

Sex-linked Inheritance in Man

The chromosomal mechanism of sex inheritance can be traced back to some of the experiments carried out in insects. A human female has 23 pairs of chromosomes, and a human male has 22 similar pairs and one pair consisting of two chromosomes that are dissimilar in size and structure. The 23rd pair in both the sexes is called sex chromosomes.

The inheritance of a trait determined by a gene located on one of the sex chromosomes is called sex-linked inheritance.

Sex-linked inheritance is the transmission of characters and their determining genes and sex-determining genes on the sex chromosomes, therefore, are inherited together from one generation to the next.

Most of the sex-linked genes are present on the X chromosome, resulting in X-linkage formation. A gene that occurs on the Y chromosome forms Y-linkage. The Y-linked traits are transmitted only through the male. Females are usually carriers of X-linked diseases. As they are 'X' linked, fathers never transfer hemophilia or color blindness to their sons. Examples of sex-linked human

diseases are hemophilia and color blindness. Besides sex-linked inheritance, sexlimited genes and sex-influenced traits have also been observed.

Sex-limited traits: Traits controlled by autosomal genes, whose expression is limited to one of the sexes because of sex hormones, e.g., secondary sex characters, milk production in mammalian females, egg production in chicken, premature baldness in human males.

Sex-influenced traits: Traits controlled by autosomal genes, which are expressed as dominant in one sex and as recessive in the other because of sex hormones, e.g., white forelock, harelip, gouts, certain types of pattern baldness are all dominant in men, spina bifida is dominant in women.

Some examples of sex-linked inheritance are hemophilia, red-green color blindness, congenital night blindness, some high blood pressure genes, and fragile X syndrome.

Types of sex-linked Inheritance

Sex-linked inheritance is of two types.

X-linked inheritance

When certain sex-linked genes are located only on X-linked chromosomes, their alleles are absent from Y-chromosome.

Dominant X-linked inheritance:

Whey Single x-chromosome has the gene of a character that expresses itself in the absence of another alternate gene known as dominant X-linked inheritance. Example-Rett syndrome.

Recessive X-linked inheritance:

Sometimes only one x-chromosome bears the gene of characters, and characters do not express themselves in the absence of another alternate gene. But when both chromosomes bear the gene of a character, then the character expresses itself; such a condition is called recessive X-linked inheritance. Example-Hemophilia.

Y-linked inheritance

When Y-chromosome bears the gene of a character and expresses itself only in males it is known as Y-linked inheritance. Example-hypertrichosis of the ears

Characteristic of sex-linked Inheritance

As we all know that sex-linked inheritance is a criss-cross inheritance in which the father does not pass the sex-linked allele of a trait to his son. The same is passed to the daughter, from where it reaches the grandson, which is digenic inheritance.

In sex-linked inheritance, the mother who is in a homozygous state passes the alleles of a sex-linked trait to both sons and daughters.

The majority of the sex-linked traits are present on the X chromosome, which is a recessive gene. Hence we conclude that the majority of sex-linked traits are recessive.

In males, sex-linked traits show more continually in comparison to that of females.

Males suffer more from sex-linked disorders because they are heterozygous, and traits frequently show in a heterozygous state.

As we all know, recessive genes can only express themselves when they are in a homozygous state, and female contains homozygous gene and generally function as a carrier of sex-linked disorders.

Sex-linked non- criss-cross inheritance is holandric (if it passes directly from father to son) and Hologic (if it passes directly from mother to daughter).

Most common disorders

≻Haemophilia - A, B

≻Colour blindness

>Duchenne muscular dystrophy

≻Becker's muscular dystrophy

➤X – linked ichthyosis (STS- Steroid sulfatase)

≻X – linked agammaglobulinemia (XLA)

≻Glucose – 6 – phosphate dehydrogenase deficiency

Haemophilia is an X – linked recessive disorder. It is of two types Haemophilia-A and Haemophilia -B. they are caused by lethal genes

1)Haemophilia – **A** is also known as royal disease as it was first identified in royal family of Queen Victoria and some other families. This is due to lack of blood protein called clotting factor VIII (AHG- anti haemophilic globulin).

