how common the alleles are, and the kind and number of individuals in the group being studied. Mapping by two parents has been found to be helpful in growing crops. The main problem with a biparental population is that not many genetic changes happen during its growth, so the regions where specific genes are located can only be narrowed down to 10-20 units of measure called centimorgans (cM). Moreover, finding QTL in two-parent populations relies on the differences in physical characteristics of the two parents, which might only represent a small portion of the genetic differences in the species [5], [6].

Scientists have made multiparent mapping populations to solve the problems of using just two parent populations. The variety of genes from different parents helps create a group of individuals with many different physical traits. This makes it easier to study and identify specific genes that are responsible for certain traits. Two experimental designs called NAM and MAGIC are becoming more popular. NAM and MAGIC are used with multiparent populations. In recent years, researchers have studied various crops using a method called multiparent mapping. This method allows them to detect differences in traits without having to create a special group for testing. It provides detailed information and can be used in many different types of crops. This approach has been used successfully since 2001 in over 12

crops. The main steps in natural population-based mapping are shown in the figure. This means they first gather a group of plants, including important types, older types, wild types, and rare types. Then, they study the physical traits of these plants to estimate how those traits are determined by genetics. They also look at how closely related the plants are to each other. They also look at how the plants are influenced by their environment and how they are related to each other. Finally, they use mathematics to see how the genetics of the plants are related to their physical traits. Further tests, such as changing genes or studying how genes are used, need to be done to confirm the findings. The accuracy of mapping in natural populations depends on how closely related individuals are, the size of the sample, and the frequency of less commonly occurring gene variants. Single-marker analysis is a method used to find specific genes in a certain type of population. It looks at the differences in physical traits between groups of individuals with different genes, without considering how close these genes are on a map [7], [8].

It does this by using maximum-likelihood parameter estimation, which is an efficient way to calculate these values. A simpler way to compute IM is using a regression version developed by Haley and Knott. IM assumes that there is only one genetic factor that influences the quantitative traits we are interested in, and it doesn't consider the impacts from other genetic factors. But we know that traits are often controlled by multiple genes, so to accurately find the regions of the genome that are responsible for these traits, we need to analyze multiple genes at the same time. To overcome the problem of IM, CIM uses a method called composite interval mapping. It combines two techniques, regression and interval mapping, to find a smaller group of markers that can help to reduce errors by accounting for the effects of linked QTL. The main question in CIM is how to pick the right marker characteristics, and we can answer this question by using stepwise regression or preliminary interval mapping. The inclusive composite interval mapping (ICIM) is an improved algorithm that reduces errors from sampling and eliminates the need for selecting marker covariates, while still keeping all the benefits of the previous CIM technique. Multiple interval mapping (MIM) is a method that improves upon interval mapping by allowing the identification of multiple QTL (quantitative trait loci) with greater accuracy and power. It also allows for the estimation of multiple QTL simultaneously, considering the interactions between them. There are many software programs available for biparental mapping, such as QTL Cartographer, QTL Network, and R/qtl. Even though many detailed marker maps have been made, QTL interval mapping is still helpful today because not all crop species have complete genomic information and highly detailed marker maps.

Studying populations with multiple parents is similar to studying populations with two parents, but it is not easy to determine which parent the alleles come from based on the markers observed. Because of this, the methods used for mapping with two parents cannot be applied to mapping with multiple parents. Xu came up with a way to study a four-way cross design. He used regression analysis and found that both fixed and random models work well for mapping multiple parents. He found the values for the fixed model using a special algorithm called iteratively reweighted least squares, which separates the effect of QTL from other factors. A special program has been created for finding genetic markers in groups of organisms with multiple parents. For instance, if we assume that the QTL positions are identical in all instances, according to Jourjon et al. The MCQTL software was created to help with QTL mapping in multicross designs using CIM and iterative QTL mapping. can write about what the authors of this research paper did Researchers created a computer program called HAPPY that helps find specific traits in mice populations. HAPPY uses a hidden Markov model (HMM) to guess the chance that a gene comes from each original strain. This package was helpful in finding QTL in MAGIC populations of A. Thaliana is a

plant. Another computer program, called mpMap, was created to find genetic regions that are linked to trait differences in plants that have been bred from multiple parents. This program can also analyze data using a statistical model called linear mixed models.

However, all of the methods mentioned can only be used for instant messaging (IM) and computer-integrated manufacturing (CIM). Whole-genome average interval mapping (WGAIM) is a method for finding genetic traits in populations with two parents. It looks at all marker information at the same time and has been found to be better than another method called CIM. For populations with multiple parents, the method has been changed a bit and is called MPWGAIM. This new method uses the likelihood of inheriting certain genes from their original parents. You can figure out the chances of a founder using either three-point or HMM methods. In a MLM system called MPWGAIM, it is possible to represent the organization of a population and simplify the model using a forward selection approach. But it is still expensive if there are a lot of markers and QTL included. Wei and Xu created a new way to study MAGIC populations. They assumed that the effects of genes on traits are random and follow a normal distribution. They found that this approach is better and faster than the previous method called MPWGAIM. Bayesian techniques, like hierarchical Bayes and empirical Bayes, are helpful in multiparent mapping because they can handle uncertainty when determining how alleles are inherited from multiple parents. However, using a Bayesian method with a lot of data points requires a lot of computer processing power because it needs to frequently use a technique called Monte Carlo sampling. Many ways have been created for mapping with many parents, but they haven't fully used these special designs yet. We still need statistical methods that can quickly and accurately find QTL in populations with multiple parents.

DISCUSSION

Quantitative trait loci (QTL) can be matched to specific areas on chromosomes, thanks to the identification of molecular signposts. In the past, studies had limited and far apart indicators, which made it difficult to accurately estimate the effects of QTL. Using more markers and more samples can make it easier to find and locate QTLs (genes that control specific traits). We are currently in a situation where we have to remove markers that have the same information. A genome can have lots of SNPs (small changes in DNA) and doesn't need interval mapping anymore. You can look at one marker at a time and check the whole genome for important markers (Figure 1). This kind of marker analysis is not difficult to do on a computer. However, this method is not correct if there are multiple QTL in the genome. Different ways of doing a one-dimensional scan have been suggested, like the composite-interval mapping (CIM) procedure. The aim of CIM is to find one important genetic factor that can be detected and, at the same time, to adjust for the impact of other important genetic factors that can also be detected, as well as the effects of multiple genes that cannot be detected. The CIM method has a new problem on choosing the cofactors to capture background information. The outcomes are not reliable because choosing different markers as cofactors can give different outcomes.

A more effective method for mapping QTLs is the multiple-interval mapping (MIM) procedure. In the year 1999, a study was conducted where all possible intervals were considered as potential regions and then an analysis called stepwise regression was used to identify the specific intervals associated with QTLs. When there are too many markers close together, the number of sections can become very large. This makes it difficult to use the method because it requires a lot of computing power. So, the original MIM method is not the best choice anymore. If someone only looks at a certain amount of positions in the genome, the model size will stay the same even if there are more markers. In this situation, using

markers that are close together will make it more certain to determine the genotype at the positions being studied. The size of the model will get bigger as more positions are evaluated. However, the size of the model cannot be bigger than the sample size. This is because of the limitation of the maximum-likelihood method. One big benefit of the Bayesian method is that we can choose a prior distribution for QTL parameters, like QTL effects, that gives us useful information. An informative prior makes big estimated effects less important and reduces the estimated QTL effects towards zero. Using shrinkage priors helps in dealing with models that have a lot of dimensions. The MCMC-based Bayesian methods face a difficulty when the model dimension changes because it often takes a long time for the Markov chains to reach stability. Also, it becomes more difficult to process lots of markers when there are millions of them [9], [10].

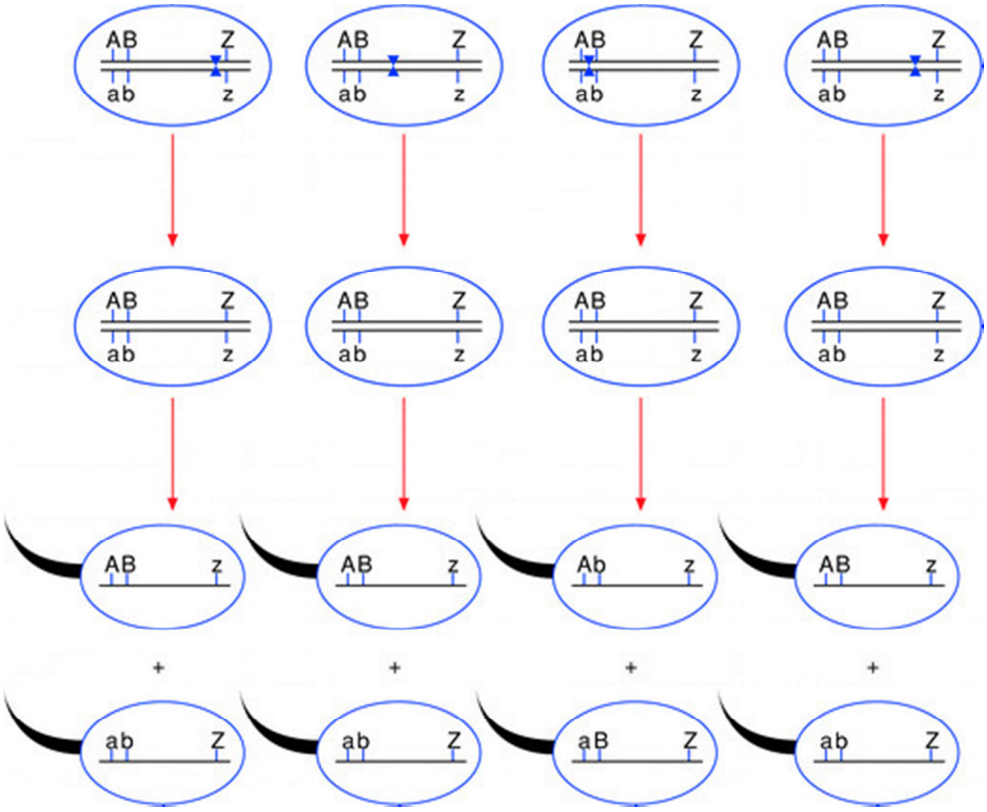

Figure 1: Representing the basics of the genetic mapping [Research Gate. Net].

Their goal was not to find QTL, but instead to predict breeding values using a new method called marker-assisted selection. Their work was not well noticed until recently when small markers that can hold a lot of information became commonly used in many living things. It was discovered that this concept can be used in experiments where we cross different lines to detect specific genes that influence certain traits, as well as in selecting desired traits using genetic information. In genomic selection, we look at all positions in the genes, but we make some changes so that they are a certain distance apart. This helps us better understand how the genes are connected to each other. They used special markers to find where DNA is exchanged during reproduction and then turned this data into simpler information. All markers in a bin are arranged in the same way.

Each trash bin is thought of as a fresh marker. After collecting the bin data, QTL mapping is done. Using the bin data for genetic mapping is easier than using the original markers because there are fewer bins, which are always a finite amount compared to the original

markers. The size of the model can be much smaller, but it still contains the same amount of information. This is a different way to decrease the number of dimensions in data, without needing complex statistical methods. The analysis of bin data can be more helpful than the analysis of original markers in finding interactions between genes (G × G) and between genes and the environment (G × E).

Maize (Zea mays) is a very popular crop that is grown all over the world. It is used for food, animal feed, and making bioenergy. Corn production in the United States has become eight times larger than it was 80 years ago. Half of this increase is because breeders have carefully chosen the best corn plants to grow.

Even though the main goal is to produce a lot of grain per hectare, the increase in grain yield is mostly because there are more plants packed together. In the past, there were fewer maize plants in an area of land in the United States. Now, there are more maize plants in the same area. This increase in plant density has made maize plants stand more upright. This information comes from a study done in 2005. The best way that plants grow in high-density fields of maize can help them get more sunlight, use energy better, and avoid falling over. This means that they can produce a lot of grain.

In simple words, plant architecture in maize means how the aboveground parts of the plant are arranged. This is about many different traits of plants, like how tall they are, how high their ears are, how many branches their tassels have, how long their main tassel is, how long and wide their leaves are, the angle of their middle leaves, and the number of leaves above and below their ears. When breeders choose plants that are close together, the leaves and tassels of the plants grow larger and point upwards. This helps more sunlight reach the plants and they produce a lot of crops. The parts of a maize plant that are above the ground come from the main growing point of the plant and its flowers.

The way plants grow and develop explains why their different parts are often related to each other. Earlier research on the SAM has shown that several genes, particularly homeobox genes and phytohormone genes, work together to control how the meristem grows and changes. So, the way plants grow and look can be different and this is because of complex genes. Although it is still difficult to understand the genes responsible for these traits, studying the genetic makeup of plant structures will help in improving crop yield when plants are densely planted. This will ultimately lead to further advancements in corn productivity.

CONCLUSION

In simple words, genetic mapping means arranging markers in a certain order to show how close they are genetically. Creating a genetic map is not a new idea. The main goal is to better understand how genes work and choose the best genes for certain traits more easily. PCR-based markers are widely used in genetic mapping because they are easy to create from even small amounts of DNA. In the last ten years, the AFLP method was commonly used in plant genetic mapping because it was very effective at creating many molecular markers. SSRs became the popular choice of marker because they were found frequently in plant genomes, had high rates of mutation, provided accurate results, and were easy to use with automation. But with the advancements in SNP technology and the fact that we now have access to the complete genetic data of organisms, it is likely that SNP markers will become the preferred choice for studying and improving economically important crops.The CMap program helps to find and connect similar molecular markers in genetic maps. These programs were used successfully to make the first maps of wheat and barley. By combining groups of molecular markers, consensus maps increase the total number of markers that can be used for breeding plants and studying genetics. These maps connected the old genetic maps to the newer

marker maps. The marker maps are better for quickly screening plants in breeding programs. Consensus maps can also be used to analyze QTL. The MetaQTL method combines different genetic maps and QTL datasets using consensus models. The similarity within a species makes it more likely to find true genetic regions linked to traits by combining information from different genetic sources and multiple sets of traits data.

REFERENCES:

[1] D. Altshuler, M. J. Daly, and E. S. Lander, "Genetic mapping in human disease," *Science*. 2008. doi: 10.1126/science.1156409.

[2] Y. Xu, P. Li, Z. Yang, and C. Xu, "Genetic mapping of quantitative trait loci in crops," *Crop J.*, 2017, doi: 10.1016/j.cj.2016.06.003.

[3] S. Swinnen, J. M. Thevelein, and E. Nevoigt, "Genetic mapping of quantitative phenotypic traits in Saccharomyces cerevisiae," *FEMS Yeast Research*. 2012. doi: 10.1111/j.1567-1364.2011.00777.x.

[4] X. Song *et al.*, "Development of INDEL markers for genetic mapping based on whole genome resequencing in soybean," *G3 Genes, Genomes, Genet.*, 2015, doi: 10.1534/g3.115.022780.

