II M.SC. BOTANY

GENETICS AND PLANT BREEDING

UNIT: I, II & IV

Sub.code:18PBT8

UNIT : I MENDELIAN GENETICS

MENDELISM:

The contribution of Mendel in Genetics is called Mendelism.

Mendel instead believed that heredity is the result of discrete units of inheritance, and every single unit (or gene) was independent in its actions in an individual's genome. According to this Mendelian concept**,** inheritance of a trait depends on the passing-on of these units.

Mendel's Experiments

The district of Moravia, then part of the Austro-Hungarian Empire. At the end of high school, he entered the Augustinian monastery of St. Thomas in the city of Brünn, now Brno of the Czech Republic. His monastery was dedicated to teaching science and to scientific research, so Mendel was sent to a university in Vienna to obtain his teaching credentials. However, he failed his examinations and returned to the monastery at Brünn. There he embarked on the research program of plant hybridization that was posthumously to earn him the title of founder of the science of genetics.

Mendel's studies constitute an outstanding example of good scientific technique. He chose research material well suited to the study of the problem at hand, designed his experiments carefully, collected large

amounts of data, and used mathematical analysis to show that the results were consistent with his explanatory hypothesis. The predictions of the hypothesis were then tested in a new round of experimentation.

Mendel studied the garden pea (Pisum sativum) for two main reasons. First, peas were available from seed merchants in a wide array of distinct shapes and colors that could be easily identified and analyzed. Second, peas can either self (self-pollinate) or be cross-pollinated. The peas self because the male parts (anthers) and female parts (ovaries) of the flower—which produce the pollen containing the sperm and the ovules containing eggs, respectively—are enclosed by two petals fused to form a compartment called a keel. The gardener or experimenter can cross (cross-pollinate) any two pea plants at will. The anthers from one plant are removed before they have opened to shed their pollen, an operation called emasculation that is done to prevent selfing. Pollen from the other plant is then transferred to the receptive stigma with a paintbrush or on anthers themselves. Thus, the experimenter can choose to self or to cross the pea plants.

Monohybrid cross:

A **monohybrid cross** is one in which both parents are heterozygous (or a hybrid) for a single (mono) trait. The trait might be petal color in pea plants. When conducting crosses, the first generation is called P (or P_0), the second generation is F_1 (F is for filial), and the next generation is F_2.

Using monohybrid crosses, Mendel observed that although different alleles could influence a single trait, they remained indivisible and could be inherited separately. This is the basis of **Mendel's First Law**, also called **The Law of Segregation**, which states: during gamete formation, the two alleles at a gene locus segregate from each other; each gamete has an equal probability of containing either allele.

Punnett Squares

Given the genotypes of any two parents, we can predict the genotypes of gametes that will be produced during meiosis. Using that information, we can predict all of the possible genotypes of the offspring. Furthermore, if we also know the dominance relationships for all of the alleles, we can predict the phenotypes of the offspring. A convenient method for calculating the expected genotypic and phenotypic ratios from a cross was invented by Reginald Punnett. A **Punnett square** is a matrix in which all of the possible gametes produced by one parent are listed along one axis, and the gametes from the other parent are listed along the other axis. Each possible combination of gametes is listed at the intersection of each row and column. Punnett squares can also be used to calculate the frequency of offspring.

Dihybrid cross:

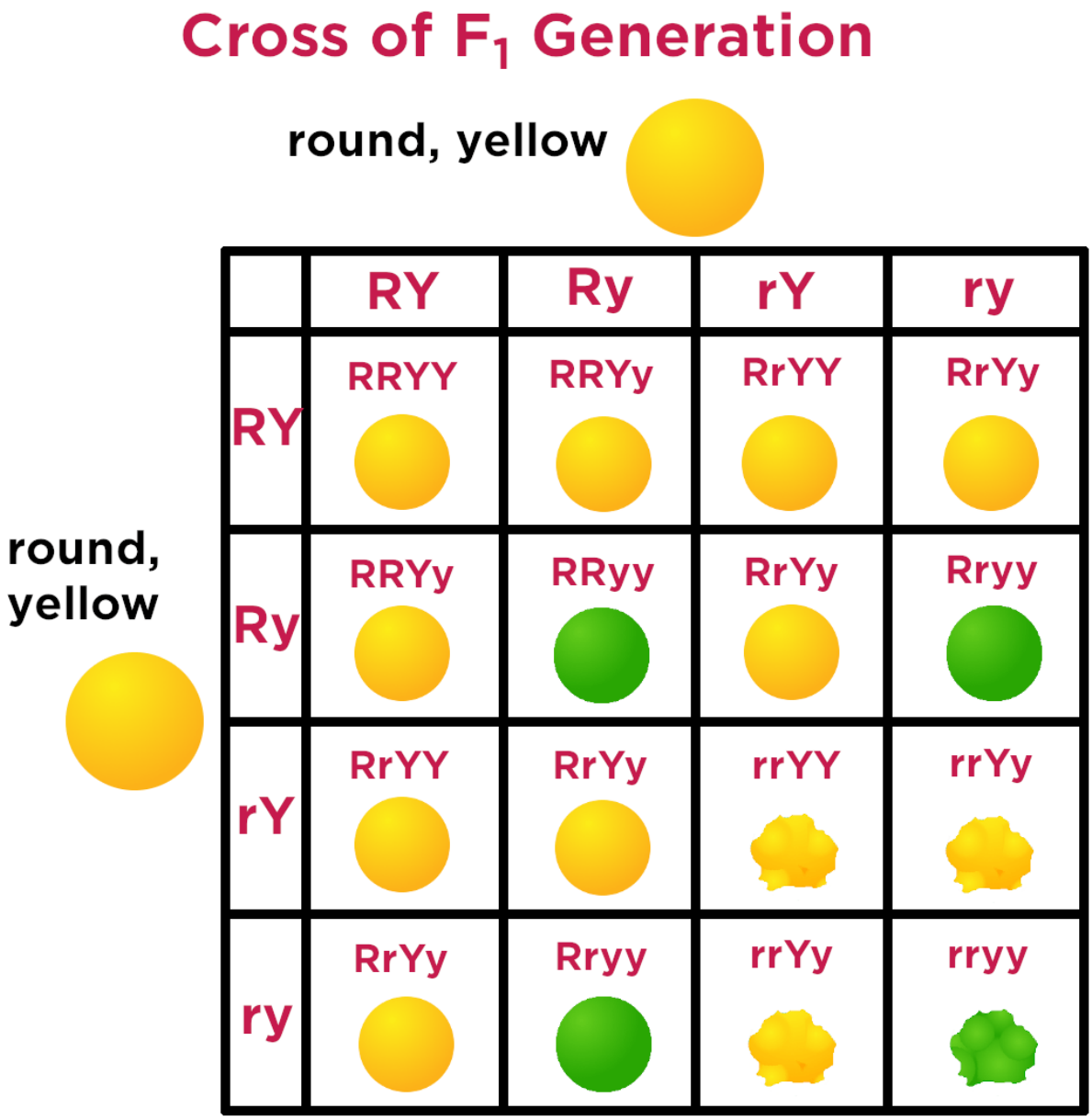
A dihybrid cross is a breeding experiment between two organisms which are identical hybrids for two traits. In other words, a dihybrid cross is a cross between two organisms, with both being heterozygous for two different traits. The individuals in this type of trait are homozygous for a specific trait. These traits are determined by DNA segments called genes.