2)Haemophilia - B is also known as bleeder's disease because the person suffering with this disease has an inability of their blood to clot normally. This is due to the lack of blood protein called clotting factor IX (Christmas factor).

In the inheritance pattern if the female is possessing one recessive X chromosome for haemophilia (XH) she will not be affected by the disease but she will be a carrier to the disease. She will be affected only when she possess both X recessive chromosomes (XHX H). in case of males if he possess one X recessive chromosome for haemophilia (XH) he will be affected by the disease. He passes this character to his grandson through his daughter If the mother is a carrier (XHX) and father is normal (XY), the offspring received X chromosome from mother possess character of haemophilia, in which the daughter will be carrier and son will be haemophilic.



If the mother is a carrier (XHX) and father is haemophilic (XHY), the daughters who receive recessive X chromosome from mother and father will be affected and those who receive normal X chromosome from mother will be carrier as they receive affected X chromosome from father. Sons who receive recessive X chromosome from mother will be haemophilic and those who receive normal chromosome will be normal. Males does not receive X chromosome from father will not be carriers.





b) Colour blindness is another common X-linked recessive disease in which the people

have a defect in the cones of retina. Lack of colourable pigment in retinal cones is known as Deuteranopia or deutan colour blindness. Lack of erythrolable pigment for differentiating the red and green is known as Protanopia or protan colour blindness. Colour blindness is detected by Ishihara's test introduced by Dr. Shinobu Ishihara.

Example 1: Cross between a normal woman and a colourblind man. If a colour blind male marries with a normal female the possibilities would be – All children (son and daughters) would be normal, but the daughters would be carriers. Because they are heterozygous, they will not be colourblind. The woman acts as a carrier of colourblindness.





Conclusion: Following are conclusions regarding heredity of colour blindness:

1. Identification of colours in the vision is a sex-linked character. Its gene is

located on the X chromosome and its allele is not found in males.

2. Males are more sufferer from this disease. Because if the normal gene for colour differentiation is absent on X chromosome man than the colour blindness the disease is found.

3. Women have two X-chromosomes and both X chromosomes have the gene for colour identification and expression.

4. While only one chromosome in man has a gene for colour identification and expression. If only one X chromosome carries the recessive gene for colourblindness in woman than the normal woman becomes the carrier of the disease.

5. Gene for the identification of colours is dominant. If both X chromosomes of woman carry this recessive gene then a woman will be colour-blind.

6. Father and son of a colourblind woman are also colour blind. If the husband of a colour blind woman is also colour blind than their daughters will be colour blind. Daughter (normal) of a colour blind father produce 50% sons of normal vision and 50% sons as colourblind.

2)X-Linked Dominant Inheritance It is a pattern of inheritance where there the dominant gene for a disease is carried on the X chromosome and only one is sufficient to cause the disease both in males and females. EX.- Follicular hyperkeratosis (excess keratin in hair follicles), Incontinentia pigmenti (loss of melanin), Fragile X-syndrome and Mental retardation

II) Y – Linked Inheritance Genes that are present on the non-homologous region of the Y chromosome and are inherited from father to the grandson through son is known as Y-linked

inheritance. This pattern of inheritance is also known as Holandric Inheritance and the genes responsible for this inheritance are called Holandric genes. In humans holandric inheritance is signified by disease called **Hypertrichosis** or Ichthyosis hystrix gravis hypertrycosis. The disease is identified by the presence of excess hair on the body completely or restricted to a particular region like external pinna. Porcupine man is also caused by holandric inheritance.

III) XY- Linked Inheritance This is a peculiar type of inheritance where the genes are present on homologous region of X and Y chromosomes. This type of inheritance occurs when the mutant X and mutant Y chromosomes pair during fertilization. These genes are called as Pseudo autosomal genes or incompletely or partially sex-linked genes. In humans this inheritance causes Total colour blindness, skin diseases etc.