[5] K. M. Gardner *et al.*, "Fast and cost-effective genetic mapping in apple using next-generation sequencing," *G3 Genes, Genomes, Genet.*, 2014, doi: 10.1534/g3.114.011023.

[6] E. L. Clark and D. P. Blake, "Genetic mapping and coccidial parasites: Past achievements and future prospects," *J. Biosci.*, 2012, doi: 10.1007/s12038-012-9251-1.

[7] G. V. James *et al.*, "User guide for mapping-by-sequencing in Arabidopsis," *Genome Biol.*, 2013, doi: 10.1186/gb-2013-14-6-r61.

[8] M. J. Sadhu, J. S. Bloom, L. Day, and L. Kruglyak, "CRISPR-directed mitotic recombination enables genetic mapping without crosses," *Science (80-.).*, 2016, doi: 10.1126/science.aaf5124.

[9] H. Bruining *et al.*, "Genetic mapping in mice reveals the involvement of Pcdh9 in long-term social and object recognition and sensorimotor development," *Biol. Psychiatry*, 2015, doi: 10.1016/j.biopsych.2015.01.017.

[10] R. Murayama, G. Akanuma, Y. Makino, H. Nanamiya, and F. Kawamura, "Spontaneous transformation and its use for genetic mapping in Bacillus subtilis," *Biosci. Biotechnol. Biochem.*, 2004, doi: 10.1271/bbb.68.1672.

CHAPTER 10

COINCIDENCE AND INTERFERENCE: BASICS OF THE GENE INHERITANCE

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ABSTRACT:

In some species, there are differences in how often chromosomes cross over during reproduction. These differences can occur at the whole chromosome level or in specific regions. In this study, we looked at how CO is distributed on Chromosome 4 of Arabidopsis thaliana in males and females during a specific type of cell division. We used detailed genetic maps created from a large group of plants to study this. We noticed big differences between the map lengths of males and females. The male map length was 88 cM and the female map length was 52 cM. This difference is very similar to the difference between the lengths of the synaptonemal complex in male and female cells. This was measured by labeling a component of the synaptonemal complex called ZYP1. Furthermore, the CO landscapes were noticeably distinct. Specifically, at both ends of the map, the rate of male CO was higher (up to four times the average), while the rate of female CO was either the same or even lower than the usual rate. This special material allowed us to study how CO interferes with Chromosome 4 during the process of making eggs and sperm in males and females. There is clear evidence that the amount of COs on each chromosome and the distances between them are not random. Interestingly, the amount of interference (measured by coincidence) changed a lot in different parts of the chromosome during male meiosis. This change was related to how close the crossing overs (COs) were to each other. This discovery is important for understanding how current models of CO interference are relevant.

KEYWORDS:

Coefficient coincidence, Distance, Double Crossovers, Genes, Map.

INTRODUCTION

In addition to telling us how far apart genes are, map distances also tell us how many different types of gametes are produced during reproduction. A map distance of 7. 5 mu is equivalent to a distance of 7. 5 units on the map. The information suggests that when an organism is heterozygous for two genes, about 7. 5% of the offspring's sex cells will have a different genetic combination compared to the parents. Double crossovers, which were discussed before, make map distances appear smaller than they actually are. If we imagine calculating the ratio of gametes with double recombination using probability, and then multiplying this ratio by the total number of offspring, we would get the predicted number of offspring with double crossovers. In reality, we see far fewer in the offspring that are produced. This is because the calculated number assumes that each crossover is not affected by any other crossovers. Crossovers on a chromosome can affect each other, meaning one crossover can interfere with or reduce the occurrence of other crossovers nearby. As a result, double crossovers happen less often than we would expect. The word interference is used to explain how much one crossover affects other crossovers in that specific area of the chromosome. The coefficient of coincidence is a measure used in cryptography to determine

how similar two sets of data are. It is calculated by comparing the frequency of certain patterns or characters in the data sets [1], [2].

We can make gene maps by doing some tests on parents that have different pairs of genes. One of the parents has different forms of the same gene. We use the results of these tests to figure out how often the genes recombine with each other. A two-point test cross is when we cross two genes to see what traits are passed on to the offspring. Let's look at an example that shows how we can map genes using recombination frequencies. First, we will examine two-point test crosses, and then we will study three-point test crosses, which are usually more precise. When independent assortment is happening, the chance of getting a specific combination is 50%. So, we can understand that genes q and r are found either on different chromosomes or are very far apart from each other on the same chromosome. So, they are thought to be part of different groups. This means that q and s are not connected, and q and t are also not connected. Now, the RF (recombination frequency) between r and s is 20%. This means that these genes are separated by 20 map units. The genes r and t are connected to each other, and they have a recombination frequency of 10%. To find out if gene t is 10 map units away. We measure the distance between genes r and s by looking at the space on either side of gene r. If t is 10 units of measurement. If you go left from r, the distance between t and s should be about the same as the distance between r and s plus the distance between s and t, which is 20 m. u + Ten monetary units. Rewrite this text using simpler words: 30 m. u [Please note that the distance given is an estimate because of the occurrence of "double crossovers. "] Another option is that gene t is on the right side of gene r. If this is the case, the distance between gene t and s will be shorter, about 20 m. u 10 mu means 10 monetary units [3], [4].

10 monetary units (m. u) From the information presented, it seems clear that the R. F The percentage between s and t is 28%, which means that t is to the left of r. So, when we want to make a genetic map, we can use a two-point test cross. This is a way to figure out how far apart genes are on the map by looking at how often they recombine. We also said that when double crossovers happen, it makes the map distances seem smaller than they actually are. Basically, if the recombinant frequency is high, it is not a very accurate measure of map distance. Actually, when we calculate map units from larger recombinant frequencies, they are actually smaller compared to map units calculated from smaller recombinant frequencies. Usually, when studying recombination between three connected genetic regions, the combined frequency of recombination within the middle regions is higher than the frequency of recombination between the outer regions. The most accurate way to measure map distance is by adding up the distances of smaller sections.. 23 which shows "a double crossover" where only the middle gene is changed in these situations, compared to the outcome from the process of single crossovers. A genetic map is a diagram showing different spots on a chromosome. A very effective way to study three genes at the same time is by doing a three-point cross. This method helps us figure out the order and distance between these genes by only doing one experiment [5], [6].

This is very helpful when finding the position of a new mutation that is not known in relation to two other positions that are already known. The main plan is similar to the experiment we talked about before, except this time we crossbreed two different pure breeding lines that have different genetic patterns. This produces an individual with a mix of the two patterns at three different spots in their genes (called a trihybrid). We then mate this individual with another one that has a completely different genetic pattern. By doing this, we can figure out how often the genes swap places and recombine, among the three spots in the genes. A Punnett square helps us figure out what might happen when we do a test cross. If we cross a

trihybrid (having three different traits) with another organism, it can create eight different types of cells that can combine to create eight different looks in the babies. The next thing to do is to figure out if the alleles are mixed up or the same as the parent gametes. You can do this by looking at only two locations at once and comparing them to the parent's sex cells. In this example, the parents of the trihybrid have specific genetic traits. One parent has traits a, B and c, and the other parent has traits A, b and C. This means that the genetic material they contribute to their offspring will be aBc and AbC respectively. Now, if you compare two locations at the same time, you can find out if they are different or the same. For instance, the baby in the top row of was created from gamete aBC. When we compare loci A and B, we can see that it matches one of the gametes from the parents. This means it is from the parent and not a new combination. When we look at A and C, we can tell that it doesn't match either parent. This means it is a combination of different traits. The same idea applies when we are comparing B and C.

DISCUSSION

Besides telling us how far apart genes are, map distances also tell us how many different types of reproductive cells are produced in a cross. A map distance of 7. 5 mu means that on the map, the distance between two places is 7. 5 units The space between two genes shows that 7. 5% of the cells produced by an organism that has different traits for both genes will be different from the original forms. Double crossovers, like I said before, make the map distances seem smaller than they really are. If we calculate the number of double recombinant gametes using probability and then multiply it by the total number of offspring, we'll get the expected number of offspring with double crossovers. In truth, we see much fewer in the offspring. This is because the number we calculated assumes that each crossover is not affected by the others. Crossovers on a chromosome can affect each other, meaning that one crossover may prevent other crossovers from happening nearby. As a result, double crossovers happen less often than anticipated. The word "interference" is used to explain how much one crossover affects or disrupts other crossovers in a specific area of the chromosome. We can figure out the interference by using this equation: Interference = 1 minus the coefficient of coincidence. The coefficient of coincidence can be found by using this equation: Coefficient of coincidence = Number of observed double crossovers divided by the number of expected double crossovers [7], [8].

Once we know the different classes of offspring, and we know which pairs of genes come from the parents and which are mixed up, we can calculate how often recombination happens for each pair of genes separately. This is similar to what we did before in our dihybrid cross in Chapter 18. Then, we can use these numbers to create the map by putting the loci with the biggest RF on the edges. However, please keep in mind that in the three-point cross, if we add up the distances between A and B, and A and C, it is less than the distance between B and C. This happens because there are two crossovers between B and C that were not noticed when we only looked at the data for B and C together. We can easily count some of these double crossovers and use them to calculate the distance between B and C on the map. Here's how we do it. However, there may be other events where two genetic changes happen at the same time that we haven't noticed yet. For example, changes between points A&B or A&C. Scientists who study genetics have created different math techniques to try to fix problems that happen when mapping experiments involve a double crossover. As scientists discover more genes, they can create a more accurate map of our genetic information. Then, when we find a new gene, we can figure out where it is by comparing it to genes that we already know the location of. To map a gene, only two types of alleles are required: a normal allele and a

changed allele. Now that we understand what the map looks like, we can explain how frequently each type of offspring occurs.

Parental gametes (AbC and aBc) are created when there are no exchanges or swaps between two alleles. Since we know that all three genes are connected, we can expect this frequency to be quite high, similar to what we observed in the example mentioned earlier. There are special cells called gametes that are made from a combination of two different forms of a gene (aBC, Abc, ABC, and abc) through a process called crossover. Single crossover events happen more often. They are more likely to occur between loci B and A because they are 25 cM apart, which is a greater distance compared to the 10 cM distance between loci A and C. So, we hope to see more mixed sperm and egg cells with the first one. Lastly, there are mixed gametes created by double crossover events (ABc and abC). It is not common for three genes to undergo double crossovers like this. Therefore, we do not anticipate a high number of offspring with these changed gametes. The frequencies we observed from this cross match what we were anticipating. In the example, all the genes are connected, but some are more connected than others (A and C are more connected than A and B). When you select three genes to map, it may not always happen like this. Sometimes, all your genes will be connected. Sometimes, you can have two genes that are connected and one gene that is not connected. And sometimes, they may all not be connected. Just like we did before, if you compare the number of offspring with certain characteristics, you can figure out if the genes in the trihybrid are connected or not [7], [9].

If all three genes are not connected to each other, we would predict that they will separate independently from one another and produce an equal number of all possible offspring types. In simpler terms, if everything is connected like the example, there will be lots of different combinations of traits from each parent, and sometimes you might see new combinations if just one trait gets mixed up. Rare gene combinations resulting from two instances of genetic recombination will be uncommon. The number of each will be different depending on whether the linked genes are at the same distance from each other or if one pair is more connected than the other. If two genes are connected and one gene is not connected, the following is true. Like the previous example, we will use the same parent cells (AbC and aBc), but this time we will assume that the genes A and C are connected, while gene B is not connected. In this situation, when two things are connected together, there will be more instances of one type and less instances of another type. Whether or not parent B is present or absent does not matter in this situation, as it is not connected and will be distributed separately.

By including a third gene, we now have multiple variations of materials produced through crossing over. The picture below displays different things that can be made by combining different parts together (Figure 1). If we were to do a test with F1, we would predict an equal ratio of 1: 1:1:1: 1:1:1:1. Just like with the two-point analyses mentioned earlier, if the ratio is different from what was expected, it means that linkage is happening. The easiest way to understand how to analyze three-point test cross data is to look at an example. We will use the made-up example of genes A, B, and C. We start by breeding individuals with two sets of genes each (AABBCC) with individuals that have two sets of different genes each (aabbcc). Then, the F1 is crossed with an individual that has the traits aabbcc. We will use this information to figure out the order of genes and how close they are to each other. Like with the two-point data, we will look at the F1 gamete makeup. The easiest way to fix these problems is to create a step-by-step plan. First, figure out which of the genotypes are the parents' genotypes. The most common genotypes are the ones inherited from the parents. The table shows that the ABC and abc genotypes were the original genotypes.

First, we have to figure out the sequence of the genes. Once we know the parents' genetic makeup, we combine that information with the data from the double-crossover. The gametes that have two crossovers always occur less often. From the table, it can be seen that the ABc and abC genotypes occur the least often. The next important thing to know is that during a double-crossover event, the middle allele is switched from one sister chromatid to the other. This means that the gene in the middle is put together with genes from its neighboring chromosomes. From the table, we can determine that the C gene should be in the middle because the weaker c gene is now located on the same chromosome as the A and B genes. Meanwhile, the stronger C gene is on the same chromosome as the weaker a and b genes.

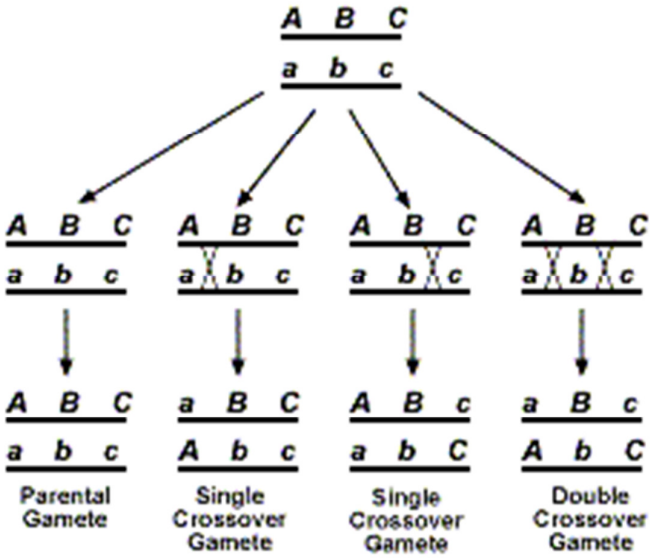

Figure 1: Representing the overview about the parental and recombinants genes derived from three points crosses [World Press.Com].

Now that we know the order of the genes is ACB, we can figure out how far apart A and C are, and how far apart C and B are. The linkage distance is found by dividing the total number of gametes that have undergone recombination by the total number of gametes. This is the same method we used before for the two-point analyses. What's new is that we have to think about the double-crossover events as well. For these calculations, we include the double-crossovers when measuring the distances between intervals. The distance between genes A and C is 17. 9 centimorgans This is calculated by adding up the percentages of the recombination between genes A and C (81, 85, 5, and 8) and then multiplying by 100 and dividing by 1000. Similarly, the distance between genes C and B is 7. 0 centimorgans Now, we will attempt a problem using the same ideas we used in the previous example, but this time it will involve Drosophila. The table below shows the results we will study.