In a dihybrid cross, the parents carry different pair of alleles for each trait. One parent carries homozygous dominant allele, while the other one carries homozygous recessive allele. The offsprings produced after the crosses in the F1 generation are all heterozygous for specific traits.

Dihybrid Cross Examples

Mendel took a pair of contradicting traits together for crossing, for example colour and the shape of seeds at a time. He picked the wrinkled-green seed and round-yellow seed and crossed them. He obtained only

round-yellow seeds in the F1 generation. This indicated that round shape and yellow colour of seeds are dominant in nature.



Meanwhile, the wrinkled shape and green colour of seeds are recessive traits. Then, F1 progeny was self-pollinated. This resulted in four different combinations of seeds in the F2 generation. They were wrinkled-yellow, round-yellow, wrinkled-green seeds and round-green in the phenotypic ratio of 9:3:3:1.

During monohybrid cross of these traits, he observed the same pattern of dominance and inheritance. The phenotypic ratio 3:1 of yellow and green colour and of round and wrinkled seed shape during monohybrid cross was retained in dihybrid cross as well.

Consider “Y” for yellow seed colour and “y” for green seed colour, “R” for round shaped seeds and “r” for wrinkled seed shape. Thus, the parental genotype will be “YYRR” (yellow-round seeds) and “yyrr” (green-wrinkled seeds).

Back cross:

Backcross, the mating of a hybrid organism (offspring of genetically unlike parents) with one of its parents or with an organism genetically similar to the parent. The backcross is useful in genetics studies for isolating (separating out) certain characteristics in a related group of animals or plants. In animal breeding, a backcross is often called a topcross. Grading usually refers to the mating of average, or “grade,” females to a superior male, then backcrossing the female offspring to the same or a similar sire.

Test cross:

Under the law of dominance in genetics, an individual expressing a dominant phenotype could contain either two copies of the dominant allele (homozygous dominant) or one copy of each dominant and recessive allele (heterozygousdominant).[1] By performing a test cross, one can determine whether the individual is homozygous or heterozygous dominant.[1]

In a test cross, the individual in question is bred with another individual that is homozygous for the recessive trait and the offspring of the test

cross are examined. Since the homozygous recessive individual can only pass on recessive alleles, the allele the individual in question passes on determines the phenotype of the offspring.[3] Thus, this test yields 2 possible situations:

1. If any of the offspring produced express the recessive trait, the individual in question is heterozygous for the dominant allele.
2. If any of the offspring produced all express the dominant trait, the individual in question is homozygous for the dominant allele.

Mendelian inheritance:

Mendelian inheritance refers to the inheritance of traits controlled by a single gene with two alleles, one of which may be completely dominant to the other. The pattern of inheritance of Mendelian traits depends on whether the traits are controlled by genes on autosomes or by genes on sex chromosomes.

- **Autosomal traits** are controlled by genes on one of the 22 pairs of human autosomes. Autosomes are all the chromosomes except the X or Y chromosome, and they do not differ between males and females, so autosomal traits are inherited in the same way regardless of the sex of the parent or offspring.

- Traits controlled by genes on the sex chromosomes are called **sex-linked traits.** Because of the small size of the Y chromosome, most sex-linked traits are controlled by genes on the X chromosome. These traits are called **X-linked traits**. Single-gene X-linked traits have a different pattern of inheritance than single-gene autosomal traits because males have just one X chromosome. Males always inherit their X chromosome from their mother, and they pass on their X chromosome to all of their daughters but none of their sons.

Law of Segregation:

> Gregor Mendel's law of segregation states that the two alleles for each trait segregate, or separate, during the formation of gametes, and that during the formation of new zygotes, the alleles will combine at random with other alleles. The law of segregation ensures that a parent, with two copies of each gene, can pass on either allele. Both alleles will have the same chance of ending up in a zygote.

In sexually reproducing organsisms, the genome is carried in two identical copies. A copy was inherited from each parent, in the form of a gamete. These organisms are known as diploid when they have both copies of the genome, and haploid when they are gametes and have only one copy. Though Gregor Mendel was not clear on exactly how the process took place, modern microscopes and molecular techniques have revealed that alleles are separated during the process of meiosis.

Meiosis occurs in specialized cells known as gametocytes, which form haploid cells from diploid cells. In order for the ploidy of the cell to be reduced, the chromosomes in the cell must be equally divided. To start the process, all of the DNA in a cell is duplicated. This creates two copies of each allele. In this cell, there are now 4 alleles for each gene, although 2 of them are simply identical copies of the original 2. As meiosis begins, the chromosomes condense and align with their homologous pairs. Homologous chromosomes are those which contain identical portions of DNA, originally inherited from different parents.

During prophase I of meiosis I, the homologous chromosomes bind together. Special sections of the DNA can overlap, causing breakages in the DNA. Due to the similarity of the DNA, the breaks simply exchange segments in a process called crossing-over. This crossing-over helps establish both the randomness of allele inheritance and also the separation of different genes. The separation of different genes during meiosis is known as the law of independent assortment. During metaphase I of meiosis I, these bonded homologous pairs are aligned in the middle of the cell and separated. In doing this, the different alleles for each gene are affectively separated. During meiosis II, the copies of the alleles will be separated into individual gametes. This insures that each allele makes it to a new gamete, giving it an essentially equal chance of finding a gamete to fuse with and create a new organism.

Due to the law of segregation each allele is its own entity and always has an equal chance of being passed on to the next generation. This means that regardless of whether the allele is dominant or recessive in its relationship with the other allele it will be passed on in the same way, with the same frequency. The law of independent assortment states that while genes may exist on the same chromosomes, they too are inherited independently of each other due to the mechanisms of meiosis.

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UNIT: II

Linkage and Crossing over:

Linkage:

Linkage:- In every plants, there are present thousands of genes. The number of genes is more as compared to the number of chromosomes e.g., **pea.** It is clear that every chromosome possesses more than one gene. In 1903, **William Sutton** had expressed the possibility that every chromosome must have more than one unit factors.

Linkage may be complete, incomplete, or absent (not detectable), depending upon the distance between linked genes in a chromosome.

W. Bateson, Saunders and Punnett (1909) during experiments, found that the result obtained from a cross in sweet pea show a deviation from **law of independent assortment**

They were crossed the plant **Lathyrus odoratus** (sweet pea). In this plant, **purple colour** of flower is dominant over **red colour** and **long pollen** are dominant over **round pollen.** The **purple flowers (B)** and **long pollen (L)** were crossed with **red flowers (b)** and **round pollen (l),** in F1 generation the plants (BbLl) produced purple flower and long pollen, as expected.These plants were crossed with plants having **red flowers** and **round pollen (bbll)**

The resulting genotypes, and their actual and expected numbers under independent assortment, were as follows:

Phenotype	Genotype	Observed	Expected
Purple, long	B_L_	284	215
Purple, round	B_ll	21	71
Red, long	bbL_	21	72
Red, round	bbll	55	24
Total		381	381

In F2 generation 1 : 1 : 1: 1 ratio was expected after testcross but 7 : 1 : 1 : 7 ratio was actually obtained. This indicates that there is a tendency in dominant alleles to remain together. And similar is the case with recessive alleles.This deviation was, termed as **coupling** by **Bateson.** It was also observed that when two such dominant alleles or two recessive alleles come from different parents, they tend to remain separate and was termed as **repulsion.**When the plants with **purple flowers** and **round pollen (Bbll)** were crossed with **red flowers** and **long pollen (bbLL).** The results of testcross were similar to that coupling phase 1 : 7 : 7 : 1 ratio instead of expected 1 : 1 : 1 : 1. Therefore **Bateson and co-workers** coined two words as **coupling and repulsion.** Bateson and Punnett could not explain the exact reasons of coupling and repulsion, and it was T.H. Morgan who while performing experiments with Drosophila, in 1910, found that coupling or repulsion was not complete.