Based on inheritance of the sex-linked genes and moving from one sex to another sex, sex linked inheritance is classified as

> Diandric inheritance \rightarrow Mother \rightarrow son \rightarrow Granddaughter (Criss cross inheritance)

>Diagenic inheritance \rightarrow Father \rightarrow Daughter \rightarrow Grandson (Criss cross inheritance)

>Holandric inheritance \rightarrow Father \rightarrow son \rightarrow Grandson

> Hologenic inheritance \rightarrow Mother \rightarrow Daughter \rightarrow Granddaughter (Non-Criss cross inheritance).

PEDIGREE CHART

Introduction

Pedigree analysis is a scientific approach to studying the inheritance of human genes.Pedigree analysis can be used to easily understand family history and the inheritance of dominant and recessive traits. With the help of a pedigree chart, one can easily identify dominant and recessive traits and also the carriers of these traits.

Pedigree Chart

A pedigree chart is a diagrammatic representation (chart or family tree) where one can easily determine the occurrence of a phenotype in an organism's genes passed down from their ancestors. One can study family history through pedigree charts that might have faded away over generations.Pedigree charts are commonly used for humans, racehorses, and dogs. They utilise standard symbols: a circle represents females, and a square represents males.

Diagrammatic Representation of Pedigree Charts

Relationships are presented diagrammatically in a pedigree chart.

The parents are connected by a horizontal line, with a vertical line leading to their offspring.

A horizontal zip line connects all the offspring of the same parents. They are placed from left to right according to their order of birth.

Twin offspring are connected by a triangle.

If an offspring is no more, there is a line across it.

A small triangle represents if the offspring was aborted or has not yet been born.

Different generations are represented by Roman letters, and a numeral represents offspring of the same generation. If the pedigree chart is constructed for a family with a genetic disorder, this is represented by an arrow.



X-linked dominant pedigree

X-linked recessive pedigree

2) Autosomal pedigree

Autosomal dominant pedigree

Importance of Pedigree Diagrams

The two important uses of pedigree analysis are with:

Human usage

Pedigree analysis for humans is used to check the likelihood of a particular disorder and condition

Pedigree analysis helps locate genes such as X, Y, and autosomal chromosomes

It helps predict whether the trait is dominant or recessive

If the likelihood is 50:50 (mother and father), it is called an autosomal condition

If the pedigree analysis reveals that a male is affected by a genetic disorder, it is known as an X-linked disorder

Animal usage

The ancestors of animals can be found

Horse and dog breeds with suitable traits can be obtained

Characteristic Predictions through Pedigree Analysis

While the pedigree analysis does not help predict characteristics like height, other features, such as hair colour, blood group, and eye colour, can be easily found. To understand pedigree analysis, one needs to be clear about dominant and recessive genes.

Let's consider an example. The colour blindness trait can be found in the X or Y chromosome. However, as this trait is common in the X chromosome, colour blindness commonly occurs in males. However, females can also be affected if they

inherit the X chromosome with colour blindness from the affected mother or father. A shaded dot or half-shaded symbol represents the carrying of any defective traits.

Autosomal Recessive Inheritance

Some genetic conditions are autosomal recessive, meaning that the gene involved is found on an autosome, and affected individuals have two copies of the allele that causes the condition. If an affected individual in a pedigree has two unaffected parents, the condition is most likely recessive. Additionally, if daughters in the pedigree have two unaffected parents, the condition is most likely autosomal recessive (unlike Xlinked recessive conditions, in which an affected daughter will have an affected father). With autosomal recessive inheritance, males and females are equally likely to be affected.

utosomal recessive conditions in humans include cystic fibrosis, sickle cell disease, Tay-Sachs disease, and phenylketonuria.



example of a pedigree chart for an autosomal recessive condition. From this pedigree, we can infer that individuals 1, 2, 3, 4, and 11 are carriers. There is not enough information to determine the genotype of individuals 5, 6, 8, and 9. (Autosomal recessive pedigree.

It is helpful to be able to assign genotypes to all individuals on a pedigree chart in order to predict if it is possible that an individual might pass along a mutant allele to

their offspring. When the inheritance mechanism is autosomal recessive, it is helpful to start with affected individuals. All affected individuals are expected to be homozygous recessive in genotype (for example, genotype aa).