The genotypes that are most common are the types passed down from the father. These are the genotypes: v cv+ ct+ and v+ cv ct. What is different in our first three-point cross is that one parent didn't have all the dominant genes and the other parent didn't have all the recessive genes. To figure out the order of genes, we need to have information about the genes of the parents as well as the genes that have undergone double crossovers. The double-crossover geneotypes are the least common ones. These are the different types of genes: v, cv+, ct, and v+, cv, ct+. We can figure out the order by asking which parental allele is not connected to the same two parental alleles as in the original parental cross in the double-crossover genotypes. In the first double crossover, the ct allele is now connected to the v and cv+

alleles. These are two alleles that were not initially connected to the ct allele in the original cross. So, ct is in the middle and the gene order is v ct cv.

Distance calculation using speed and time. This distance is calculated by multiplying the sum of 89, 94, 3, and 5 by 100, then dividing by 1448. The result is 13. 2 centimorgans The calculation of the distance between two points in a coordinate system. This is how the distance is calculated: take the sum of 45, 40, 3, and 5, then divide it by 1448. Multiply the result by 100 to get the distance, which is 6. 4 centimorgans Three-point crosses can also be used to measure how often crossover events happen in a specific area of a chromosome. Specifically, the number of double crossovers can show if interference happens. The idea is that if we know how often two sections of a chromosome exchange genetic information, we can predict how often two different sections will exchange information at the same time. In the example mentioned earlier, the recombination frequency between genes v and ct was 0. 132, and the recombination frequency between ct and cv was 0. 064 So, based on our calculations, we would expect to find 0. 84% of double recombinants. With 1448 samples, there are 12 double recombinants. We found only 8. To measure interference, we start by figuring out the coefficient of coincidence (c. oc), which is the ratio of the number of observed double recombinant events to the number of double recombinants we would expect. Interference is found by subtracting the current outcome from one. Here is the formula: The interference value for the v ct cvdata is 33%, which is calculated by multiplying 8 divided by 12 by 100. Usually, interference values are between 0 and 1. Values smaller than one show that there is interference happening in this area of the chromosome.

Before gene cloning was invented, scientists used to identify genes by studying the changes that occurred when the genes were changed or mutated. This scientific method of finding the genes behind unusual traits is easiest to do in organisms that reproduce quickly and can be easily modified genetically, like bacteria, yeasts, nematode worms, and fruit flies. Sometimes, we can find spontaneous mutants by looking at a lot of organisms. But it is easier and faster to find mutants by causing mutations with agents that harm DNA. By using chemicals or radiation, we can create many different changes in organisms. We can then look at these changes to find any specific problems we are interested in. Another way to cause changes in an organism's genetic material, instead of using chemicals or radiation, is through a process called insertional mutagenesis. The added DNA has a specific sequence that helps to find and copy the gene that was changed. In fruit flies, the use of the P element helps researchers learn how genes work. Transposable elements are genetic blocks that can move around within a genome. Genetic engineering methods have also been used to create changes in bacteria, yeast, and the flowering plant Arabidopsis. Retroviruses, which can insert their DNA into the genes of the host, have been used to change genes in zebrafish and mice.

The snapdragon, also known as Antirrhinum, has a mutation that affects where certain parts of its genetic material are placed. A change in one gene that makes a protein which controls things, leads to leafy branches growing instead of flowers. The mutation changes cells to have a characteristic that would be better suited for a different situation. These types of studies work well for understanding how things work in worms and flies, but it's more difficult to study gene function in humans. Humans don't reproduce quickly and are not exposed to mutagens on purpose. Also, if a person has a major problem in a necessary function, like copying DNA, they would die even before they are born. There are two ways to study human genes. First, studying simpler organisms can give us important information about genes and processes in humans because these things have stayed mostly the same over time. After identifying the relevant human genes, they can be further examined in cells grown in a laboratory. Also, there are mutations in humans that are not deadly. These mutations can

affect specific tissues, like lysosomes or cell-surface receptors, and have occurred naturally in the population. Studying the traits and characteristics of the people affected and their cells grown in labs has helped us learn a lot about how human cells work. While it is not common, when mutations happen, they are easily found because individuals with mutations will go to the doctor for special treatment.

CONCLUSION

Right before crossing over happens, a process called synapsis takes place where homologous chromosomes pair up and create a structure called chiasma. Chiasma is a structure that looks like the letter "X" and it is formed when non-sister strands of DNA from pairs of similar chromosomes come together. This happens before the exchange of genetic material between the DNA strands. When a chiasma is close to another chiasma, it can block the formation of the second chiasma. This means that double crossovers happen less often than expected. This thing is called interference. Interference means that when crossover happens in one spot on a chromosome, it lowers the chances of crossover happening in another spot. Let's say we have three genes, x-y-z, lined up in a chromosome. If the recombination between x and y is 20% and between y and z is 40%, and if they don't affect each other, then the recombination between x and z should be 8%. But, in truth, the amount is always less because of interference. The amount of interference is different in each place. If there is no double crossover happening, it means that interference is at its maximum level of 100%. If double crossover happens as expected, it means there was no interference. Coincidence means the same thing as interference but is the opposite of it. If double crossover happens as often as expected, then coincidence would be 100%. But if there is no double crossover at all, then coincidence would be 0%. In the example shown before, the expected result of a double crossover happening is 0. 08 If we see double crossover happening 2% of the time, then the coefficient of coincidence is 25%. When two things happen at the same time a lot, they will bother each other less. While it is expected that positive interference is common, some bacteria and fungi also experience negative interference. In these living things, when recombination happens in one spot, it increases the chances of recombination taking place in another spot.

REFERENCES:

[1] N. S. Bisht, E. K. Sharma, and H. C. Kandpal, “Effects of thermal light source properties in two-photon subwavelength coincidence interference experiments,” *Optik (Stuttg).*, 2011, doi: 10.1016/j.ijleo.2009.11.019.

[2] Y. jian Cai and S. yao Zhu, “Coincidence imaging and interference with coherent Gaussian beams,” *Front. Phys. China*, 2006, doi: 10.1007/s11467-006-0002-3.

[3] Y. Zhang, S. Prabhakar, C. Rosales-Guzmán, F. S. Roux, E. Karimi, and A. Forbes, “Hong-Ou-Mandel interference of entangled Hermite-Gauss modes,” *Phys. Rev. A*, 2016, doi: 10.1103/PhysRevA.94.033855.

[4] S. Shabbir, M. Swillo, and G. Björk, “Arbitrary interference curves by coincidence detection: theory and experiment,” *J. Opt. Soc. Am. A*, 2013, doi: 10.1364/josaa.30.001921.

[5] M. Tillmann *et al.*, “Generalized multiphoton quantum interference,” *Phys. Rev. X*, 2015, doi: 10.1103/PhysRevX.5.041015.

[6] X. X. Jin, C. F. Chen, G. Lin, F. Zhao, Y. J. Huang, and L. T. Yang, "Study on Method of Coincidence Summing Correction in γ-ray Cascade Radiation," *Yuanzineng Kexue Jishu/Atomic Energy Sci. Technol.*, 2017, doi: 10.7538/yzk.2017.51.06.1077.

[7] J. Drouaud *et al.*, "Sex-specific crossover distributions and variations in interference level along Arabidopsis thaliana chromosome 4," *PLoS Genet.*, 2007, doi: 10.1371/journal.pgen.0030106.

[8] V. Leong, S. Kosen, B. Srivathsan, G. K. Gulati, A. Cerè, and C. Kurtsiefer, "Hong-Ou-Mandel interference between triggered and heralded single photons from separate atomic systems," *Phys. Rev. A - At. Mol. Opt. Phys.*, 2015, doi: 10.1103/PhysRevA.91.063829.

[9] A. V. Burlakov *et al.*, "Interference effects in spontaneous two-photon parametric scattering from two macroscopic regions," *Phys. Rev. A - At. Mol. Opt. Phys.*, 1997, doi: 10.1103/PhysRevA.56.3214.

CHAPTER 11

PHYSICAL MAPPING OF GENE AND CHROMOSOMES: ADVANCED METHDOLOGY

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ABSTRACT:

Physical and molecular mapping techniques are methods that help to locate specific parts of DNA, such as genes. These techniques can be used to quickly and effectively improve crops through marker-assisted selection in breeding programs. They are also useful for manipulating genes to enhance crop improvement. We know that using nuclear techniques like gamma irradiation is really important for making crops better all around the world. These techniques can improve things like nutrition and the plants' ability to handle tough conditions. These tools allow us to visualize specific molecules or genetic material within a cell, allowing us to better understand how cells work and how they may be affected by disease. By labeling molecules or genetic material with fluorescent dyes, we can track their movement within cells, observe their interactions with other molecules, and determine their location within the cell. This information helps researchers uncover new insights into cell processes and can aid in the diagnosis and treatment of diseases. methods to study fish, genetic testing (e. g FISH, GISH, TUNEL test, COMET assay), detailed mapping and identifying traits (e. g high-resolution mapping and genotyping). Restriction Fragment Length Polymorphism (RFLP), Amplified Fragment Length Polymorphism (AFLP), Simple Sequence Repeats (SSR), Sequence Characterized Amplified Region (SCAR), Inter Simple Sequence Repeats (ISSR), Insertion site-based polymorphism (IRAP), Expressed Sequence Tag (EST), Single Nucleotide Polymorphism (SNP), and technology that analyzes DNA sequences in millions of base pairs (megabase technology). BAC libraries with deep genome coverage help with studying chromosomes and detecting changes caused by mutations.

KEYWORDS:

Chromosomes, DNA, Genes, Genomes, Mapping.

INTRODUCTION

The quality of a genetic map is determined by how many crossovers have been counted. Microorganisms are not greatly affected by this issue because they can be easily obtained in large quantities. This allows for a lot of studying to be done, which helps create a detailed genetic map where the markers are very close together. For instance, in 1990, when the Escherichia coli genome sequencing project started, there were over 1400 markers on the genetic map of this organism. These markers were spread out on average every 3. 3 kilobases This information was detailed enough to guide the sequencing program without needing a lot of physical mapping. Similarly, the Saccharomyces cerevisiae project was helped by a detailed genetic map (around 1150 genetic markers, about one marker per 10 kilobases). The issue with humans and many other living things is that it's difficult to have a lot of offspring. This means that there are only a small amount of times we can study the process of meiosis and the ability to analyze how genes are linked together is limited. This means that genes that are many thousands of base pairs apart may appear at the same position on the genetic map.

Genetic maps are not very precise. We briefly mentioned this idea. When we evaluated Sturtevant's belief that crossovers happen randomly on chromosomes. This idea is only partly right because the existence of recombination hotspots means that crossovers are more likely to happen in certain areas rather than in others. In 1992, researchers showed that this can greatly impact the accuracy of a genetic map. The publication by Oliver et al. in 1992 presented the genetic map of cerevisiae chromosome III. This map allowed for the first direct comparison between the genetic markers and their actual positions on the chromosome, which were determined through DNA sequencing [1], [2].

There were big differences, to the point where one pair of genes had been put in the wrong order by the genetic analysis. Remember that S. Cerevisiae is a type of organism, similar to a fruit fly, that scientists have studied a lot in terms of its genetic makeup. If the map of yeast genes is not precise, how accurate are the maps of genes in other organisms that have been studied with less detail. This means that before doing a lot of DNA sequencing, scientists need to double-check and add to a genetic map for most complex organisms, because there are two problems with using genetic mapping alone. Many different ways to map physical information have been created to tackle this issue, with the most significant one being: Restriction mapping is a technique that determines where certain enzymes called restriction endonucleases can cut a DNA molecule. It helps to identify the specific positions of these recognition sequences on the DNA. Fluorescent in situ hybridization (FISH) is a method where we use a probe with a marker to find where specific genes are located on chromosomes. STS mapping is a method where we locate the positions of short sequences in a genome by using PCR and hybridization analysis of genome fragments [3], [4].

Genetic mapping helps locate specific positions in the DNA where there are differences among individuals. However, this technique is limited because not all differences in DNA can be detected. Can we make the spots closer together on a genome map by finding the positions of some of the non-changing restriction sites in a different way. This is what restriction mapping does, but it can only be used on small DNA molecules. First, we will examine the method and then think about how it is important for genome mapping. The easiest way to create a restriction map is to compare the sizes of the pieces of DNA produced when it is cut by two different restriction enzymes that recognize different sequences. First, the DNA molecule is broken down with a specific enzyme, and then we use a technique called agarose gel electrophoresis to measure the sizes of the smaller pieces that result. Afterwards, the molecule is broken down using another enzyme and the smaller pieces are measured again in a jelly-like substance called agarose gel. The current results show us how many restriction sites each enzyme has, but they don't tell us where these sites are located in relation to each other. More information is gained by using both enzymes to cut the DNA molecule. You can fix the problem by going back to the starting DNA molecule and treating it again with BamHI. This time, you need to stop the digestion from finishing completely by doing things like incubating the reaction for a short time or using a not ideal temperature. This is called a partial restriction and results in a more complicated group of goods [5], [6].

Restriction mapping is a method used to figure out the order and positions of specific regions within a DNA molecule. The goal is to locate the EcoRI and BamHI sites in a straight DNA molecule that is 4. 9 kb long The top shows the outcomes of both single and double limitations. The sizes of the pieces obtained after double cutting allow two more. A partial restriction can give enough information to finish a map. But if there are many restriction sites, it becomes complicated because there are too many different fragments to think about. A different approach is easier because it lets us not pay attention to most of the pieces. This is done by adding a radioactive or other kind of marker to both ends of the starting DNA

molecule before doing a partial digestion. This means that some of the partially restricted products are not visible because they do not have a specific part at the end, so they cannot be spotted when looking at the gel for labeled products. The visible sizes of the partial restriction products help to locate unmapped sites in relation to the ends of the starting molecule.

Restriction mapping is limited by the size of the pieces that are created when DNA is cut by restriction enzymes. Creating restriction maps is simple when there are not many places where enzymes can cut. However, when there are more places where the DNA is cut, there will also be more pieces of DNA of different sizes that need to be measured and compared to create a map. Using computers to analyze situations can be helpful, but there are still issues that will eventually come up. At some point, there will be so many pieces in a digest that they will combine on the gel, making it more likely for one or more pieces to be measured incorrectly or not measured at all. If many small pieces are the same size, it could be difficult to put them together to make a clear map, even if you can figure out what each piece is.

Restriction mapping is mainly used for small molecules, not large ones. The biggest molecule that can be mapped depends on how often the restriction sites appear in it. In real-life situations, if a DNA molecule is shorter than 50 kb, it is usually possible to create a clear restriction map using certain enzymes that recognize six-nucleotide sequences. Fifty kilobytes is very small for bacterial or eukaryotic chromosomes, but it does work for some viral and organelle genomes. Restriction maps of whole-genomes have been helpful in guiding sequencing projects with these small molecules. Restriction maps are very helpful when studying bacterial or eukaryotic genomic DNA that has been cloned. They are especially useful when the cloned fragments are smaller than 50 kb. This is because a detailed restriction map can be created before sequencing the cloned region. Can we use restriction analysis to map entire genomes larger than 50 kb. The answer is mostly yes, but there are some limits to restriction mapping. However, you can make it a bit easier by selecting enzymes that don't cut the target DNA molecule very often. These "rare cutters" can be divided into two groups.