Linked genes :

By studying the inheritance of characters in the fruitfly Drosophila, T.H .Morgan and colleagues (1910) determined that genes are not completely independent as Mendel had thought, but that they tend to be inherited in groups. Since independent assortment does not occur, a dihybrid cross following two linked genes will not produce and F2 phenotypic ratio of 9:3:3:1. They observed that genes in the same chromosome are often transmitted together as a group, but that this was

not always so and that ‘crossing-over’ between chromosomes could occur to disrupt these linkage groups. Genes that are present on the same chromosome, and that tend to be inherited (transmitted to the gametes) together, are termed **linked genes** because the DNA sequence containing the genes is passed along as a unit during **meiosis**. The closer that genes reside on a particular chromosome, the higher the probability that they will be inherited as a unit, since crossing over between two linked genes is not as common.The genes present on same chromosome, thus, would not assort (separated) independently. Such type of genes are called **linked genes** and this phenomenon is called **linkage.**

Morgan defined linkage as follows: "that the pairs of genes of homozygous parents tend to enter in the same gametes and to remain together, whereas same genes from heterozygous parent tend to enter in the different gametes and remain apart from each other. He further stated that the tendency of linked genes remaining together in original combination is due to their location in the same chromosome ".According to him the degree or strength of linkage depends upon the distance between the linked genes in the chromosome.

Chromosomes Theory of Linkage:- Morgan along with castle formulated the chromosome theory of linkage which is as follows :-

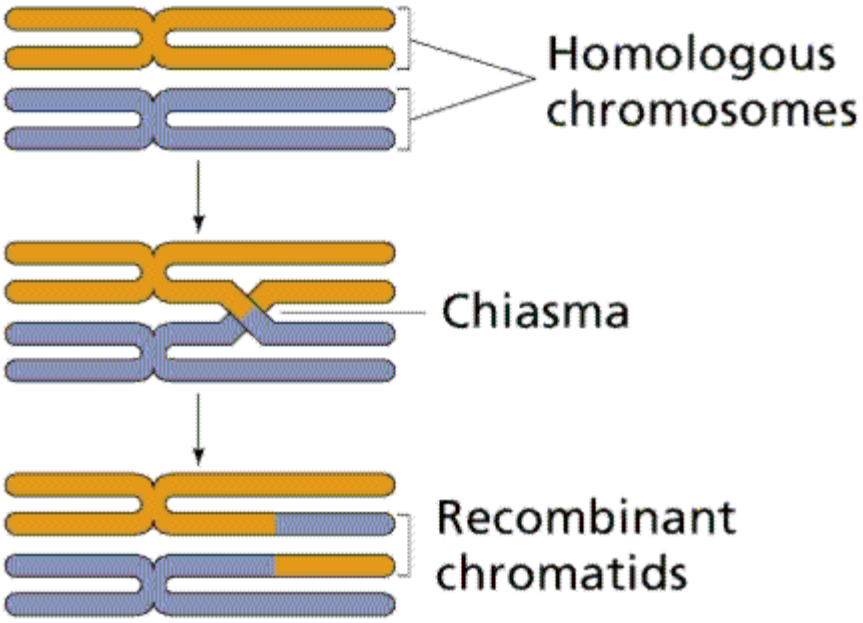
1. The genes which show the phenomenon of linkage are situated in the same chromosomes and separated during the process of inheritance.
2. The distance between the linked genes determines the strength of linkage. The closely located genes show strong linkage than the widely located genes which show the weak linkage.
3. The genes are arranged in linear fashion in the chromosomes.

Types of linkage :- Linkage may be complete, incomplete, or absent (not detectable)linkage, depending upon the distance between linked genes in a chromosome.

Linkage groups :- The group of linked genes that are located on the same chromosome, called linkage groups.Because, all the genes of a chromosome have their identical genes (alleomorphs) on the homologous chromosome, therefore linkage groups of a homologous pair of chromosome is considered as one. In any species, the number of linkage groups is equal to the number of pairs of chromosomes. **e.g. Corn (Zea mays)** has 10 pairs of chromosomes and 10 linkage groups.

Crossing over:

Crossing-over takes place during prophase I of meiosis. Crossing-over is another name for recombination or physical exchange of equal pieces of adjacent non-sister chromatids. During the process of crossing-over one of the paired chromosome arms exchanged physically at one or more locations .The two homologous chromosomes are connected at a certain point called **chiasma (pl chiasmata).** When crossing-over occurs chromatids break at chiasma and reattached to a different homologous chromosome . The chromatids resulting from the interchange of segments are known as the **cross over recombinants** and the chromatids that remain intact are called **non-crossover parental chromatids.**When these chromosomes segregate in meiosis, they form gametes that have completely new combinations of alleles. Generally, the longer the chromosome, the greater the number of chiasmata. The ability of genes to recombine is called recombination frequency.



Types of crossing over:

Crossing overs are of many types depending on number of chiasma:-

1. **Single crossing over**:- when the chiasma formation takes place at a single point of The chromosome pair this type of crossing over is known as single crossing over. In this types two crossed over chromatids and two non crossed over chromatids are formed.

2. **Double crossing over**:- When the chiasmata occur at two places in the same chromosomes known as double crossing over In the double crossing over formation of each chiasma is independent of the other and in it four types of recombination is possible.

   Two types of chiasma may be formed in double cross over:-

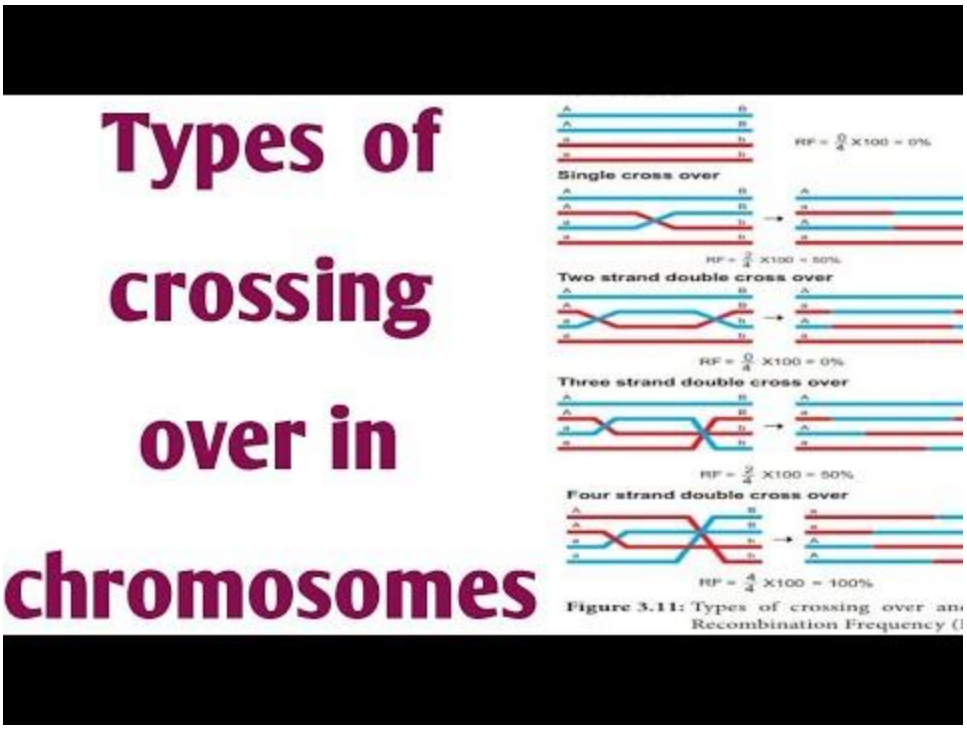
A. **Reciprocal chiasma** in this type both the chiasma are formed on two same chromatids. So, the second chiasma restores the order which was changed by the first Chiasma, and as a result two non- cross over chromatids are formed.