Autosomal Dominant Inheritance

Genetic conditions can display autosomal dominance. In this mode of transmission, a single mutant allele is sufficient to cause the condition because the mutant allele is dominant over the normal allele. Affected children generally have at least one affected parent (although not always, because some conditions display incomplete penetrance, meaning that not every individual with the allele will display the phenotype). Males and females are equally affected.



Autosomal Dominant Inheritance

X-linked Recessive Inheritance

Some conditions are sex-linked, meaning the gene that causes the condition is on one of the sex chromosomes. In mammals, this is usually the X chromosome, because the X chromosome is much larger and has many more genes. X-linked recessive conditions are much more common in males, because they have only one X chromosome. Therefore, if they inherit an X chromosome with the mutated allele that causes the condition, they will display the condition. Females must inherit two copies



Conclution

Pedigree Analysis - Key takeaways Pedigrees can help us to analyze the inheritance patterns of many traits Pedigrees are typically used in the setting of genetic disorders The most common inheritance patterns include autosomal recessive, autosomal dominant and X-linked recessive. Some other less common inheritance patterns include X-linked dominant, Y-linked and mitochondrial inheritance. To solve a pedigree analysis, first look for dominance, than look for possible sex-linkage.

CYTOPLASMIC INHERITANCE

Definition

The Transmission of characters controlled by plasma genes is called Cytoplasmic Inheritance or Extra Chromosomal Inheritance. Described by Correns in 1908. Plasmogenes or Self replicating and transmitted by cytoplasm only. Like Chromosomal genes, these are also capable of mutation. The offsprings receive Cytoplasm only from female gamete, not from male gamete. As a result, Cytoplasmic inheritance is known as Maternal Inheritance (Plasma genes of female parent alone are contributed to the offsprings) The results of reciprocal crosses are not same in Cytoplasmic Inheritance.



Shell coiling in Snail

• In Snail Limnaea, the nature of shell coiling is under cytoplasmic inheritance. The phenotype of the offspring is determined by the genotype of the female parent Maternal inheritance. In shell coiling, the genotype of the female parent is not expressed in its own body, but in the offsprings of F1 generation hence it is called delayed inheritance. In reciprocal crosses, results are different.

• Shell coiling – 2 types

(i) Dextral (clockwise)



• The dextral shell is dominant- genes DD

• The Sinistral shell is recessive- genes dd

• When female dextral snail(DD) is crossed with male Sinistral snail(dd) - F1

snails(Dd) dextral like female parent. When female sinistral snail (dd) is crossed

. 1917 - 1911 - 1917 - 1911 - 1911 - 1917 - 1911 - 1911 - 1917 - 1911 - 1917 - 1911 - 1911 - 1917 - 1911 - 1917 -

with male dextral snail(DD)- F1 snails(Dd)

sinistral like female parent.





In the above 2 crosses, F1 Snails have same genotypes, but different phenotypes.

• Here phenotype of offsprings determined by genotype of mother.

• In first cross, the offsprings : Dextral shell because mothers genotype is "DD"

In second cross, the offsprings : Sinistral shell because mothers genotype is "dd"
Thus in reciprocal crosses, results are different. The F2 generation is obtained by seil fertilization of single snail (hermaphrodite) When dextrally coiled F1 Snail (Dd) is self fertilized F2 offsprings appear in the genotypic ratio 1DD:2Dd:1dd. But Phenotypically, all the F1 individuals are dextral, because the parental genotype(Dd has dominant gene D. When Sinistral F1 Snail(Dd) is self fertilized, all the F2 offsprings dextral because parental genotype(Dd) has 'D' gene. The inheritance of shell coiling follows simple mendelian character. In F2 generation the genotypes appear in ratio1DD:2Dd:1dd. The phenotypic ratio 3:1 of F2 generation appear only in F3 generation. This is due to delayed inheritance.

KAPPA PARTICLES IN PARAMECIUM

Two Strains in Paramecium (i) Killer (ii) Sensitive

Killer Strain produce toxic substance - Paramecin, which kills the other type.

Production of Paramecin in killer type controlled by Cytoplasmic Particles – "Kappa Particles".

In sensitive Strain lack Kappa Particles.

The Kappa Particles pass from one generation to other during cell division.

These particles also multiply during division & transmitted through Cytoplasm.