Enzymes that can identify and bind to sequences of seven or eight nucleotides. Some restriction enzymes can cut DNA at certain patterns of seven or eight nucleotides. Two examples are SapI and SgfI. SapI is made up of the letters G-C-T-C-T-T-C. SgfI is made up of the letters G-C-G-A-T-C-G-C. The enzymes made of seven nucleotides are likely to cut a DNA molecule with equal amounts of the nucleotides G and C about once every 16,384 base pairs. The enzymes with eight nucleotides should cut the DNA once every 65,536 base pairs. These numbers compare to 4096 base pairs for enzymes with six nucleotides like BamHI and EcoRI. Researchers often use seven- or eight-nucleotide cutters for restriction mapping of large molecules. However, this method is not very effective because there are not many enzymes of this type that are currently known.

Enzymes that have special patterns in the DNA they recognize and these patterns are not common in the target DNA. The DNA strands in the genome have patterns and some of them are lacking certain specific sequences. For instance, the sequence 5'-CG-3' is not common in human DNA because cells in our body have a special enzyme that puts a methyl group on carbon 5 of the C molecule in this sequence. The resulting 5-methylcytosine is not very stable and often changes into thymine through a process called deamination. As humans evolved over time, many of the 5'-CG-3' sequences in our genetic code changed to 5'-TG-3'. Restriction enzymes can cut human DNA less often when they identify a site that has 5'-CG-3'. For example, SmaI cuts human DNA every 78,000 base pairs on average, while BssHII cuts every 390,000 base pairs. Keep in mind that NotI, which is a tool that cuts DNA, also targets specific sequences of DNA called "5'-CG-3'". In human DNA, it cuts very rarely,

happening only about once every 10 million base pairs. The sequence 5'-CG-3' is not common in human DNA because the C molecule gets modified with a methyl group, and then it turns into T. Using rare cutters can increase the potential of restriction mapping. We cannot currently create restriction maps for the genomes of animals and plants. However, we can use this technique with larger cloned pieces of DNA and with smaller DNA molecules from organisms like bacteria and fungi [3], [7].

If you use a rare cutter, you may need a special type of agarose gel electrophoresis to examine the restriction fragments that are produced. This is because when a DNA molecule is longer, it does not migrate at the same speed as a shorter DNA molecule in an electrophoresis gel. The resolution of the DNA molecules also decreases as they become longer. This means that you cannot separate molecules longer than approximately 50 kb because they all move together as one slow band on a gel. To separate them, instead of using a simple electric field, we need to use a more complicated field in traditional gel electrophoresis. Orthogonal field alternation gel electrophoresis (OFAGE) is a technique that uses two pairs of electrodes that are positioned at a 45° angle to the gel. The DNA molecules continue to move through the gel, but each change in the field makes them line up again. Smaller molecules can move faster than larger molecules through the gel. The final outcome is that very long molecules, which couldn't be separated with normal gel electrophoresis, can now be distinguished apart. Other techniques that are similar to CHEF are FIGE.

DISCUSSION

If we can understand how genes work, how they interact with each other, and where they are located, we can better change and control them. This will directly result in making crops better. Before, different types of abnormal chromosome numbers, genetic variations, and gene mutations (some happening by chance and others caused by exposure to radiation or chemicals) have made crop production happen faster. Molecular markers help create maps of characteristics in different plants by identifying and tracking changes in their genetic material. On the other hand, when we physically map traits that add value, we have a specific target in mind. We have made significant progress in changing and improving genes and gene groups in crops by combining traditional breeding methods with molecular marker and cytogenetic techniques. But, plant breeders still can't use a lot of the genomic information that's available to them. They need to understand genome structures and variations better, both natural and caused by humans, so they can manipulate genes more effectively. After studying the entire genetic makeup of various plants, we now have a lot of information about their DNA sequences. However, it is difficult to interpret these sequences and understand how genes work together, which is slowing down advancements in using them for plant breeding. Finding the exact location of genetic markers on chromosomes and genomes is very important for physical mapping. fluorescent dyes and genetic tools have greatly advanced our ability to study the structure and function of cells.

Regular and non-traditional methods of using agarose gel to separate DNA fragments. In regular agarose gel electrophoresis, the electrodes are put on opposite sides of the gel and the DNA moves towards the positive electrode. Bigger molecules We can use different methods instead of electrophoresis to locate restriction sites in DNA molecules. In optical mapping, we use a microscope to directly find the locations of restriction sites on DNA molecules that have been cut. First, the DNA needs to be put on a glass slide in a way where the strands are stretched out and not stuck together in a bunch. There are two methods of doing this: using gel stretching or molecular combing. To make gel-stretched DNA fibers, scientists take chromosomal DNA and mix it with melted agarose. Then, they place the mixture on a microscope slide. When the gel gets cold and hard, the DNA molecules stretch out. To use

gel stretching in optical mapping, a restriction enzyme is first applied to the microscope slide where the melted agarose is placed. The enzyme is not working right now because it needs magnesium ions to be able to work. After the gel becomes hard, it is cleaned with a solution that has magnesium chloride. This solution makes the restriction enzyme start working. A special type of dye called fluorescent dye, like DAPI, is used to color the DNA. This helps us see the DNA fibers when we use a powerful microscope that can detect fluorescence. The places where the DNA is cut become spaces when the DNA becomes less stretched, allowing the positions of the cuts to be noted [8], [9].

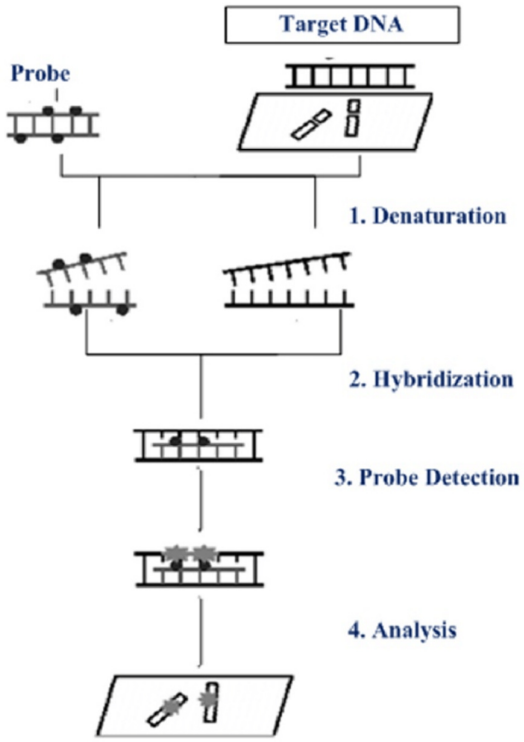

Figure 1: Representing the steps involved in the fluorescent in situ hybridization technique DNA [Research Gate. Net].

The optical mapping method mentioned earlier is related to another physical mapping procedure called FISH. In simple terms, FISH is like optical mapping because it allows us to see where a marker is on a chromosome or a long piece of DNA. In optical mapping, a marker is a specific spot on a DNA strand that can be seen as a break or gap in the strand when it is stretched out. In FISH, the marker is a specific part of DNA that can be seen using a special fluorescent probe. In situ hybridization is a method of studying chromosomes by using a labeled DNA molecule to examine them. The place on the chromosome where hybridization happens gives us information about where the DNA sequence used as the probe is located on the map. To make the method work, the DNA in the chromosome needs to be made into a single strand by breaking the pairs that hold the double helix together. The chromosomal DNA can only bind with the probe at that time. The usual way to change the structure of chromosomal DNA without damaging the shape of the chromosome is to make the DNA dry on a glass slide and then put a chemical called formamide on it.

In the past, researchers used a radioactive label in their in situ hybridization experiments. However, this method didn't work well because it was challenging to get both sensitivity and resolution with a radioactive label, which are two important things needed for successful in

situ hybridization. Being sensitive means that the radioactive label needs to have a lot of energy when it releases radiation (like 32P). However, if the radioactive label has too much energy, it causes the signal to spread out and makes it hard to see clearly. A better quality picture is possible if a low-energy radiolabel like 3H is used. However, these labels are not very sensitive, so it takes a long time to capture the image. This results in a lot of noise in the background and makes it hard to see the real information. These issues were fixed in the late 1980s with the creation of special markers that glow but don't use radiation to do so. These labels are very good at detecting things and showing them clearly, and they are great for studying things in their natural environment. Researchers have created special labels that glow in different colors. These labels can be used to attach a variety of probes to a single chromosome (Figure 1). By doing this, scientists can see where each probe attaches to the chromosome and make a map of their locations. To make the probes more sensitive, they need to be labeled with a lot of information. In the past, this usually meant using long DNA molecules, like cloned DNA fragments that are at least 40 kilobases (kb) in size. This rule is not as important anymore because we have found ways to create lots of labels on smaller molecules [10], [11].

When creating a physical map, a cloned DNA fragment can be seen as a marker. However, using clones as markers adds another dimension because the cloned DNA is used to determine the DNA sequence. Mapping the positions of clones helps connect a genome map with its DNA sequence. If the probe is a long piece of DNA, there may be a problem, especially with complex organisms, because it may have repetitive DNA sequences. This means it could attach to many spots on the chromosomes, not just the exact spot it's supposed to match. To lower the non-specific mixing of DNA, the probe is combined with DNA that doesn't have a label from the organism being studied. all the DNA present in the nucleus of a cell) or it can include additional DNA found in other parts of the cell, such as in the mitochondria. Using a smaller part of the genome that has more repeat sequences is preferable. The idea is that the DNA without labels sticks to the repetitive DNA sequences in the probe, stopping them from working. This makes the following process of hybridization only happens with the special sequences. This means that non-specific hybridization is decreased or completely eliminated.

To create a detailed physical map of a big genome, we ideally require a mapping process that is fast, not difficult to do, and provides accurate results. Neither of the two methods we have talked about - restriction mapping and FISH - fulfills these criteria. Restriction mapping is quick, simple, and gives lots of information, but it can't be used for large genomes. FISH is a method that can be used on big genes, and there is a version called fiber-FISH that provides detailed information. However, FISH is not easy to do and it takes a long time to gather data. In one experiment, only about three or four markers can be mapped. If we want to have detailed physical maps, we need a stronger method. Currently, the most effective way to make maps of big genomes is using STS mapping. This technique has produced the most detailed maps so far. A sequence tagged site, also known as STS, is a small piece of DNA that is easy to identify and only appears once in the chromosome or genome being researched. It is usually around 100 to 500 base pairs long. To create a map of STSs, we require a group of DNA pieces that overlap with each other. These pieces can come from one specific chromosome or from the entire genome. In the example given in Figure 5. 31, a group of chromosome pieces was made.

Each point on the chromosome was included about five times in the collection. To create the map, we collect information about which fragments contain certain markers (STSs). Hybridization analysis can be done, but PCR is usually preferred because it is faster and

easier to automate (Figure 2). The likelihood of two STSs being on the same fragment relies on how close they are to each other i n the genome. If they are very near each other, they will most likely always be on the same part. If they are farther apart, sometimes they will be on the same part and sometimes they won't. This information can be used to figure out how far apart two points are, like how we find out distances on a map. In linkage analysis, we calculate a map distance by looking at how often crossovers happen between two markers. The STS mapping is similar, but instead of measuring the distance between markers, it looks at how often breaks occur between them.

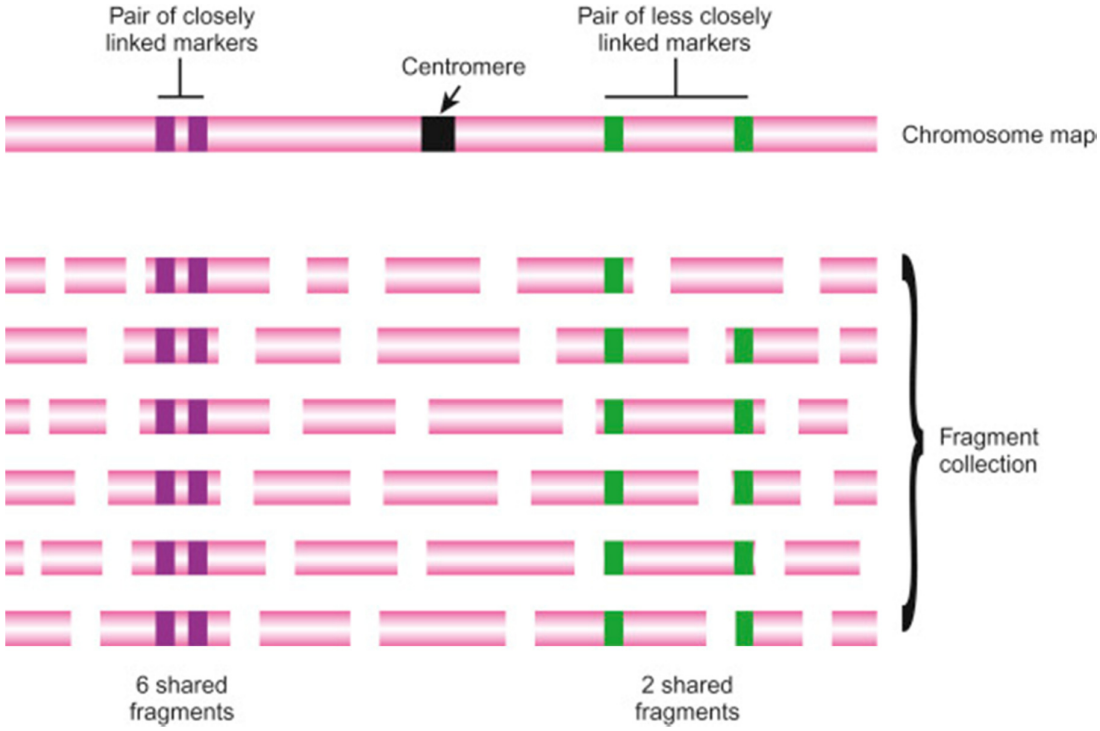

Figure 2: Representing the overview about the sequence tagged site (sts) mapping [Research Gate. Net].

The second part of an STS mapping process involves collecting small pieces of DNA that cover the entire chromosome or genome under investigation. The collection can be called the mapping reagent. Currently, there are two ways to make it: as a clone library or as a group of radiation hybrids. First, we will talk about radiation hybrids. A radiation hybrid is a small cell from a rodent that has pieces of chromosomes from another organism in it. The invention of this technology started in the 1970s. Scientists found out that when human cells are exposed to high levels of X-rays, the chromosomes inside them break into small pieces. This treatment kills human cells, but the broken pieces of chromosomes can be passed on if the affected cells are combined with healthy hamster or other rodent cells. Fusion can be made to happen either by using a chemical called polyethylene glycol or by being exposed to the Sendai virus. Not all of the cells in hamsters take in pieces of chromosomes, so we need a way to find the hybrids. The normal method of selecting cells involves using a type of cells from hamsters that cannot produce two specific enzymes called thymidine kinase and hypoxanthine phosphoribosyl transferase. If the cells are missing either of these enzymes, they will die when grown in a special mixture known as HAT medium, which contains hypoxanthine, aminopterin, and thymidine.