In this type out of four chromatids only two are involved in the double crossing over.

B. **Complimentary chiasma** When both the chromatids taking part in the second chiasma are different from those chromatids involved in the first. In this type four single cross overs are produced but no non cross over. Complimentary chiasma occurs when three or four chromatids of tetrad undergo crossing over.

3. **Multiple crossing over**

When crossing over take place at more than two point in the same chromosome pair it is known as multiple crossing over. It occurs rarely.



There are two theories on the physical nature of the process:-

1. **Classical theory or two plane theory (L. W. Sharp):-**

   Proposes that cross-over and formation of the chiasma occur first, followed by breakage and reunion with the reciprocal homologues. According to this theory, chiasma formation need not be accompanied by chromosome breakage. But this theory was not accepted.

3. **Chiasmatype theory or one plane theory**

This theory was proposed by F.A. Janssens (1909) breakage occurs first, and the broken strands then reunite. Chiasmata are thus evidence, but not the causes, of a cross-overs.

Recombination During Meiosis:-

John Belling(1928) suggested that no break was necessary and proposed the **copy choice model.** He believed that crossing over might occur during duplication of homologous chromosomes and might brought about due to novel attachments formed between newly synthesized genes. While studying meiosis in some plant species. He visualized genes as beads (described as chromomeric), connected by non-genic interchromomeric regions. The newly synthesized daughter chromatids is derived due to copying of one chromosomes upto certain region and then switching on to the other homologous chromosome for copying the remaining portion or region of the chromosomes. The new chromatid would have a new arrangement, but no breaks and rejoining need be involved. This was such an attractive idea that the hypothesis in some form held center stage for nearly thirty years.

Chiasma Frequency:

In genetics, a **chiasma** (pl. **chiasmata**) is the point of contact, the physical link, between two (non-sister) chromatids belonging to homologous chromosomes. At a given chiasma, an exchange of genetic material can occur between both chromatids, what is called a chromosomal crossover, but this is much more frequent during meiosis than mitosis.[1] In meiosis, absence of a chiasma generally results in improper chromosomal segregation and aneuploidy.

Points of crossing over become visible as chiasma after the synaptenemal complex dissembles and the homologous chromosomes slightly apart from each other.

The phenomenon of genetic chiasmata (chiasma type) was discovered and described in 1909 by Frans Alfons Janssens, a Professor at the University of Leuven in Belgium.

When each tetrad, which is composed of two pairs of sister chromatids, begins to split, the only points of contact are at the chiasmata. The chiasmata become visible during the diplotene stage of prophase I of meiosis, but the actual "crossing-overs" of genetic material are thought to occur during the previous pachytene stage. Sister chromatids also form chiasmata between each other (also known as a chi structure), but because their genetic material is identical, it does not cause any noticeable change in the resulting daughter cells.

In humans, there seems to be one chiasma per chromosome arm, and in mammals, the number of chromosome arms is a good predictor of the number of crossovers. Yet, in humans and possibly other species, evidence shows that the number of crossovers is regulated at the level of an entire chromosome and not an arm.

The grasshopper Melanoplus femurrubrum was exposed to an acute dose of X-rays during each individual stage of meiosis, and chiasma frequency was measured. Irradiation during the leptotene-zygotene stages of meiosis, that is, prior to the pachytene period in which crossover recombination occurs, was found to increase subsequent chiasma frequency. Similarly, in the grasshopper Chorthippus brunneus, exposure to X-irradiation during the zygotene-early pachytene stages caused a significant increase in mean cell chiasma frequency. Chiasma frequency was scored at the later diplotene-diakinesis stages of meiosis. These results suggest that X-rays induce DNA damages, likely including double-

strand breaks, and these damages are repaired by a crossover pathway leading to chiasma formation.

Gene mapping:

Researchers begin a genetic map by collecting samples of blood., saliva, or tissue from family members that carry a prominent disease or trait and family members that don't. The most common sample used in gene mapping, especially in personal genomic tests is saliva. Scientists then isolate DNA from the samples and closely examine it, looking for unique patterns in the DNA of the family members who do carry the disease that the DNA of those who don't carry the disease don't have. These unique molecular patterns in the DNA are referred to as polymorphisms, or markers.

The first steps of building a genetic map are the development of genetic markers and a mapping population. The closer two markers are on the chromosome, the more likely they are to be passed on to the next generation together. Therefore, the "co-segregation" patterns of all markers can be used to reconstruct their order. With this in mind, the genotypes of each genetic marker are recorded for both parents and each individual in the following generations. The quality of the genetic maps is largely dependent upon these factors: the number of genetic markers on the map and the size of the mapping population. The two factors are interlinked, as a larger mapping population could increase the "resolution" of the map and prevent the map from being "saturated".

In gene mapping, any sequence feature that can be faithfully distinguished from the two parents can be used as a genetic marker. Genes, in this regard, are represented by "traits" that can be faithfully distinguished between two parents. Their linkage with other genetic markers is calculated in the same way as if they are common markers and the actual

gene loci are then bracketed in a region between the two nearest neighboring markers. The entire process is then repeated by looking at more markers that target that region to map the gene neighborhood to a higher resolution until a specific causative locus can be identified. This process is often referred to as "positional cloning", and it is used extensively in the study of plant species. One plant species, in particular in which positional cloning is utilized is in maize. The great advantage of genetic mapping is that it can identify the relative position of genes based solely on their phenotypic effect.

Genetic mapping is a way to identify exactly which chromosome has which gene and exactly pinpointing where that gene lies on that particular chromosome. Mapping also acts as a method in determining which gene is most likely recombine based on the distance between two genes. The distance between two genes is measured in units known as centimorgan. A centimorgan is a distance between genes for which one product of meiosis in one hundred is recombinant. The further two genes are from each other, the more likely they are going to recombine. If it were closer, the opposite would occur.[citation needed]

Physical mapping

Since actual base-pair distances are generally hard or impossible to directly measure, physical maps are actually constructed by first shattering the genome into hierarchically smaller pieces. By characterizing each single piece and assembling back together, the overlapping path or "tiling path" of these small fragments would allow researchers to infer physical distances between genomic features. The fragmentation of the genome can be achieved by restriction enzyme cutting or by physically shattering the genome by processes like sonication. Once cut, the DNA fragments are separated by electrophoresis.[6] The resulting pattern of DNA migration (i.e. its genetic fingerprint) is used to identify what stretch of DNA is in the clone. By analyzing the fingerprints, contigs are assembled by automated (FPC) or manual means (pathfinders) into overlapping DNA

stretches. Now a good choice of clones can be made to efficiently sequence the clones to determine the DNA sequence of the organism under study.