The multiplication of Kappa particles is controlled by dominant nuclear gene **"K"**. **The gene "K" can only maintain Kappa particles but cannot initiate its production.**

When killers **KK** conjugate with non-Killers **kk**, the ex- conjugants are **Kk**. The development of particular type depends on the duration of Cytoplasmic exchange. In normal case of Conjugation, the nuclear material alone is exchanged & there is no exchange of Cytoplasmic material. In such cases, each ex conjugant gives rise to the organism of its own type i.e. the killers -> killers & non killer -> non killer ex conjugnt. Sometimes the conjugation period is prolonged & cytoplasmic bridge between the two conjugant is larger. In such cases, in addition to the nuclear material, the cytoplasmic material are also exchanged. During this time, the Kappa particles present in the cytoplasm of the killer type enter the non killer type& convert it into killer type. Hence, the offsprings produced by the ex conjugants are killer type. This shows that paramecium becomes a killer, when it receives Kappa

particles & it becomes sensitive when it does not deceive Kappa Particles.

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 (Fig. 18.5). If conjugation persists, sensitive strain will receive kappa particles and will become killer, so that exconjugants will be killers having genotype Kk





ABO blood groups in humans

On the basis of presence or absence of certain antigens, ABO blood groups have been established in man by **K. Landsteiner. It** was found that there can be two antigens A or B and as a result four blood groups. These blood groups are called ABO blood groups or Landsteiner blood groups after the name of the discoverer.

With these antigens A and B, there are certain naturally occurring antibodies in the serum of the blood. General principle of antibody and antigen relationships is, that antibodies in a particular individual will be found only for those antigens which are absent in blood of this individual.

Blood groups	Antigen	Antibody for antigen
А	А	В
В	В	A
AB	A and B	None
0	None	A and B

Antibodies in blood group A will be able to agglutinize red blood corpuscles of the blood group B and vice versa. AB blood group will not agglutinize any other group, since no antibodies are present. Similarly, O blood group should be able to agglutinize all other three groups except its own. On this relationship is based the test whether a particular blood group can be donated for another specific blood group.

If donor's blood has antibodies against that of recipient, it does not matter, since they get diluted. However, there should be no antibodies in recipient's blood against donor's blood, since this will cause agglutinization of transfused blood. It would also be obvious, that AB is **universal recipient** and no test of donor's blood in such case is necessary. Similarly, O group is **universal donor** and no test for the recipient's blood is necessary, if blood of O group is available for transfusion.

Blood group of donor	Blood group of recipient	
A	A and AB	
В	B and AB	
AB	AB (universal recipient)	
O (universal donor)	O, A, B, AB	



Fig. Effects of anti-A (containing antibody A) and anti-B (containing antibody B) sera on corpuscles of four blood groups (O, A, B and AB). A clumped pattern within a circle shows agglutinization of red blood corpuscles.

In ABO blood groups discussed above, **antigens** (also called **agglutinogens**) are present in the red blood cells and **antibodies** (also called **agglutinins**) in the serum. Consequently, transfusions can be made after examining the compatibilities of blood groups, so that the transfused blood may not get agglutinated. As shown, the blood group A contains anti-B serum (antibodies for B antigen) and blood group B contains anti-A serum. The genetics of these blood groups indicates that three alleles are present (i) 1° or i or + (ii) IA or A and (iii) IB or B. 1A and IB are mutant alleles and are dominant over the wild allele 1° or i or +. This is based on the concept that wild type is one which is more frequent. O and AB blood groups are more frequent and if O is assumed as wild, it yields a convenient system.

Blood group	Possible genotypes
0	$I^{O}I^{O}$ or <i>ii</i> or ++
A	$I^{A}I^{A}$ or AA ; $I^{A}I^{O}$ or $I^{A}i$ or $A/+$
В	$I^{B}I^{B}$ or BB ; $I^{B}I^{(i)}$ or $I^{B}i$ or $B/+$
AB	$I^A I^B$ or A/B

MN series of blood groups in humans

Besides series of ABO blood groups, there are other series like MN blood groups. In case of MN series, human blood serum does not contain any specific antibodies, which will cause agglutinization of any blood group in this series. But if the human blood is injected in rabbit, it causes production of specific antibodies responsible for agglutinization of blood type injected. For instance, if blood of M group is injected in rabbit, it will cause production of antibodies in serum (anti-M serum) causing agglutinization of M as well as MN but not of N. Reverse will be the case, when blood of N type is injected in rabbit, so that anti-N serum produced in rabbit causes agglutinization of N and MN, but not of M.