After the joining of cells, they are put into a liquid called HAT medium. The cells that are growing are a mix of hamster cells and human DNA. These cells have genes for enzymes called TK and HPRT, which are made by the cells themselves. This allows the cells to grow in a specific environment. The treatment makes cells with a mix of human and hamster DNA pieces. Usually, the pieces are 5–10 Mb big, and each cell has pieces that make up 15–35% of the human genome. A group of cells is called a radiation hybrid panel. It can be used to help map and locate specific DNA sequences. However, it can only be used for this purpose if the PCR test used to identify the DNA sequences doesn't also amplify the same DNA sequences in hamsters.

Another kind of radiation hybrid panel can be made if the DNA from only one human chromosome is used. This panel is made using a cell line from a different type of rodent hybrid, not a human cell line. Scientists who study genes have created several types of cells from rodents that contain one human chromosome in their nucleus and can be grown and studied consistently. If a specific type of cell is exposed to radiation and combined with cells from hamsters, the resulting hybrid cells from the hamsters will have pieces of either human or mouse chromosomes, or a combination of both. Those that have human DNA can be found by testing with a specific sequence called Alu, which is found in the human genome about once every 4 kilobases. Only cells that have human DNA will react with Alu probes. This allows us to get rid of the not important hybrids with mice DNA and focus on finding the cells that have fragments of human chromosomes for STS mapping.

Initially, scientists used chromosome-specific panels to map the human genome using radiation hybrid mapping. They believed that it would be easier to map a single chromosome compared to the entire genome, so they used fewer hybrids for this purpose. A detailed map of a single human chromosome needs about 100-200 hybrids. This is the maximum number that can be easily managed in a PCR screening program. However, whole-genome and single-chromosome panels are made in different ways. The whole-genome panel involves exposing only human DNA to radiation, while the single-chromosome panel needs a mouse cell with mostly mouse DNA and less human DNA to be exposed to radiation. This means that the amount of human DNA in each hybrid is much less in a single-chromosome panel compared to a whole-genome panel. This means that it is now possible to map the entire human genome using less than 100 whole-genome radiation hybrids. Therefore, mapping the entire genome is not any more difficult than mapping individual chromosomes. When people understood this, whole-genome radiation hybrids became very important in the mapping part of the Human Genome Project. Scientists are using whole-genome libraries to find out the locations of specific genes in the genomes of different animals, like mammals, zebra fish, and chickens.

Before we can sequence a genome, we need to divide it or the chromosomes into small pieces and copy each of these pieces into a special container that can hold large amounts of DNA. This creates a group of DNA pieces called a clone library. These pieces are several hundred kilobases in size on average. In addition to helping with sequencing, this clone library can also be used to map genes in STS analysis. Just like radiation hybrid panels, a clone library can be created from genomic DNA. This library represents the whole genome. Alternatively, a chromosome-specific library can be made if the DNA used comes from only one type of chromosome. This is possible because we can separate individual chromosomes using a method called flow cytometry. To do this technique, cells that are dividing are gently opened up so that we can get a mixture of whole chromosomes. Next, the chromosomes are colored using a special dye that glows under certain light. The bigger the chromosome, the more dye it can hold and the brighter it will glow. The chromosome preparation is made less

concentrated and put through a small hole. This creates small drops, with each drop containing only one chromosome.

The droplets go through a machine that checks how much brightness they have. This helps the machine find the droplets with the specific chromosome it is looking for. An electrical charge is used to separate specific drops that contain the desired chromosome from all the other drops. What happens if two different chromosomes have about the same sizes, for example, human chromosomes 21 and 22. Normally, they can be distinguished if a dye is used that doesn't attach randomly to DNA, but prefers specific regions rich in AT or GC. Hoechst and chromomycin A3 are both types of dyes.

Two chromosomes that are the same size usually have different GC contents. Because of this, we can tell them apart by using dyes that bind to specific parts of DNA called AT or GC. Clone libraries are better than radiation hybrid panels for STS mapping, because they have one major benefit. This means that each individual clone can give their DNA to be sequenced. The information from STS analysis helps create a physical map. This data can also be used to figure out which clones have overlapping DNA, which helps in building a clone contig. For more ways to build clone contigs. This group of overlapping clones can be used as the starting material for a long, continuous DNA sequence. The STS data can then be used to accurately place this sequence on the physical map. If the STSs have SSLPs that have been found using genetic linkage analysis, then the DNA sequence, physical map, and genetic map can all be combined.

CONCLUSION

The goal of improving the quality of food and industrial crops is important for both developed and developing countries. This is done to make the crops more valuable and increase the income of farmers. It is widely known that using mutagens can be a helpful way to change different traits of crops, including their quality. Using nuclear techniques like gamma irradiation to cause mutations is a useful way to create new types of crops with better qualities. However, we need to have a better understanding of how genes work and how they interact with each other and their location in order to be able to control and manipulate them more effectively, which will ultimately help improve our crops. Physical mapping, molecular markers, and molecular cytogenetic techniques are tools that can help us identify and select beneficial genes and gene combinations in breeding programs. These tools are particularly useful in mutation breeding. This research project was done to study how to find and understand mutated genes that affect the quality of crops. It was part of a bigger project that aims to identify, understand, and transfer mutated genes. The main goals were to help countries speed up their crop breeding programs by using physical mapping and other genetic methods. They also aimed to study and use induced mutants to make crops better quality.

REFERENCES:

[1] S. Cho, D. F. Garvin, and G. J. Muehlbauer, “Transcriptome analysis and physical mapping of barley genes in wheat-barley chromosome addition lines,” *Genetics*, 2006, doi: 10.1534/genetics.105.049908.

[2] I. V. Kirov, K. Van Laere, and L. I. Khrustaleva, “High resolution physical mapping of single gene fragments on pachytene chromosome 4 and 7 of Rosa,” *BMC Genet.*, 2015, doi: 10.1186/s12863-015-0233-9.

[3] P. Zhang, W. Li, J. Fellers, B. Friebe, and B. S. Gill, "BAC-FISH in wheat identifies chromosome landmarks consisting of different types of transposable elements," *Chromosoma*. 2004. doi: 10.1007/s00412-004-0273-9.

[4] A. Bolino *et al.*, "Genetic refinement and physical mapping of the CMT4B gene on chromosome 11q22," *Genomics*, 2000, doi: 10.1006/geno.1999.6088.

[5] P. Gregor *et al.*, "Genetic and physical mapping of the GLUR5 glutamate receptor gene on human chromosome 21," *Hum. Genet.*, 1994, doi: 10.1007/BF00211029.

[6] S. Taketa, M. Choda, R. Ohashi, M. Ichii, and K. Takeda, "Molecular and physical mapping of a barley gene on chromosome arm 1HL that causes sterility in hybrids with wheat," *Genome*, 2002, doi: 10.1139/g02-024.

[7] P. Zhang, W. Li, B. Friebe, and B. S. Gill, "Simultaneous painting of three genomes in hexaploid wheat by BAC-FISH," *Genome*, 2004, doi: 10.1139/G04-042.

[8] C. L. Rodríguez Delgado, P. D. Waters, C. Gilbert, T. J. Robinson, and J. A. M. Graves, "Physical mapping of the elephant X chromosome: Conservation of gene order over 105 million years," *Chromosom. Res.*, 2009, doi: 10.1007/s10577-009-9079-1.

[9] J. J. Coleman *et al.*, "The genome of Nectria haematococca: Contribution of supernumerary chromosomes to gene expansion," *PLoS Genet.*, 2009, doi: 10.1371/journal.pgen.1000618.

[10] W. Chen *et al.*, "Breakpoint analysis of balanced chromosome rearrangements by next-generation paired-end sequencing," *Eur. J. Hum. Genet.*, 2010, doi: 10.1038/ejhg.2009.211.

[11] C. L. Barker *et al.*, "Genetic and physical mapping of the grapevine powdery mildew resistance gene, Run1, using a bacterial artificial chromosome library," *Theor. Appl. Genet.*, 2005, doi: 10.1007/s00122-005-2030-8.

CHAPTER 12

GENE THEARPY: MODERN APPORACH FOR INTERGRATION GENE

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ABSTRACT:

Genetics helps us understand how genes work by studying what happens when genes don't function properly. Modern genetics and genomics need methods to change genes directly, by making small changes, removing parts, or adding new parts. The fact that we have information about the order of genes in many organisms means that we can quickly develop methods to understand how genes work in reverse. Until recently, scientists used a method called homologous recombination-dependent gene targeting (hrdGT) to target genes by introducing similar sequences. This method was commonly used for mammalian systems. But in more complex organisms like mammals and plants, when we add foreign DNA from outside, it usually gets placed in random spots in the genetic material by a process called illegitimate recombination. It's only rarely that we can find instances where the foreign DNA gets placed in specific positions. A new way was found to do precise reverse genetics. Specific chimeric oligonucleotides, which are made up of sections of both DNA and RNA, were discovered to cause changes in specific locations of tested genes in mammals. This new method, called chimeric oligonucleotide-dependent mismatch repair (cdMMR), has been tested on plants. In this issue of the Proceedings, there are two reports that talk about how tobacco and maize genomes have been successfully altered using this method.

KEYWORDS:

Cell, DNA, Genes, Plants, Thearpy.

INTRODUCTION

In gene therapy, a specific DNA or RNA sequence is given to a person using special methods. Scientists are looking forward to a time when they can treat patients by putting genes directly into their cells instead of using drugs or surgery. Gene therapy is a hopeful new way to treat many different illnesses like cancer, inherited diseases, and some viral infections. However, more research is needed to make sure that these methods are safe and work well. Right now, gene therapy is only used for diseases when other treatments are not effective. There are two main types of gene therapy: germline therapy and somatic gene therapy. Germline therapy refers to a type of medical treatment that involves modifying the genes in reproductive cells (eggs and sperm) to correct genetic disorders or enhance certain traits. It is a way to change the genetic make-up of future generations by directly altering their DNA [1], [2].

Germline therapy means changing the genes in sperm or egg cells. Germline therapy means making changes to the cells that will be passed down to future generations. It is done when a person is having a baby, and the modified cells join together to start forming a baby. After coming together, the zygote splits and gives the changed gene to all the other cells in the body as the baby grows. Germline therapy changes the genes that will be passed on to future generations. Some countries like Switzerland, Australia, and Germany have laws against

using germline therapy because they are concerned about the potential risks and unknown effects it may have on future generations. Even though it could potentially help with inherited diseases, these countries do not allow it. Germline therapy is very costly, which makes it even less usable in practice. Somatic gene therapy is a method used to treat genetic disorders by modifying the genes in a person's body cells [3], [4].

Somatic gene therapy is different from germline therapy because it puts the therapeutic DNA into body cells instead of reproductive cells. This means that the therapy only affects the person receiving it and does not get passed down to their children. The area of somatic gene therapy has fewer ethical problems than germline gene therapy. This method of treatment is still being worked on and is not fully developed yet. The first challenge in somatic gene therapy is making sure the new gene becomes a part of the person's DNA. Actually, if the modified gene is put in the wrong place in the DNA, it could cause disease instead of preventing it. Besides needing the desired gene to be active, we also need to control the activity of the new gene to avoid excessive expression, which could cause illness. Gene inhibition therapy is a type of treatment that aims to turn off or reduce the activity of specific genes in the body. This can be done to treat certain diseases or conditions caused by overactive or malfunctioning genes. By targeting and controlling the gene's activity, gene inhibition therapy may help to alleviate symptoms or even reverse the effects of the disease. In many diseases, fixing proteins to work normally again is not enough to cure the condition. They need to stop the mutant gene from being active [5], [6].

Gene inhibition therapy (also called gene silencing therapy) could be useful for treating conditions caused by dominant genetic disorders, some infectious diseases, or certain types of cancer. To treat genetic disorders that are passed down from parents, the gene responsible for the disorder can be stopped or controlled by adding another gene. The RNAi pathway allows us to use the body's own system to change how defective genes work. When we put in a gene that makes small interfering RNAs, which are short pieces of RNA that match up with the messenger RNA of a faulty gene, it can break down the messenger RNA of the faulty gene and stop it from being used to make proteins. If we stop the mutant gene from being active, the cell will be able to work normally. Gene-editing tools like CRISPR have helped to make gene therapy better. Gene editing is a way to change the genetic information by fixing or getting rid of a gene that is causing problems. The two methods mentioned above try to bring back the normal functions and activities of cells and change the unhealthy characteristics. But, for some illnesses like cancer, we want to get rid of the faulty cell instead of making it go back to normal [7], [8].

DISCUSSION

In the mouse system, using hrdGT is a normal and common practice. In the past, scientists discovered a way to change genes in stem cells before they became mice. This caused the creation of many mice with missing genes. The ability to target genes at a frequency of 10−2 made the work much easier. It also helped create unique phenotypes and animal models for human diseases that were very interesting. In comparison, even though academia and agriculture require it, the process of hrdGT in plants is very inefficient and hasn't improved much since it was first done. Homologous recombination in plants was studied in old research and also using molecular markers.. Ways to increase its occurrence by making the similar parts longer or using negative selection to focus on specific events did not have much impact. Most experiments had very low success rates of producing the desired changes, with frequencies ranging from one in 750 to one in 2,580 events. These frequencies were typically around 10^-4 or 10^-5. The reasons for these differences are not clear, but it could be because of the different methods used to transfer the DNA (either directly or using Agrobacterium),

the specific areas in the DNA where the transfer occurs, the type of tissue being targeted, and the specific plant species used in the experiment. Ways to make these frequencies better are to use plans to make the recombination target better or to make it stronger by giving enzymes from a type of bacteria called Escherichia coli (Figure 1). For example, the restriction enzyme I-SceI was used to create breaks in a specific gene in the tobacco genome. This caused a hrdGT frequency of up to 10−2 at that location. But, only specific sites can be chosen, which means this process doesn't work for all situations. Increasing the production of certain enzymes in prokaryotic cells that are involved in homologous recombination can greatly enhance the efficiency of recombination both within and between chromosomes. This improvement is significant, with recombination rates increasing by at least ten times. We can only know if this increase in recombination also affects gene targeting by doing more research [9], [10].