In physical mapping, there are no direct ways of marking up a specific gene since the mapping does not include any information that concerns traits and functions. Genetic markers can be linked to a physical map by processes like in situ hybridization. By this approach, physical map contigs can be "anchored" onto a genetic map. The clones used in the physical map contigs can then be sequenced on a local scale to help new genetic marker design and identification of the causative loci.

Macrorestriction is a type of physical mapping wherein the high molecular weight DNA is digested with a restriction enzyme having a low number of restriction sites.

There are alternative ways to determine how DNA in a group of clones overlaps without completely sequencing the clones. Once the map is determined, the clones can be used as a resource to efficiently contain large stretches of the genome. This type of mapping is more accurate than genetic maps.

Mapping of mutational sites within a gene

In the early 1950s the prevailing view was that the genes in a chromosome are discrete entities, indivisible by genetic recombination and arranged like beads on a string. During 1955 to 1959, Benzer performed genetic recombination experiments using rII mutants of bacteriophage T4. He found that, on the basis of recombination tests, the sites of mutation could be mapped in a linear order.[7][8] This result provided evidence for the key idea that the gene has a linear structure

equivalent to a length of DNA with many sites that can independently mutate.

In 1961, Francis Crick, Leslie Barnett, Sydney Brenner and Richard Watts-Tobin performed genetic experiments that demonstrated the basic nature of the genetic code for proteins. These experiments, involving mapping of mutational sites within the rIIB gene of bacteriophage T4, demonstrated that three sequential nucleobases of the gene's DNA specify each successive amino acid of its encoded protein. Thus the genetic code was shown to be a triplet code, where each triplet (called a codon) specifies a particular amino acid. They also obtained evidence that the codons do not overlap with each other in the DNA sequence encoding a protein, and that such a sequence is read from a fixed starting point.

Edgar et al. performed mapping experiments with r mutants of bacteriophage T4 showing that recombination frequencies between rII mutants are not strictly additive. The recombination frequency from a cross of two rII mutants (a x d) is usually less than the sum of recombination frequencies for adjacent internal sub-intervals (a x b) + (b x c) + (c x d). Although not strictly additive, a systematic relationship was demonstrated that likely reflects the underlying molecular mechanism of genetic recombination.

Tetrad Analysis:

The meiotic products of ascomycetes (occasionally some other organisms) stay together as the four products of single meiosis, as a tetrad. In some organisms, tetrad formation is followed by a post-meiotic mitosis within the ascus, resulting in spore octads. If the four spores are situated in the same linear order as produced by the two divisions of meiosis it is an ordered tetrad.

In the ordered tetrad, considering two genes A and B, three arrangements of the spores (parental ditype [PD], tetratype [TT], non-parental ditype [NPD]) can be distinguished as seen in the Figure T35. The parental ditype (PD) indicates no crossing over; tetratype (TT) reveals one recombination between the two genes and the second division segregation of the B/b alleles reveals recombination between the B/b gene and the centromere

Sex determination in Plants:

Sex determination is as important for the fitness of plants as it is for animals, but its mechanisms appear to vary much more among plants than among animals, and the expression of gender in plants differs in important respects from that in most animals. In this Minireview, I provide an overview of the broad variety of ways in which plants determine sex. I suggest that several important peculiarities of plant sex determination can be understood by recognising that: plants show an alternation of generations between sporophytic and gametophytic phases (either of which may take control of sex determination); plants are modular in structure and lack a germ line (allowing for a quantitative expression of gender that is not common in animals); and separate sexes in plants have ultimately evolved from hermaphroditic ancestors. Most theorising about sex determination in plants has focused on dioecious species, but we have much to learn from monecious or hermaphroditic species, where sex is determined at the level of modules, tissues or cells. Because of the fundamental modularity of plant development and potentially important evolutionary links between monoecy and dioecy, it may be useful to relax the distinction often made between ‘developmental sex determination’ (which underpins the development of male versus female flowers in monoecious species) and ‘genetic sex determination’ (which underpins the separation of males and females in dioecious species, often mediated by a genetic polymorphism and sex chromosomes). I also argue for relaxing the distinction between sex determination involving a genetic

polymorphism and that involving responses to environmental or hormonal cues, because non-genetic cues might easily be converted into genetic switches.

Land plants are remarkably diverse in their sexual systems [1], but the rich variety of ways in which they express their sexuality has largely been built on a conserved foundation that determines whether reproductive tissues produce egg cells or sperm. In this sense, plants are similar to most other eukaryotes [2]. But when should the cell cycle end in the ultimate production of eggs versus sperm, which individuals or flowers should make this decision, and how should the decision be acted upon in developmental genetic terms? The first of these questions recognises that different parts of a plant, at different times, might be channelled towards being male versus female, and implies that selection in a given lineage will often optimise a distribution of decisions among modules in the plant 3, 4. The second question is prompted by the fact that the life cycle of all land plants involves an alteration of generations between haploid gametophytes and diploid sporophytes, and that one of these stages might take full or partial control of sex determination. The third question refers to the developmental genetic means by which sex is determined in a given lineage, with the subtext that there might be some sort of general answer we might hope to find from a survey among different lineages 5, 6. In this Minireview, I address these questions by taking an evolutionary perspective — we should expect sex to be determined in ways influenced both by a lineage's phylogenetic history, and by the action of selection on its populations' sex ratio or sex-allocation strategies, ultimately by modifying suitable gene networks.

Dominant and recessive sex link:

Sex-linked dominant is a rare way that a trait or disorder can be passed down through families. One abnormal gene on the X chromosome can cause a sex-linked dominant disease.

X-linked dominant inheritance:

Sometimes referred to as X-linked dominance, is a mode of genetic inheritance by which a dominant gene is carried on the X chromosome. As an inheritance pattern, it is less common than the X-linked recessive type. In medicine, X-linked dominant inheritance indicates that a gene responsible for a genetic disorder is located on the X chromosome, and only one copy of the allele is sufficient to cause the disorder when inherited from a parent who has the disorder. In this case, someone who expresses an X-linked dominant allele will exhibit the disorder and be considered affected.

X-linked dominant traits do not necessarily affect males more than females (unlike X-linked recessive traits). The exact pattern of inheritance varies, depending on whether the father or the mother has the trait of interest. All fathers that are affected by an X-linked dominant disorder will have affected daughters but not affected sons. However, if the mother is also affected then sons will have a chance of being affected, depending on whether a dominant or recessive X chromosome is passed on. When the son is affected, the mother will always be affected. Some X-linked dominant conditions are embryonic lethal in males, making them appear to only occur in girls.

Inheritance

In X-linked dominant inheritance, when the mother alone is the carrier of a mutated, or defective gene associated with a disease or disorder; she herself will have the disorder. Her children will inherit the disorder as follows:

- Of her daughters and sons: 50% will have the disorder, 50% will be completely unaffected. Children of either sex have an even chance of

receiving either of their mother's two X chromosomes, one of which contains the defective gene in question.

When the father alone is the carrier of a defective gene associated with a disease or disorder, he too will have the disorder. His children will inherit the disorder as follows:

- Of his daughters: 100% will have the disorder, since all of his daughters will receive one copy of his single X chromosome.
- Of his sons: none will have the disorder; sons do not receive an X chromosome from their father.