Landsteiner and Levine (1927) divided human populations into three groups on the basis of reaction with anti-M serum and anti-N serum produced in rabbits. In honour of Landsteiner, the gene for this MN series was named as L and alleles as LM and LN, also now called M and N (Table 5.10). These alleles are codominant, so that the heterozygote LMLN gives rise to the blood group MN.

Blood group Genotypes	Genotynes	Cellular	Serum anti-	Reactio seru	on with 1m*
	antigen	body	Anti-M serum	Anti-N serum	
М	$L^M L^M$ or MM	М	None	+	-
MN	$L^{M}L^{N}$ or MN	M, N	None	+	+
N	$L^{N}L^{N}$ or NN	N	None	_	+

New varieties of MN blood types were discovered

by **Walsh** and **Montgomery** in 1947. A new blood-type system S3 was shown to be closely tied to the inheritance of MN gene system. Thus four alleles, namely LMS, LMs, LNS, LNs (or MS, Ms, NS, Ns) could be identified and nine blood groups were possible .

Blood	Genotypes	Reaction with immunized ser			
group	Genotypes	anti-M	anti-N	anti-S	anti-s
MS	L ^{MS} L ^{MS} or M ^S M ^S	+	-	+	_
MSs	$L^{MS}L^{Ms}$ or $M^{S}M^{s}$	+		+	+
Ms	$L^{Ms}L^{Ms}$ or $M^{s}M^{s}$	+	-	-	+
MNS	$L^{MS}L^{NS}$ or $M^{S}N^{S}$	+	+	+	-
MNSs	$L^{MS}L^{Ns}$ or $M^{S}N^{s}$	+	+	+	+
	or				
	$L^{Ms}L^{NS}$ or $M^{s}N^{S}$				
MNs	L ^{Ms} L ^{Ns} or M ^s N ^s	+	+	-	+
NS	$L^{NS}L^{NS}$ or $N^{S}N^{S}$	_	+	+	-
NSs	$L^{NS}L^{Ns}$ or $N^{S}N^{s}$	-	+	+	+
Ns	$L^{Ns}L^{Ns}$ or $N^{s}N^{s}$	-	+	-	+

Blood groups and disputed parentage

Blood groups sometimes help to decide cases of disputed parentage in criminal courts, because a particular pair of blood groups in parents may give some and

not all blood groups in progeny. Therefore, if a child has a blood group, which is not likely to result from the blood groups of a married couple claiming parentage, then it is proved that the child has a doubtful parentage or in other words is illegitimate. Various possible pairs of blood groups of the parents, their children and the impossible blood groups are given in Table.

ABO blood groups

Parents with blood groups and genotypes	Progeny with blood groups and genotypes	Improbable blood groups
$O(l^0 l^0) \times O(l^0 l^0)$	O (1°1°)	A, B, AB
$2 O (I^0 I^0) \times A (I^A I^A, I^A I^0)$	O (1010), A (110)	B, AB
3. $O(I^0I^0) \times B(I^{B}I^{B}, I^{B}I^0)$	$O(I^O I^O)$, $B(I^B I^O)$	A, AB
4. $O(I^0I^0) \times AB(I^AI^B)$	A $(I^A I^O)$, B $(I^B I^O)$	O, AB
5. A $(I^AI^A, I^AI^O) \times A (I^AI^A, I^AI^O)$	A (1A1A, 1A1O), O (1010)	B, AB
6 A (PAPA PAPO) × B (PBPB PBPO)	A (1^10), B (1810), AB (1^18), O (1010)	none
7 A $(I^A I^A I^A I^A I^G) \times AB (I^A I^B)$	A $(I^{A}I^{A}, I^{A}I^{O})$, B $(I^{B}I^{O})$, AB $(I^{A}I^{B})$	0
S B $(I^B I^B I^B I^O) \times AB (I^A I^B)$	A (1 ^A 1 ^O), B (1 ^B 1 ^B , 1 ^B 1 ^O), AB (1 ^A 1 ^B)	0
0 AB $(I^A I^B) \times AB (I^A I^B)$	A $(I^A I^A)$, B $(I^B I^B)$, AB $(I^A I^B)$	0