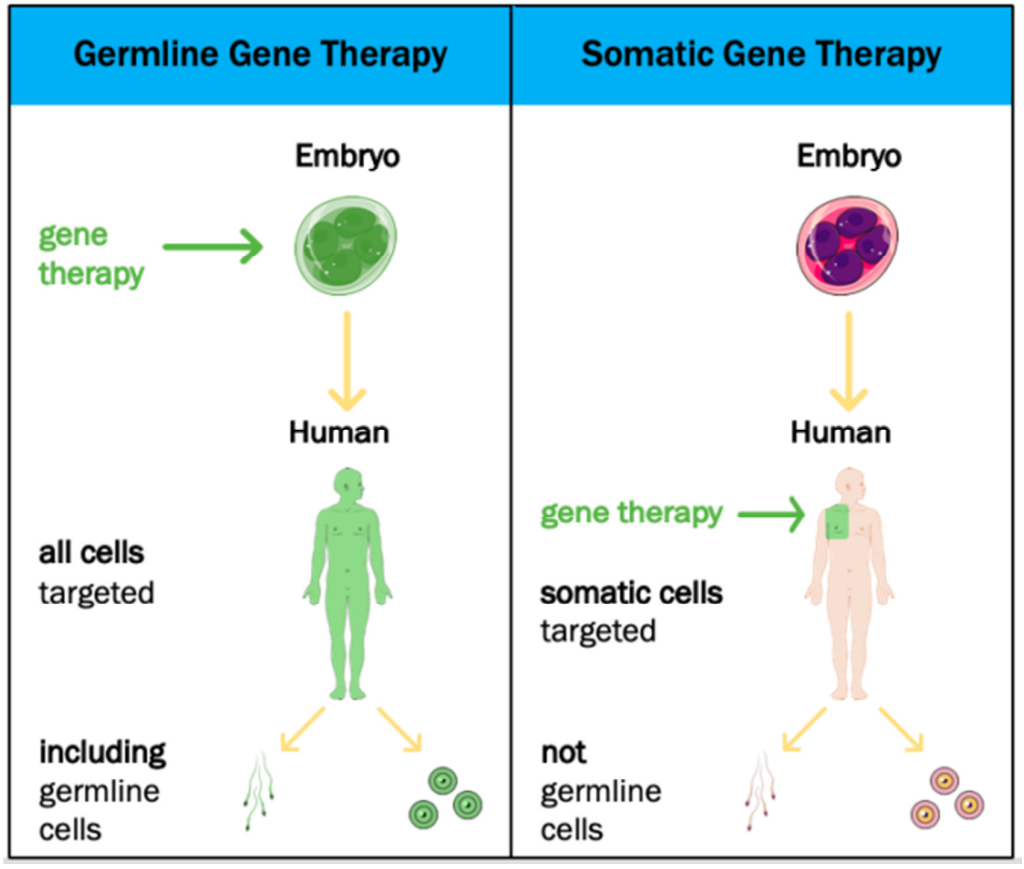

Figure 1: Representing the overview about the germline and somatic gene therapy [Test Book].

One interesting plant is the moss Physcomitrella patens, which is mostly made up of half the usual number of chromosomes. This living thing is similar to yeast because it usually adds foreign DNA by matching it to already existing DNA. Moss could be a great subject for studying basic cell processes. For example, scientists have successfully disabled a gene related to cell division in moss. cdMMR is a process in mammals where specific small pieces of genetic material called oligonucleotides are made. These oligonucleotides are made by combining DNA and RNA building blocks in a certain way. The "mutator" region has 5 building blocks called nucleotides that are opposite to the target but have a single change. This region is surrounded by two sections made of 10 nucleotides each called 2'-O-methyl-

RNA bridges which are also opposite to the target location. O-methylation helps protect the RNA from breaking down in cells. The loops of 4 T nucleotides are attached to a DNA sequence that matches the chimeric strand. A break in the lower part of the DNA allows the chimera to become intertwined with the target DNA. The "GC clamp" helps protect the end of the DNA from breaking down.

These COs were initially used to fix a mistake in co-transferred plasmids that contain instructions for making a specific protein in a type of animal cells called Chinese hamster ovary cells. Furthermore, researchers discovered that a faulty gene causing sickle cell anemia can be fixed very effectively using a mutator CO, despite other possible explanations put forth in references. Injecting a substance called COs complexed with lactosylated polyethylenimine into the tail veins of rats was found to effectively and accurately decrease the activity of the factor IX gene in liver cells affected by a mutation. This outcome shows that genes can be fixed in animal organs. Changes in genes caused by cdMMR have been proven to stay the same in multiple cell generations. This was demonstrated when pigmentation was restored by specifically reversing a certain gene called tyrosinase. All the reported cases showed that mutation was effective, except for a few cases mentioned in the note added in the reference. In simpler terms, it appears that only specific parts of the text were modified. Also, it was important that the mutagenic oligonucleotide had a mixture of different parts. Targeted gene repair in mammals is starting to become more common, but it is still not a regular occurrence [11], [12]. Human gene therapy focuses on specific tissues in the body, while generating mutant mice and plants requires changing every cell in the organism. This is useful for studying genes and potentially creating new products in plants. Plant transformation is mostly done by changing the genetic material of cells and then growing new plants from those cells. In the two reports included in this publication, we are using well-known methods to test if cdMMR can be applied. These methods involve transforming, screening/selection, and sometimes regenerating samples.

We used a type of tobacco cells and cultured maize cells, along with immature embryos, as the tissues we wanted to study. In both researches, they focused on a gene that produces the first enzyme for making branched amino acids. This gene is called acetolactate synthase (ALS) in tobacco and acetohydroxy acid synthase (AHAS) in maize. Changes to specific amino acids in this protein have been proven to make plants able to resist the effects of imidazoline and sulfonylurea herbicides. Scientists created COs that were changed in some way. Delivering COs to plant cells is harder than delivering them to animal cells because the mutagenic oligonucleotide has to pass through the tough plant cell wall, in addition to the cell membrane. So, tiny particles were shot at the COs to introduce them. The success rates of regrowing chlorsulfurone-resistant tobacco plants were 10 to 20 times higher than normal. For maize plants resistant to imazethapyr and chlorsulfurone, the success rates were about 4 and 15 times higher than normal, respectively. This means that for maize, the efficiency of bombardment is very low, with only several out of every million cells being affected. However, if the cells receive an oligonucleotide, the efficiency is higher, with about 1 out of every 10,000 cells being affected. These frequencies are okay for choosing specific changes in plants, but they are much lower compared to the frequencies seen in mammalian cells.

But, it was a surprise when they looked closely at the sequences. They thought that using two different COs in the tobacco ALS gene would cause a change in CCA, which codes for proline, to either CAA, which codes for glutamine, or CTA, which codes for leucine. However, when plant cells were treated with the first CO, the CCA changed to ACA. In the experiment with the second CO, TCA was discovered, which codes for a different amino acid. It seems like the repair was moved from the second to the first position in the codon.

However, it seems that the observed changes in the sequence are because of the attacks by the two specific COs. This is supported by the fact that other control treatments, such as using a nonspecific oligonucleotide or a specific DNA-only oligonucleotide, did not show any significant changes in resistance. The study of maize clones found a good result: Out of 40 clones that were tested, 34 of them had the expected change in the AHAS gene. However, in 6 clones, there were different changes in the targeted codon and the codon before it. In the other position we wanted to hit, only 2 out of 12 events were accurate. But in the other situations we talked about, the targeted building blocks were changed in various ways.

Both studies also did experiments to focus on a genetically modified but nonworking green glowing protein gene. A change in the gene in tobacco was reversed, but the details about the specific changes and how often they occurred were not provided. The number of cells containing the GFP gene in maize was between 2 in every 10,000,000 cells and 1. 6 in every 1,000,000 cells. The number of cells that received the oligonucleotide and contained the GFP gene was between 1. 5 in every 10,000 cells and 1. 1 in every 1,000 cells. The plants that glow green could be grown again. One of them grew up and had babies with the GFP gene separated in the correct way. The repair was in the right place, but not on the right foundation. The things that happen are still not common and can only be seen with the help of a strong marker gene. However, the GFP assay system is beautiful and relatively easy to use. It can be used with the mutant GFP tobacco and maize plants that are already available. These plants will help to make the technique even better.

In simple words, chimeric oligonucleotide-mediated gene mutation can be like a gene conversion. New research looked at human cells and found that a protein called Msh2 is important for repairing mistakes in the DNA. When this protein was missing or reduced, the repair activity was also reduced. This shows that this process, called cdMMR, is a real repair process in mammals. That's why it's called cdMMR. The research suggests that in mammalian systems, changes in the target location happen more often through small changes in the DNA code rather than adding or removing larger sections. Msh2, an important part of mismatch repair, is in plants too. Mismatch repair has been shown to happen in a plant virus with a fake mismatch. So, it seems probable that the same actors are involved in mismatch repair in plants. However, it is not very well understood how the other mutations happened. Bringing the necessary tools to fix DNA or RNA at a specific location enables other tasks to be done on them.

The recent advances in using gene therapy in plants need to be compared with similar approaches in animals. The information about cdMMR in mammals, which is mentioned in the referenced publication, is summarized below. Compared to the new information on fixing plant cells, it is clear that gene correction in plants needs to be improved to be more efficient and precise. There are several important steps to reach cdMMR: delivering COs, aligning them to chromosomal sequences, recognizing mismatches, and getting rid of mismatches. Using different coats on packaging can help with fast delivery and protection from enzymes. Additionally, giving extra repair proteins to cells in need during delivery can improve the accuracy and effectiveness of the repair process. In addition, comparing cdMMR and hrdGT is interesting for a few reasons. If you want to make a certain change to a certain gene, cdMMR would be the best method if it is effective and specific. The methods used here are not currently able to detect specific changes in genes that are not able to be chosen. For hrdGT, the success rate of targeting was estimated to be between 0. 001% and 000001% of targeted events compared to non-targeted events.

The measure called cdMMR had a success rate of 10^-4 repaired events per cell when a repair-promoting oligonucleotide was used. These numbers cannot be compared directly to

how well homologous recombination processes work. This is because when using homologous recombination, not every cell that receives a targeting DNA unit will result in a successful integration. Only about one in every hundred cells may be successful. Both processes take a long time because of the current efficiencies. cdMMR is not expected to cause big changes in DNA, based on studies done on animals. hrdGT is currently the expert in making big changes to plants by replacing genes to create knockout plants. However, other kinds of changes are also possible in the future. To fix damaged genes in plants, we don't rely on a process called homologous recombination. In simple words: Mixing different parts like transposable elements and T-DNA will help deactivate or activate genes reliably in the future. Less reliable but potentially more effective is the accidental or intentional turning off of plant genes at the epigenetic level. We need to improve the methods to make lasting and inheritable changes in genes, so that we can replace changes caused by epigenetics. The findings published in this edition are a significant and challenging first step in moving in this direction. According to the suggestion, our data shows that we can use chimeric RNA/DNA molecules to test the poorly understood mismatch repair pathways in plants. There is something, but there is also something else.

CONCLUSION

With the progress of medical science, doctors have found many ways to treat dangerous illnesses. However, some diseases can be passed down through families and are not easily treatable. In some situations, gene therapy has been found to be helpful. This is a method where healthy genes are put into a person or unborn baby to help with sickness. Gene therapy is a way to change or swap out genes that aren't working properly or have changes in them. If you want to understand what gene therapy means, keep reading this article. The plant is mostly made up of somatic or stem cells. This method uses good genes to substitute bad ones. The therapy is used to treat the cells that are not working properly in someone who has a disease. Somatic cells are mostly not involved in reproduction. That means the benefits of this treatment will not be passed down to the next generation. Therefore, it is believed to be one of the safest ways to use gene therapy. This treatment focuses on the cells in the body that make eggs or sperm. In germline gene therapy, doctors put good genes into cells. But, this therapy can have an impact on future generations. So, in many places, this therapy can't be used very much. For instance, the European Union says no to this procedure.

REFERENCES:

[1] H. Lisa Li, T. Nakano, and A. Hotta, “Genetic correction using engineered nucleases for gene therapy applications,” *Development Growth and Differentiation*. 2014. doi: 10.1111/dgd.12107.

[2] A. S. Lewin and W. W. Hauswirth, “Ribozyme gene therapy: Applications for molecular medicine,” *Trends in Molecular Medicine*. 2001. doi: 10.1016/S1471-4914(01)01965-7.

[3] D. F. Swamy, S. S. R. Dessai, E. S. Barretto, and K. M. Dsouza, “Gene therapy applications in dentistry: A review,” *Journal of Clinical and Diagnostic Research*. 2017. doi: 10.7860/JCDR/2017/28150.10703.

[4] D. Wang and G. Gao, “State-of-the-art human gene therapy: Part II. gene therapy strategies and clinical applications,” *Discov. Med.*, 2014.

[5] X. Cai, S. Conley, and M. Naash, “Nanoparticle applications in ocular gene therapy,”

Vision Research. 2008. doi: 10.1016/j.visres.2007.07.012.

[6] A. Negrete and R. M. Kotin, "Strategies for manufacturing recombinant adeno-associated virus vectors for gene therapy applications exploiting baculovirus technology," *Briefings in Functional Genomics and Proteomics*. 2008. doi: 10.1093/bfgp/eln034.

[7] A. Strods *et al.*, "Preparation by alkaline treatment and detailed characterisation of empty hepatitis B virus core particles for vaccine and gene therapy applications," *Sci. Rep.*, 2015, doi: 10.1038/srep11639.

[8] S. Goverdhana *et al.*, "Regulatable gene expression systems for gene therapy applications: Progress and future challenges," *Molecular Therapy*. 2005. doi: 10.1016/j.ymthe.2005.03.022.

[9] Y. Seow and M. J. Wood, "Biological gene delivery vehicles: Beyond viral vectors," *Molecular Therapy*. 2009. doi: 10.1038/mt.2009.41.

[10] S. D. F., D. S.S.R., B. E.S., and D. K.M., "Gene therapy applications in dentistry: A review," *J. Clin. Diagnostic Res.*, 2017.

[11] M. Nakanishi and M. Otsu, "Development of Sendai Virus Vectors and their Potential Applications in Gene Therapy and Regenerative Medicine," *Curr. Gene Ther.*, 2012, doi: 10.2174/156652312802762518.

[12] K. Lundstrom, "Alphavirus Vectors for Gene Therapy Applications," *Curr. Gene Ther.*, 2006, doi: 10.2174/1566523013349039.

CHAPTER 13

SOMACLONAL VARIATION: APPLICATION IN PLANT CROP DEVELOPMENT

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ABSTRACT:

Somaclonal variation is the difference noticed in plants that have been created through plant tissue culture. Chromosomal rearrangements are a big reason for this difference. Somaclonal variation is when plants regenerated from callus show differences in their characteristics. There are different types of variations that can occur. They can be genotypic or phenotypic. If it's phenotypic, it can be caused by either genetics or epigenetics. Genetic changes can happen in different forms like changes in chromosome numbers, changes in chromosome structure, and changes in DNA sequence. Common epigenetic-related occurrences are the amplification of genes and the methylation of genes. If you can't see any changes in the way the plant looks and grows, you need to use different methods to check it. Somaclonal variation has both advantages and disadvantages. Somaclonal variation is when there is a lot of difference among individuals from plant cell cultures or adventitious shoots. This can be described as changes that happen to genes because of mutations in a laboratory or when cells are separated. Somaclonal variation is generally not wanted. Sometimes, somaclonal variation can create new types of plants. These plants might have pretty features or be able to fight off pests better.