If both parents were carriers of a defective gene associated with a disease or disorder, they would both have the disorder. Their children would inherit the disorder as follows:

- Of their daughters: 100% will have the disorder, since all of the daughters will receive a copy of their father's X chromosome.
- Of the sons: 50% will have the disorder, 50% will be completely unaffected. Sons have an equal chance of receiving either of their mother's X chromosomes.

In such a case, where both parents carry and thus are affected by an X-linked dominant disorder, the chance of a daughter receiving two copies of the X chromosome with the defective gene is 50%, since daughters receive one copy of the X chromosome from both parents. Were this to occur with an X-linked dominant disorder, that daughter would likely experience a more severe form.

Some X-linked dominant conditions such as Aicardi syndrome are fatal to boys, therefore only girls with these conditions survive, or boys with Klinefelter's syndrome (and hence have more than one X chromosome).

A few scholars have suggested discontinuing the use of the terms dominant and recessive when referring to X-linked inheritance, stating that the highly variable penetrance of X-linked traits in females as a result of mechanisms such as skewed X-inactivation or somatic

mosaicism is difficult to reconcile with standard definitions of dominance and recessiveness.[1]

List of dominant X-linked diseases

- Vitamin D resistant rickets: X-linked hypophosphatemia
- Rett syndrome (95% of cases are due to sporadic mutations)
- Most cases of Alport syndrome[2]
- Incontinentia pigmenti[3][4]
- Giuffrè–Tsukahara syndrome[5]
- Goltz syndrome
- X-linked dominant porphyria[6]
- Fragile X syndrome
- Aicardi Syndrome

Sex-linked recessive

Sex-linked diseases are passed down through families through one of the X or Y chromosomes. X and Y are sex chromosomes.

Dominant inheritance occurs when an abnormal gene from one parent causes disease, even though the matching gene from the other parent is normal. The abnormal gene dominates.

But in recessive inheritance, both matching genes must be abnormal to cause disease. If only one gene in the pair is abnormal, the disease does not occur or it is mild. Someone who has one abnormal gene (but no symptoms) is called a carrier. Carriers can pass abnormal genes to their children.

The term "sex-linked recessive" most often refers to X-linked recessive.

X-LINKED RECESSIVE DISORDERS IN FEMALES

Females can get an X-linked recessive disorder, but this is very rare. An abnormal gene on the X chromosome from each parent would be required, since a female has two X chromosomes. This could occur in the two scenarios below.

In each pregnancy, if the mother is a carrier and the father has the disease, the expected outcomes are:

If both the mother and the father have the disease, the expected outcomes are:

- 100% chance of the child having the disease, whether boy or girl

The odds of either of these two scenarios are so low that X-linked recessive diseases are sometimes referred to as male only diseases. However, this is not technically correct.

Female carriers can have a normal X chromosome that is abnormally inactivated. This is called "skewed X-inactivation." These females may have symptoms similar to those of males, or they may have only mild symptoms.

Holandric Gene:

The holandric gene is Y-chromosome linked. The mammalian Y chromosome appears largely heterochromatic under the light microscope and it carries few genes. The H-Y antigen gene has been assigned to the proximal region of the long arm of Y and the testis-determining factor, formerly called TDF, now SRF, is proximal to the centromere in the same arm in humans. The long arm also contains the pseudoautosomal region (PAS); this DNA sequence Yp (SMCY) is homologous to a X-chromosomal tract, Xp (SCX), the region where X and Y crossing over can occur. The gene for surface antigen MIC2Y was assigned to the euchromatic region Ypter - q1 of the Y chromosome. The homolog was assigned to a X-chromosomal band between Xp22.3 and Xpter. The

azoospermia factor (AZF) Sp3 or HGM9 maps at the site of H-Y and may be identical with it. Genes controlling body height and tooth length were suspected to be in the Y-chromosome. An arginosuccinate and an actin pseudogene were located to the human Y chromosome.

Sex linked diseases:

Haemophilia

Haemophilia is a mostly inherited genetic disorder that impairs the body's ability to make blood clots, a process needed to stop bleeding. This results in people bleeding for a longer time after an injury, easy bruising, and an increased risk of bleeding inside joints or the brain.[1] Those with a mild case of the disease may have symptoms only after an accident or during surgery. Bleeding into a joint can result in permanent damage while bleeding in the brain can result in long term headaches, seizures, or a decreased level of consciousness.

There are two main types of haemophilia: haemophilia A, which occurs due to low amounts of clotting factor VIII, and haemophilia B, which occurs due to low levels of clotting factor IX. They are typically inherited from one's parents through an X chromosome carrying a nonfunctional gene. Rarely a new mutation may occur during early development or haemophilia may develop later in life due to antibodies forming against a clotting factor. Other types include haemophilia C, which occurs due to low levels of factor XI, and para-haemophilia, which occurs due to low levels of factor V. Acquired haemophilia is associated with cancers, autoimmune disorders, and pregnancy. Diagnosis is by testing the blood for its ability to clot and its levels of clotting factors. Prevention may occur by removing an egg, fertilizing it, and testing the embryo before transferring it to the uterus. Treatment is by replacing the missing blood clotting factors. This may be done on a regular basis or during bleeding episodes. Replacement may take place at home or in hospital. The clotting

factors are made either from human blood or by recombinant methods. Up to 20% of people develop antibodies to the clotting factors which makes treatment more difficult. The medication desmopressin may be used in those with mild haemophilia A.] Studies of gene therapy are in early human trials.

Typically, females possess two X-chromosomes, and males have one X and one Y-chromosome. Since the mutations causing the disease are X-linked recessive, a female carrying the defect on one of her X-chromosomes may not be affected by it, as the equivalent dominant allele on her other chromosome should express itself to produce the necessary clotting factors, due to X inactivation. Therefore, heterozygous females are just carriers of this genetic disposition. However, the Y-chromosome in the male has no gene for factors VIII or IX. If the genes responsible for production of factor VIII or factor IX present on a male's X-chromosome are deficient there is no equivalent on the Y-chromosome to cancel it out, so the deficient gene is not masked and the disorder will develop.

Since a male receives his single X-chromosome from his mother, the son of a healthy female silently carrying the deficient gene will have a 50% chance of inheriting that gene from her and with it the disease; and if his mother is affected with haemophilia, he will have a 100% chance of being a haemophiliac. In contrast, for a female to inherit the disease, she must receive two deficient X-chromosomes, one from her mother and the other from her father (who must therefore be a haemophiliac himself). Hence, haemophilia is expressed far more commonly among males than females, while double-X females are far more likely to be silent carriers, survive childhood and to submit each of her genetic children to an at least 50% risk of receiving the deficient gene. However, it is possible for female carriers to become mild haemophiliacs due to lyonisation (inactivation) of the X-chromosomes. Haemophiliac daughters are more common than they once were, as improved treatments for the disease have allowed more

haemophiliac males to survive to adulthood and become parents. Adult females may experience menorrhagia (heavy periods) due to the bleeding tendency. The pattern of inheritance is criss-cross type. This type of pattern is also seen in colour blindness.

As with all genetic disorders, it is also possible for a human to acquire it spontaneously through mutation, rather than inheriting it, because of a new mutation in one of their parents' gametes. Spontaneous mutations account for about 33% of all cases of haemophilia A.[21] About 30% of cases of haemophilia B are the result of a spontaneous gene mutation.

Color blindness

"Colorblind" redirects here. For color blindness in other species, see Color vision. For other uses, see Colour blind (disambiguation).

Not to be confused with Color blindness (racial classification).

Color blindness (**color vision deficiency**) is the decreased ability to see color or differences in color. It can impair such tasks as selecting ripe fruit, choosing clothing, and reading traffic lights. Color blindness may make some educational activities more difficult. However, problems are generally minor, and most color-blind people adapt. People with total color blindness (achromatopsia) may also be uncomfortable in bright environments and have decreased visual acuity.

The most common cause of color blindness is an inherited problem in the development of one or more of the three sets of the eyes' cone cells, which sense color. Among humans, males are more likely to be color blind than females, because the genes responsible for the most common forms of color blindness are on the X chromosome. Females have two X chromosomes, so a defect in one is typically compensated for by the other.

Non-color-blind females can carry genes for color blindness and pass them on to their children. Males only have one X chromosome and therefore always express the genetic disorder if they have the recessive gene. Color blindness can also result from physical or chemical damage to the eye, the optic nerve, or parts of the brain. Diagnosis is typically with the Ishihara color test; other methods include genetic testing.

Pedigree Chart:

A pedigree is a chart of the genetic history of a family over several generations

- Males are represented as squares, while females are represented as circles
- Shaded symbols mean an individual is affected by a condition, while an unshaded symbol means they are unaffected
- A horizontal line between man and woman represents mating and resulting children are shown as offshoots to this line
- Generations are labeled with roman numerals and individuals are numbered according to age (oldest on the left)

UNIT:IV

METHODS OF MOLECULAR MAPPING:

Molecular mapping methods:

Gene mapping describes the methods used to identify the locus of a gene and the distances between genes. Gene mapping can also describe the distances between different sites within a gene. The essence of all genome mapping is to place a collection of molecular markers onto their respective positions on the genome.

Restriction fragment length polymorphism

In molecular biology, **restriction fragment length polymorphism** (**RFLP**) is a technique that exploits variations in homologous DNA sequences, known as polymorphisms, in order to distinguish individuals, populations, or species or to pinpoint the locations of genes within a sequence.The term may refer to a polymorphism itself, as detected through the differing locations of restriction enzyme sites, or to a related laboratory technique by which such differences can be illustrated. In **RFLP** analysis, a DNA sample is digested into fragments by one or more restriction enzymes, and the resulting restriction fragments are then separated by gel electrophoresis according to their size.

Although now largely obsolete due to the emergence of inexpensive DNA sequencing technologies, RFLP analysis was the first DNA profiling technique inexpensive enough to see widespread application. RFLP analysis was an important early tool in genome mapping, localization of genes for genetic disorders, determination of risk for disease, and paternity testing.

RFLP analysis

The basic technique for the detection of RFLPs involves fragmenting a sample of DNA with the application of a restriction enzyme, which can selectively cleave a DNA molecule wherever a short, specific sequence is recognized in a process known as a restriction digest. The DNA fragments produced by the digest are then separated by length through a process known as agarose gel electrophoresis and transferred to a membrane via the Southern blot procedure. Hybridization of the membrane to a labeled DNA probe then determines the length of the fragments which are complementary to the probe. A restriction fragment length polymorphism is said to occur when the length of a detected fragment varies between individuals, indicating non-identical sequence homologies. Each fragment length is considered an allele, whether it actually contains a coding region or not, and can be used in subsequent genetic analysis.

Chromosome walking :

Chromosome walking is a method of positional cloning used to find, isolate, and clone a particular allele in a genomic library.

Chromosome walking is conducted to isolate a particular allele (say gene B) for a genetically transmitted disease in the vicinity of a previously mapped sequence (say gene A).

_____A_____B_____

An allele is a gene for a particular genetic trait passed on from adults to their offspring, such as the allele for brown eyes in a gene for eye colour. To locate a particular disease gene (say gene B), the walking starts at the closest gene that has already been identified (say gene A), known as a

marker gene. Each successive gene in the sequence is tested repeatedly for what are known as overlap restrictions and mapped for their precise location in the sequence. Eventually, walking through the genes reaches the mutant gene (gene B) in an unmapped sequence that binds to a fragment of a gene of that particular disease. Once the gene is cloned, its function can be fully identified. Throughout this process, tests are done to fully identify the properties of each successive clone, to map their locations for future use. Chromosome walking is a method used to clone in an orderly fashion the DNA segments along the chromosome starting at any point for which we have a probe 2 (gene A).

To build up a series of overlapping cloned DNA fragments, it begins with one clone from genomic library, and then a second clone is identified whose insert overlaps with the insert in the first clone. This was the first method, called chromosome walking. But there is a limitation to the speed of chromosome walking and that is because of the small size of the fragments that are to be cloned, another limitation is the difficulty of walking through the repeated sequence that are scattered through the gene.

A more straightforward approach thus is to use the insert-DNA from the starting clone as a hybridization probe to screen all the other clones in the library. Positive hybridization signals that are given by clones, whose inserts overlap with the probe, are used as new probes to continue the walk. The insert from one of the selected clones is then used as a hybridization probe with all other clones in the library.

Chromosome Jumping:

Chromosome jumping is a tool of molecular biology that is used in the physical mapping of genomes. It is related to several other tools used for the same purpose, including chromosome walking.

Chromosome jumping is used to bypass regions difficult to clone, such as those containing repetitive DNA, that cannot be easily mapped by

chromosome walking, and is useful in moving along a chromosome rapidly in search of a particular gene.

Chromosome jumping allows more rapid movement through the genome compared to other techniques, such as chromosome walking, and can be used to generate genomic markers with known chromosomal locations.

Chromosome jumping enables two ends of a DNA sequence to be cloned without the middle section. Genomic DNA may be partially digested using restriction endonucleases and with the aid of DNA ligase, the fragments are circularized. From a known sequence, a primer is designed to sequence across the circularised junction. This primer is used to jump 100 kb-300 kb intervals: a sequence 100 kb away would have come near the known sequence on circularisation. Thus, sequences not reachable by chromosome walking can be sequenced. Chromosome walking can be used from the new jump position (in either direction) to look for gene-like sequences, or additional jumps can be used to progress further along the chromosome.

Cytoplasmic inheritance:

The existence of genes as segments of nucleic acid molecules, located in chromosome of nucleus, has been demonstrated by several experiments. The nuclear genes control the phenotypes of the organisms and are concerned with the transmission of hereditary character from one generation to next generation is known and predictable Mendelian fashion.

The inheritance of genes of nuclear chromosomes is characterised by the fact that the genes from male and female parents contribute equally to the genetic constitution of the offspring. Therefore, in it the reciprocal crosses

between parents of different homozygous genotype will produce offspring's of identical phenotypes except for sex-linked genes.

However, in certain cases, although male and female parents contribute equally their nuclear genes to the offspring's, the results show a non-Mendelian inheritance pattern and the result of reciprocal crosses varies.

These variations suggest that the genes for the inheritance of certain characters do not occur within the nucleus, but they are present ill cytoplasm and play an important role in transmission of certain specific traits, which are not controlled by nuclear genes. Therefore, it builds up the concept of cytoplasmic inheritance. The genes for cytoplasmic inheritance are independent, self-replicating nucleic acids.

Terms and Definitions of Cytoplasmic Inheritance:

Extra-chromosomal inheritance, extra-nuclear inheritance, chromosomal inheritance and maternal inheritance are all synonyms. All these terms can be defined as the inheritance of characteristics of only one of the two parents, usually the female parent to the progeny. The reciprocal crosses show consistent differences as well as there is a lack of segregation in F_2 and subsequent generations.