KEYWORDS:

Genes, Genetic, Plants, Somaclonal, Variation.

INTRODUCTION

Micropropagation is a big achievement in using lab cultures for growing trees and fruit crops. One important thing to think about when growing perennial plants from micropropagation is making sure they stay genetically the same as the original plant. Different changes have been observed in plants that have been grown in a lab. These changes can happen in different ways, like how the plant looks, what it's made of, and on a very small level, like its chemistry. The economic impact of somaclonal variation in fruit crops and woody plants is very big because these plants have long lifespan. As a result, we should check how micropropagated plants grow in the field after they have been growing for a long time. The occurrence of somaclonal variation is something that worries any micropropagation system. To find somaclonal variants, researchers tried different ways to detect them. They looked at things like how the plants looked, analyzed the chromosomes, and used markers to study the plant's molecules and biochemicals. Moreover, researching somaclonal variation is significant for managing and possibly stopping it, in order to create plants that have the same genetic makeup. It is also useful for generating genetic diversity, which will help breeders enhance the genes of plants. Many studies have looked at somaclonal variation in plants that are not woody, but there have been very few studies on fruit crops that grow back every year in cooler climates. This chapter is a summary of different ways to identify changes in perennial fruit crops and collects information from various sources. It has been reported in

detail about recent progress in understanding and identifying changes in olive plants grown in a lab using pieces of nodes and somatic embryogenesis [1], [2].

Growing trees and fruits in a lab has been a big success in the business of plant cultivation. When growing perennial plants from micropropagation, it is important to keep the same genetic makeup as the original plant. In simpler terms, researchers have found that micropropagated plants can have different variations in their appearance, cells, chemicals, and molecules. The economic impact of somaclonal variation in fruit crops and woody plants is very big because they take a long time to grow. Therefore, we need to evaluate how micropropagated plants behave in the field after they have been growing for a long time as young plants. The presence of somaclonal variation is worrisome for any system that grows plants in a lab. To find somaclonal variants, different methods were used. These methods looked at things like how the plants looked, changes in their chromosomes, and markers in their genes and chemicals. Moreover, the research on somaclonal variation is crucial for managing and reducing it to create identical plants genetically. It is also useful in generating genetic diversity as a tool for breeders to enhance their genetic improvement efforts. Scientists have studied a lot about somaclonal variation in plants that have soft stems, but not as much has been done on fruit crops that grow in temperate climates and live for many years. This chapter gives an overview of the different methods used to find changes in perennial fruit crops caused by tissue culture techniques. It aims to combine all the information from previous research on this subject [3], [4].

Furthermore, this article provides detailed information about new developments in analyzing and identifying genetic variations in olive plants that are grown in a controlled environment using tissue samples from the nodes and somatic embryos. In nature, the variety and differences in genes within a group of living things are created through the mixing of genetic material. Things like how animals are chosen to survive, changes in their genes, moving to new places, and how many of them there are all affect how different their genes are from one another. In 1958, researchers found a new way to create genetic variation in plants. They discovered that when plant cells were grown outside of their normal environment, they became genetically unstable. This was also true for cells that were regenerated after being damaged or removed. The first time that somaclonal variation was noticed was mentioned in a report. Afterwards, scientists became very interested in the differences found in plant tissue and cell cultures. Larkin and Scowcroft came up with new terms, called neologisms, to describe the outcomes of growing plants in a laboratory instead of in nature. The word 'somaclone' means plants that come from cell culture, and the term 'somaclonal variation' means genetic differences among these plants. The development of plant cells in a laboratory setting and their ability to grow into complete plants is a process that doesn't require the union of two cells [5], [6].

It only involves the cells dividing. In this situation, it is a big problem when growing plant tissue and it changes in an uncontrolled and random way. In a laboratory setting, the conditions for growing plants outside of their natural environment can cause changes in their genetic makeup and physical appearance. These changes can be seen in plants that are grown from cells taken from different parts of the plant, such as the stem or leaves, or from individual cells that have been separated from the plant. Some or all of the somaclones might look different from the plants they were derived from. Normally, changes can happen by themselves and may be caused by temporary or permanent genetic changes in cells or tissue when they are grown in a lab. Temporary changes occur due to effects on genes or body functions and can't be passed on or undone. On the other hand, permanent changes are inheritable and usually show the existing differences in the original plant or are the result of

new differences. The current research shows that somaclonal variation can vary from affecting only one specific trait to impacting the entire genetic makeup of a plant. Somaclonal variation refers to when plants have changes in their genetic makeup, which can be helpful in making crops better. These changes can make the plants more resistant to diseases, or they can make the crops of higher quality or yield more [7], [8].

Epigenetic variation is another term for developmental variation. It means that there are ongoing changes in the physical traits of an organism, like having thorns in a young Citrus tree, which are caused by specific genes. Tissue samples from fully grown parents change to become more young in a gradual way when placed in a laboratory setting. Tissue cultures can be at different stages of growth, from fully grown to very young. Plants that grow from these tissues will be different depending on how developed the tissue was when it was told to grow again. When shoots are grown from plant cells that have changed their specific functions, they can produce a younger version of the original plant. However, this younger version may or may not remain the same as it grows older. For example, Eucalyptus plants could produce new plants with leaves that don't move and are similar to those of young plants. However, as time goes on, the childhood characteristics stop being shown and the traits of the parent return. One example of epigenetic variation that is well-known is when callus no longer needs auxin, cytokinin, or vitamins. These changes in requirements are commonly referred to as tissue or cellular adaptation. Other changes that happen outside of the organism, such as extreme energy or vitality, might be connected to becoming young again or getting rid of viruses. Tissue culture-grown plants grow like young plants until they are encouraged to bloom.

During that time, the plant frequently returns to the same physical features as its parent plant. The tissue culture industry uses this short-lived energy to make strong young plants that are easy to move, grow fast, and get settled in quicker than other plants that are grown in the usual way. Transient dwarfism is likely caused by epigenetic factors, meaning that it is influenced by changes in gene expression rather than alterations in the actual DNA sequence. It may occur because growth regulators from the tissue culture medium are carried over and affect growth patterns. Some small plants can grow normally again after being stunted for a season or two in a field or greenhouse. An example of heritable somaclonal variation is when thornless blackberries are grown from parents that have different types of cells mixed together. The TE blackberry is a type of blackberry that grows a lot in the northwestern part of the United States. It is one of the most important blackberry varieties for the economy in the United States. The TE plant looks like it doesn't have any thorns, but its thornlessness is not always permanent because the plant can produce thorny root suckers. This unstableness happens because TE is a type of plant with different layers that are arranged in a certain way. One layer of tissue without thorns, called LI, is on top of another layer of tissue with thorns, which is normal. Root cuttings of TE come from tissue found inside the plant, which comes from a certain genetic type that causes the plant to have thorns. This tissue goes back to having thorns after being cut and grown into a new plant. Also, the reproductive cells of TE that come from the LII-derived tissue show the presence of sharp thorns, not the absence of thorns, when they are crossed with other plants.

These reproductive systems create new plants from existing side buds, keeping the chimeral status. Shoot-tip cultures grew quickly and made a lot of shoots in a lab setting. Almost half of the new plants were unusually short and had a special curved stem characteristic. The small plants made new plants without thorns, while the big plants made new plants with thorns. The thornless blackberries likely grew from special cells on the surface of the plant. These plants were still a mix of different types of cells and grew from buds on the side of the

main plant. Because the small plants didn't have any thorns, it was expected that their reproductive cells would also not have any thorns. When we crossbred and allowed these pure plants without thorns to reproduce on their own, we discovered that the bending petiole trait was connected to a strong gene for being without thorns (Ste) that was passed down through reproduction.

Groups of cells that are made up of many individual cells working together. or offspring of a plant, are referred to as monoclonal explants. These explants are genetically identical and can be used for various purposes in research and cultivation. Monoclonal explants are commonly used in tissue culture techniques for micropropagation, which involves growing plant tissues in a controlled environment to produce multiple identical clones. This process allows for the rapid and efficient production of large quantities of plants with desirable traits. Overall, monoclonal explants play a significant role in agriculture and scientific studies by providing a consistent and reliable source of genetically identical plant material. Seeds or baby plants are believed to have the same genes, allowing for easy comparison and study. In simpler terms, it has been found that genetic instability can happen on its own in a lab setting.

These cells have different functions. Phloem cells transport sugars and other nutrients throughout the plant. Parenchyma cells are responsible for storage and support. Xylem cells help with water transport and provide structural support. The cortex and xylem parenchyma are parts of a plant. shape and size, and they have different functions in the body. Ploidy level refers to the number of sets of chromosomes in a cell or organism. organisms, scientists have developed a system called taxonomy. This system categorizes organisms based on their shared characteristics, placing them into groups such as species, genus, family, order, class, phylum, and kingdom. This helps scientists organize and understand the vast diversity of life on Earth. The authors of this study are Bright and colleagues. Other than protoplasts, sources that come from multiple cells are called "complex cultures. Chimeras are creatures that have features or characteristics of different animals combined together. Some plants have two or more sets of genes, making them chimeras. They are made up of differences in their genetic makeup and characteristics are called cell sectors or tissues. Cells Lots of imaginary creatures, and a group of researchers called Whitham and others. Body organisation refers to how the different organs, tissues, and cells are structured and arranged in order to carry out various functions. This arrangement is determined by the DNA of an individual, which provides instructions for the formation and development of different body parts. The growth center of a plant, called the meristem, has an impact on the stability of chimeras.

The chimera that is the most steady or secure is the one we are referring to. A periclinal chimera is a plant that has different genetic traits in different layers or areas within its tissues. However, even these mixed creatures can become unstable if cells move around and are replaced. Half of the plant shoots that were grown from tissue culture of a thornless plant with mixed characteristics. Blackberries were small in size and didn't have any thorns. Many differences this was caused by the separation of chimeral creatures. Some types of Somaclonal variation happens when new plants are grown from tissue cultures, and these new plants may have differences due to genetic changes. A difference in characteristics that does not follow a straightforward pattern of inheritance. To check for existing changes in plants caused by tissue culture, the newly grown plants may go through another process of growing in a laboratory. Clones that already have differences should result in more differences in the first group than in the second group. And so on. After a few generations, people might assume that any existing differences would either disappear or remain the same. Subsequent means happening after something else has already happened. It is more likely that the variation comes from tissue culture.

DISCUSSION

Plant tissue culture techniques provide an alternative way to grow new plants from small pieces of existing plants. Clonal propagation through tissue culture, also called micropropagation, can happen quickly in a small area. In 2008, Eftekhari and his team conducted a study. The fact that all the plants in a clone population look the same is a big advantage for clonal cultivars when they are grown for business purposes. However, changes in genes can happen in cells that haven't developed into specific types, protoplasts that have been separated, calli (cell masses), tissues, and physical characteristics of plants that have been grown in a lab. Currently, it is more popular to grow plants like strawberry, papaya, banana, grapes, pineapple, citrus, tomato, cucumber, watermelon, rhododendron, and orchids using a method called micropropagation instead of traditional ways. But, since the first report of different types of sugarcane plants in 1971, many other cases of changes in plants have been reported in different crops. The banana plant can sometimes produce plants that are different from the original ones.

In Cavendish bananas, this happens between 6% and 38% of the time, according to Sahijram et al. According to Smith in 1988, the percentage could be as high as 90%, but in 2003 it was around 75%. When it comes to growing plants for business purposes, any kind of change, especially changes in genes, can be seen as a problem and not valuable. This is because these changes can cause the plant to not stay true to its original genetic makeup. On the other hand, growing and studying plant cells and tissues in a lab can create more genetic differences more quickly and without using complicated methods. This technology has a lot of potential when it comes to improving crops that are mainly grown by planting cuttings or other parts of the plant, instead of growing from seeds (Figure 1). This is because these crops have challenges like taking a long time before they start producing fruits, sometimes having genetic problems from close breeding, problems with self-pollination and cross-pollination, and not having a lot of genetic diversity, especially in decorative plants. In simpler terms, somaclonal variations can be examined for desirable traits in a lab, which is easier and faster compared to studying cross seedlings of crops that grow for a long time and require a lot of land. Somaclones can be useful in plant breeding and making genetic improvements. We can find and develop these new variants by using the right selection methods in the lab [9], [10].

Tissue culture is a good way to make copies of plants, but sometimes the new plants have differences from the original plant. These somaclonal variations happen when new changes appear in the cells during tissue culture. The causes of genetic changes in tissue culture have been linked to many stress factors. These include injuring the tissue, using chemicals to sterilize, using incomplete tissue, using unbalanced media components such as high levels of plant growth regulators, using sugar instead of sunlight for energy, having different lighting conditions, and having a disrupted balance between humidity and water loss. Many differences in micropropagated plants may be caused by damage to plant tissues during laboratory growth. Oxidative stress happens when there are too many harmful chemicals in the body called pro-oxidants or reactive oxygen species (ROS). These chemicals include superoxide, hydrogen peroxide, hydroxyl, peroxyl, and alkoxyl radicals. These ROS can cause changes in the way DNA is methylated, either too much or too little. They can also cause changes in the number and structure of chromosomes as well as deletions and substitutions in the DNA base. All of these changes can lead to mutations in plant cells grown in a lab. Both of these processes result in similar types of DNA changes [11], [12].

Tissue culture can turn on sleeping genes, which leads to variations in plants. Insertions of transposable elements and retrotransposons can cause changes in the DNA of plants. This can lead to mutations in the plant's genes. If these mutations happen in many places in the

genome, it can cause major changes in the structure of the chromosomes. These changes can cause genes to not work properly, have the wrong number of chromosomes, and new pieces of DNA being added. However, we still don't fully understand many parts of how somaclonal variations happen. So, it's important to look at all the genetic information of the crop by sequencing its entire genome., like Illumina/Solexa, ABI/SOLiD, 454/Roche, and Helicos, allow scientists to study many genes at once and learn more about how they work. Somaclonal variations are changes that can occur in plants when they are grown from cells in a laboratory. These changes can be useful for improving crops.

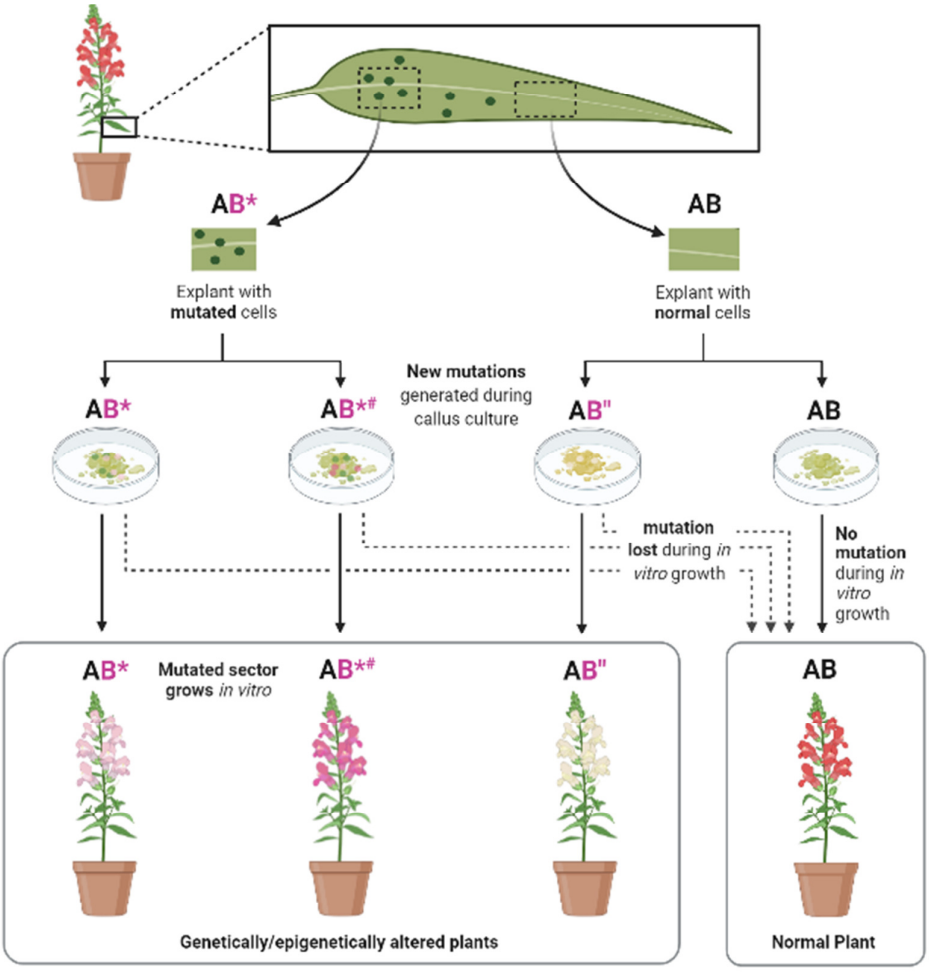

Figure 1: Representing the overview about generation of somaclonal variation in the plants [BioRender.Com].

Having different characteristics in plants and crops is important for regular breeding programs. The usual process of improving crops takes about 10-15 years. It involves changing the genetic material of the plants, choosing and stabilizing the best genes, testing different varieties, growing more of the chosen variety, protecting it legally, and finally growing the crops for production. Plant tissue culture is a useful technology that has helped create new tools to help plant breeders. Tissue culture-induced somaclonal variation is similar to the changes caused by chemicals and physical mutagens. It allows us to find natural differences that could be useful for improving crops. The benefits include It is not as expensive as other ways of changing genes and does not need special safety procedures. There are more plant species that can be grown in tissue culture systems than can currently be

altered through somatic hybridization and transformation methods. It is not important to have found the genes responsible for the trait, or in the case of transformation, to have separated and copied them. New types have been found in somaclones, and evidence shows that the rate and pattern of genetic recombination can change when passing through tissue culture. This means that changes in the genes can come from different parts of the genome that traditional breeding and mutating cannot reach. It is not possible to get mixed gene expression if somaclones are grown using cell culture.

Somaclonal variation has been most successful in crops that have limited genetic systems, such as plants that reproduce without seeds or through vegetative methods, and crops with a small range of genetic diversity. In plants used for decoration, the use of variability created in a lab is now a common practice in the breeding industry. Somaclonal variation is a process where plants can have unexpected changes in their traits. However, it is difficult to use because even if we know what factors cause these changes, we cannot predict what will happen in each case. This is because the changes are random and cannot be reproduced.

Additionally, since many genetic changes are caused by small alterations in the DNA or rearrangement of chromosomes, most R1 separate or divide into different groups. So, for traits like yield, it is really hard to choose individuals with better qualities in the first generation. Although methods for choosing somaclones that are resistant to different diseases and environmental stresses have been developed for many plants we grow for food, unfortunately, there are currently no ways to select for more complex traits like how much fruit a plant produces, how sweet it tastes, its texture, or how long it stays fresh. Somaclonal variation can be used in plant breeding if it is passed down from generation to generation and remains consistent in its genetic makeup. Only a few good types of plants that have been created using somaclonal variations have been made available up until now. This may be because plant breeders and tissue culture scientists don't communicate, and somaclones are not easy to predict. Additionally, although new types of plants have been created through somaclonal variation, in many cases, better versions have not been chosen for the following reasons the differences were all harmful, positive changes were also affected negatively, the changes were not unique, or the changes did not remain reliable when reproducing or combining with other plants.

Somaclonal variation means that plants grown from tissue cultures can have different genes than their parents. This can happen naturally or because of the tissue culture process used to grow them. The first reports of somaclonal variation were mainly from plants like potatoes or wheat, and they showed different characteristics such as how tall the plant grew, how it looked, when it matured, how resistant it was to diseases, how much it produced, and other biochemical traits. Sweet orange and other types of citrus plants that have multiple embryos are good for studying changes in their characteristics caused by tissue culture because of their biology and success in tissue culture. The sweet orange can produce many new embryos from its own tissues. This happens when special cells called callus and suspension cultures are grown. The researchers found that the sweet orange can be easily grown from a single cell and can also be grown from a small piece of tissue. This is important because it allows scientists to modify the sweet orange using a specific technique. Better qualities include faster growth, no seeds, and better quality fruits/juices, all while keeping the same type of plant. Somaclonal variation is a useful but not well explored way to introduce genetic changes for improving citrus plants. It works well for citrus varieties that are hard to improve through regular breeding methods. Somaclonal variation can also be used in certain types of plants, like Clementines, if each plant can grow well on a certain type of media.

Understanding the different ways that sugarcane can change, it is difficult to control and manage the existing differences in modern sugarcane plants. The material used and the cell chosen for transformations happen by chance. However, changes in plants caused by tissue culture can be controlled. Tissue culture has shown that differences in genes can happen. The findings from studying non-regenerating calli and albino plants showed that there were high levels of polymorphism. This means that there were differences in the genes of these plants. These variations may also be found in the transgenic plants, although it is not certain. The results of this study are being prepared for publication. It is widely known that callogenesis causes extra differences in plant tissue cultures. All methods for changing sugarcane cells use callogenesis to make more cells and grow new plants. Because of this, our recent work has been focused on the idea that the formation of new organs should only produce shoots that show the existing differences, without creating new differences. Repeating specific steps in growing plants in a lab made it impossible to grow plants with mosaic patterns. The GUS staining looked the same in all parts of the hygromicin-resistant plants. We found that the best conditions for transforming the plants were when they were between 1. 1-3 cm in size, when they were co-cultivated for 72-96 hours in the dark, and when the bacterial concentration was DO620nm= 1. The recovery rate of plants that are resistant to hygromycin (30 mg/L) was 1. 87% when using EHA105 (pCambia1301) and 2. 5% when using LBA4404 (pTOK233). The benefits of this method involve using advanced tools to study the genetic changes in transgenic plants caused by somaclonal variants.

Selection pressure refers to the influences or factors in the environment that affect the survival and reproductive success of organisms. These pressures can include things like competition for resources, predators, and changes in the physical environment. Organisms that are better suited to their environment and can adapt to these pressures have a higher chance of survival and passing on their genes to the next generation. select for microorganisms with desired traits or abilities. By subjecting the microorganisms to different conditions and selecting those that survive and thrive, scientists can artificially accelerate the evolution of certain traits in a population of microorganisms. This process is useful for various applications in industries such as medicine, agriculture, and biotechnology. Choose cell lines that are able to resist diseases, herbicides, and specific problems. Chemical compounds are substances made up of different elements. They can be in the form of solids, liquids, or gases. These compounds have specific properties and will react with other substances in predictable ways. Salt is a common mineral substance and a liquid from fungi.

However, out of the plants that grew again Only the characteristic of being resistant to diseases was shown from these cultures Tissue culture is a method used to choose specific cell lines that are valuable to researchers and industry. Therefore, it is easier to identify and select for individuals with the desired single gene trait, whereas it is more difficult to select for individuals with the desired combination of multiple genes for polygenic traits. People who have control over many genes are less likely to be successful compared to those who only have control over a few genes health, they need to target specific traits that provide the greatest benefits to plants in their environment. By focusing on these beneficial traits, selection pressure schemes can help plants adapt and survive in changing conditions. To make things better, the specific characteristic chosen on a cellular level needs to be shown. Overall, throughout the entire plant. are responsible for creating proteins. Some genes have other functions besides protein synthesis. It's important to remember that not all genes play the same role in the body.

Levels can be described as different stages or steps in a process or journey. the tobacco plants through a process called genetic engineering. We grew cells from the new plants. This shows

that there is a difference time of stress or when the plant was damaged. At the smallest level inside our bodies. a vaccine is seen as safe and effective in preventing the spread of disease, particularly among vulnerable populations such as children and the elderly. Resistance to Xanthomonas campestris pv. is when a plant is able to fight off or not be affected by the disease caused by this specific bacteria. Pruning is the act of cutting or trimming plants to improve their shape, size, and overall health. Some of these identical copies For some people, this characteristic has only lasted for a short time. We are currently gathering seeds. To understand how traits are passed on among somaclones, researchers examine these plants. In tissue culture, plant cells are grown in a laboratory setting, where they are provided with the necessary nutrients and hormonal treatments to encourage their growth and development. This process involves various steps, including sterilization, media preparation, and subculturing, which all require precise techniques and careful monitoring. The conditions in tissue culture can be challenging to control due to factors such as the sensitivity of plant cells to changes in temperature, light intensity, hormone concentrations, and contamination risks. Therefore, maintaining the ideal conditions for plant cells to grow in tissue culture can be likened to managing a controlled chaotic environment.

Cultural circumstances or situations. The genes of plants and animals can sometimes have problems body and grown in a laboratory setting. This allows scientists to study cells outside of their natural environment. Usually, an appropriate medium is used to provide the necessary nutrients for cell growth. Cell cultures are important tools in research and can be used to understand how cells behave and respond to different conditions. Stabilizing and controlling influences that are caused by a complete living thing and put in a container that is not natural for them. Stopped suddenly. For example, when a plant gets injured, callus forms as a response to heal the wound. This barely ever leads to enough organization to form shoots or roots cells can progress in order to regenerate into whole plants. Cut plant parts usually go through a process before they form into a meristem. This means that something can produce roots, shoots, or both. plain language: Growth regulators and other substances Glass-like materials provoke these actions. people feel tired and exhausted after a long day of work.

CONCLUSION

The improvements in tissue culture methods have allowed us to grow different plants in the lab. This is useful for multiplying crops on a large scale and is available for many types of plants. Clonal propagation and preservation of very good traits in plants needs the new plants to have very similar genes. However, growing plants in a laboratory can cause changes in their genes or the way they are marked, which can create differences in their characteristics. The presence of small and unnoticeable changes in plants grown in laboratory or preserved for future use is a disadvantage for both cloning and plant preservation. So, it is very important to make sure that the genes of plants grown in a lab are all the same at the beginning. Scientists have used different methods to check if the offspring grown in a lab have the same genes as their parents. They have looked at things like how the offspring look, their physical and chemical characteristics, and even studied their cells and DNA to make sure they are genetically the same. Somaclonal variation can be a big problem in micropropagation programs, where the goal is to make sure the plants produced are exactly like the original plant. However, somaclonal variation has given breeders a new way to quickly and easily get different genes in plants that are hard to breed or have limited genetic makeup, without using fancy technology. This paper reviews the causes of differences in plants grown in tissue culture and ways to make sure they are genetically the same. It also discusses how these differences could be used to improve crops.

REFERENCES:

[1] S. M. Kaeppler, H. F. Kaeppler, and Y. Rhee, "Epigenetic aspects of somaclonal variation in plants," *Plant Mol. Biol.*, 2000, doi: 10.1023/a:1006423110134.

[2] H. Krishna *et al.*, "Somaclonal variations and their applications in horticultural crops improvement," *3 Biotech*. 2016. doi: 10.1007/s13205-016-0389-7.

[3] A. Karp, "Somaclonal variation as a tool for crop improvement," *Euphytica*, 1995, doi: 10.1007/BF00023959.

[4] M. W. Bairu, A. O. Aremu, and J. van Staden, "Somaclonal variation in plants: Causes and detection methods," *Plant Growth Regul.*, 2011, doi: 10.1007/s10725-010-9554-x.

[5] M. K. Biswas, M. Dutt, U. K. Roy, R. Islam, and M. Hossain, "Development and evaluation of in vitro somaclonal variation in strawberry for improved horticultural traits," *Sci. Hortic. (Amsterdam).*, 2009, doi: 10.1016/j.scienta.2009.06.002.

[6] K. F. Abdellatif, A. E. Hegazy, H. M. Aboshama, H. A. Emara, and A. A. El-Shahed, "Morphological and molecular characterization of somaclonal variations in tissue culture-derived banana plants," *J. Genet. Eng. Biotechnol.*, 2012, doi: 10.1016/j.jgeb.2012.05.002.

[7] Z. Cao, S. Sui, X. Cai, Q. Yang, and Z. Deng, "Somaclonal variation in 'Red Flash' caladium: morphological, cytological and molecular characterization," *Plant Cell. Tissue Organ Cult.*, 2016, doi: 10.1007/s11240-016-0996-3.

[8] F. Nurilmala, R. P. Hutagaol, I. M. Widhyastini, U. Widyastuti, and Suharsono, "Somaclonal variation induction of bogor taro (Colocasia esculenta) by gamma irradiation," *Biodiversitas*, 2017, doi: 10.13057/biodiv/d180105.

[9] L. Sahijram, J. R. Soneji, and K. T. Bollamma, "Analyzing somaclonal variation in micropropagated bananas (Musa spp.)," *In Vitro Cellular and Developmental Biology - Plant*. 2003. doi: 10.1079/IVP2003467.

[10] S. L. Sun, J. Q. Zhong, S. H. Li, and X. J. Wang, "Tissue culture-induced somaclonal variation of decreased pollen viability in torenia (Torenia fournieri Lind.)," *Bot. Stud.*, 2013, doi: 10.1186/1999-3110-54-36.

[11] I. Roostika, N. Khumaida, and S. W. Ardie, "RAPD analysis to detect somaclonal variation of pineapple in vitro cultures during micropropagation," *Biotropia (Bogor).*, 2015, doi: 10.11598/btb.2015.22.2.422.

[12] F. Bradaï, F. Pliego-Alfaro, and C. Sánchez-Romero, "Somaclonal variation in olive (Olea europaea L.) plants regenerated via somatic embryogenesis: Influence of genotype and culture age on phenotypic stability," *Sci. Hortic. (Amsterdam).*, 2016, doi: 10.1016/j.scienta.2016.10.031.