The genes controlling cytoplasmic inheritance are present outside the nucleus and, in the cytoplasm, they are known as plasma genes, cytoplasmic genes, cytogeneses, extra nuclear genes or extra chromosomal genes.

The sum total of the genes present in cytoplasm of a cell is known as Plasmon. All the genes present in a plastid are known as plastoms. Similarly, all the genes present in a mitochondrion are known as chondrioms. The genes present in plastid and in mitochondrion are located in their own DNAs and are known as cp DNA and mt DNA, respectively. These DNAs are collectively termed organelle DNA.

Characteristics and Detection of Cytoplasmic Inheritance:
Cytoplasmic inheritances do not show Mendelian inheritance.

They show the following characteristic features:

i. Hereditary traits which are transmitted by cytoplasm do not show Mendelian segregation in crosses and in reciprocal crosses with respect to a particular set of characteristics controlled by a set of cytoplasmic genes produce dissimilar hybrids.

ii. Most of the recorded cytoplasmically inherited characteristics would follow the maternal line, i.e., uniparental mode of transmission. In higher plants and animals, ovum or egg cell is comparatively large and contains large amount of cytoplasm. But male gametes or sperms have very little amount of cytoplasm. So, under this situation, most of cytoplasmic factors are transmitted to the progeny through the ovum of mother.

It is known as maternal inheritance or trans-ovarian transmission. In this mode of transmission, all the offspring's of the parents have maternal condition and only female progeny can transmit the cytoplasmic characteristics to the succeeding generations. Hence the reciprocal crosses yield different or non-Mendelian results.

Characteristics of Mendelian Inheritance:

The inheritance pattern of characters of an organism as proposed by Mendel on the basis of monohybrid and di-hybrid crosses is referred to as Mendelian inheritance.

It shows the following characteristic features:

i. Contribution of both male and female is equal, hence results from reciprocal crosses are similar.

ii. Segregation produces the phenotypes ratio 3 : 1 and genotype ratio 1 : 2 : 1 in the F_2 generation of a monohybrid cross and a typical phenotype ratio 9 : 3 : 3 : 1 in di-hybrid crosses.

Mendelian inheritance pattern is regarded as a sufficient evidence for a gene to be located in chromosomes; such genes are called nuclear genes or simply as genes.

Predetermination – virus linked inclusions:

Viruses account for a large number of acute infections and occur as a consequence of hereditary or acquired forms of immunodeficiency. Because of their small size (20–300 nm), single virus particles can only be detected by electron microscopy. Some viruses may form aggregates in the nucleus and/or cytoplasm of the infected cells. Such viral inclusion bodies may be visible by light microscopy and definitely by electron microscopy. Polyoma viruses are ubiquitous in nature. Infections can be often observed in kidney tubular cells of renal transplants. Panels Band C show viral inclusion bodies due to Polyoma viruses in a renal biopsy of a kidney transplant patient. The spherical virus particles have a diameter of 30–45 nm and are arranged in characteristic paracrystalline arrays, which occupy most of the nucleoplasm (B) and parts of the cytoplasm (C). The paracrystalline arrays formed by the viruses can be well appreciated at higher magnification (inset in B). The nuclear viral inclusions can be unequivocally distinguished from the nucleolus (Nu in B).

Infective particles:

Properties of infective particles. It is found that when virus particles are harvested from infected tissues and purified, they are composed mainly of a nucleoprotein. The particles associated with a given disease are

frequently found to be uniform in size and shape so that they can be conveniently termed macromolecules. In the study of viruses, it is observed that cursory examination of electron micrographs or ultracentrifuge patterns reveals evidence of polydispersity with respect to the size of the virus particles. Physical measurements made on objects as small as viruses must involve great numbers of particles. The process of identification consists in the separation of a virus-containing suspension. Separation may be achieved in several ways. One method is to disperse an aliquot of the particles for direct—but destructive—observation; the separation is then made mentally by recording numbers of objects in different morphological classes. The other methods require the virus suspension to be handled in a nondestructive manner because the material as separated must be assayed in that condition. Because virus particles are noninfectious subsequent to electron microscopy, morphologies must be related with infective assay by use of aliquot samples.

Kappa particles inheritance:

Kappa particles are found in certain killer strains of Paramecium and are responsible for production of substance **paramecin,** which is toxic to strains not possessing **kappa** (sensitive strain). The production of kappa particles is dependent on a dominant allele K, so that killer strains are KK or Kk and sensitive strains are ordinarily kk. In absence of dominant allele K, kappa particles cannot multiply and in absence of kappa particles, dominant allele K cannot produce them de novo. Consequently sensitive strains with genotypes KK or kk can be obtained. These will not carry any kappa particles. However, killer strain with genotype kk cannot be obtained, because even if kappa particles are present, these would be lost in absence of dominant allele.
If Paramecium clones with genotypes KK or Kk are allowed to multiply asexually at such a fast rate, that division of kappa particles cannot keep pace with division of cells, kappa particles will be eventually lost. Consequently sensitive strains with dominant genotype (KK, Kk)having

no kappa particles would be obtained.

If the killer (KK)and sensitive (kk)strains are allowed to conjugate, all exconjugants (the cells separating after conjugation) will have same genotype Kk. Phenotypes of these exconjugants will, however, depend upon duration for which conjugation is allowed. If conjugation does not persist long enough for exchange of cytoplasm, heterozygote (Kk)exconjugants will only have parental phenotypes. It means that killers will remain killers and sensitive will remain sensitive even after conjugation. If conjugation persists, sensitive strain will receive kappa particles and will become killer, so that exconjugants will be killers having genotype Kk.

Maternal Inheritance:

Maternal inheritance are of two types:

i. If some treatments (chemical poison, heat shock etc.) are applied to the female parent, it may affect the egg's cytoplasm. As a result subsequent offspring's are modified in some way. Effects of this kind are called Dauer-modifications or persisting modifications.

It is observed that when protozoa are treated experimentally with chemical poisons or heat shocks, the treatments induce several morphological abnormalities in them. Such abnormalities go on decreasing generation after generation and, eventually, disappear completely through cell division if the treatments are removed.

Further evidences also come from fruit flies subjected to heat treatment and from bacteria treated with chemicals.

ii. Other kinds of maternal inheritance are also known which do not depend upon the repeated application of an external stimulus to the

cytoplasm. In this case, maternal inheritance is truly controlled by independent cytoplasmic genes.

Maternal effects reflect the influence of the mother's gene on developing tissues. Many important characteristics of both animal and plants show maternal effects of which some examples axe described next.

(ii) **Coiling of Snail Shells (Limnaea Peregra):**

One of the earliest and classical examples of a maternal effect is that of the direction of coiling in shells of the water snail Limnaea peregra. In this snail, the shell is spirally coiled. Usually the direction of coiling of the shell is clockwise if viewed from the top of the shell. This type of coiling is called dextral. However, in some snails the coiling of shell is anticlockwise. This type of coiling is sinistral.

The direction of shell coiling of both types of snail is governed by genotype of the female parent and not by their own genotype. Further investigation suggests that coiling depends upon the early clearage in the zygote.

If the mitotic spindle is tilted to left of the median line of zygote, the successive cleavages will produce a spiral to left (sinistral) and if the orientation of spindle is tilted to the right of the median line of zygote, the successive cleavages will produce a spiral to right (dextral). The spindle orientation is controlled by the genotype of oocyte from which the egg develops.