MN blood groups

Parents with blood groups and genotypes	Progeny with blood groups and genotypes	Improbable blood group:
$M(L^{M}L^{M}) \times M(L^{M}L^{M})$	$M(L^M L^M)$	N, MN
$M(L^{M}L^{M}) \times N(L^{N}L^{N})$	MN $(L^M L^N)$	M, N
$M(L^M L^M) \times MN(L^M L^N)$	$1 \text{ M} (L^{M}L^{M}) : 1 \text{ MN} (L^{M}L^{N})$	N
$N(L^{N}L^{N}) \times N(L^{N}L^{N})$	$N(L^{N}L^{N})$	M, MN
$N(L^{N}L^{N}) \times MN(L^{M}L^{N})$	$1 \text{ N} (L^{N}L^{N}) : 1 \text{ MN} (L^{M}L^{N})$	м
MN $(L^{M}L^{N}) \times MN (L^{M}L^{N})$	$1 \text{ M} (L^M L^{\hat{M}}) : 2 \text{ MN} (L^M L^{\hat{N}}) : 1 \text{ N} (L^N L^{\hat{N}})$	None

Rh blood groups and erythroblastosis fetalis

Rh factor was discovered by **Landsteiner** and **Wiener** in 1940 from rabbits immunized with the blood of **rhesus monkeys** (Macacus rhesus). The resulting antibodies in rabbit serum agglutinated blood of not only rhesus monkeys, but also of certain percentage of human beings and these human beings were called Rh+ (Rh positive). The Rh antigen can produce antibodies in human serum also, which may be possible through wrong transfusion of blood. Therefore, to avoid agglutinization, cross-compatibility of Rh factor as well as ABO blood groups is necessary before transfusion of blood is made. Rh negative individuals should always be given Rh-negative blood to avoid subsequent reaction due to antibody formation.

A serious problem occurs, when father is Rh positive and mother is Rh negative. In such cases all children born will be Rh positive, if the father is homozygous (RR). Similarly, half the children will be Rh positive, if the father is heterozygous (Rr). If Rh negative mother carries a Rh positive fetus, in the first case of pregnancy, no serious problem due to Rh+ antigen in child's blood arises, since the concentration of antibodies produced in mother's blood due to immunization by child's Rh+ antigen, will be rather low. But subsequent Rhpositive children will increase the concentration of antibodies in mother's blood due to immunization and this blood while circulating through the fetus may cause death of the **fetus** due to **homolytic jaundice** and **anemia**. This disease is called **erythroblastosis fetalis**.

IN BREEDING AND OUT BREEDING

Mating between closely related individuals is called inbreeding.

Self fertilization is an ideal inbreeding.

Mendel carried out inbreeding among the F1 plants in his monohybrid and dihybrid experiments.

Breeding system	Inbreeding	Close inbreeding	
		Line inbreeding	
-		Pure breeding	
	Outbreeding	Line crossing	
		Out crossing	
		Cross breeding	
		Grading up	
		Species Hybridization	

Production of Pure lines - Inbreeding produces homozygotes. The homozygotes reproduce only homozygotes by inbreeding. These homozygotes are pure lines as they breed pure. Hence inbreeding produces pure lines.

Elimination of Deleterious Recessive Characters – Inbreeding produces recessive homozygotes. If the recessive character is deleterious, it is possible to eliminate the undesirable recessive genes from the population by repeated inbreeding and selection.

Production of Valuable Breeds - High quality commercial breeds of plants and animals are produced by inbreeding followed by selection. Best races of horses. dogs, bulls and sheep are produced by inbreeding and selection. This kind of inbreeding is also called line breeding.

Intraspecific outbreeding is the mating between the of the same species.

Interspecific outbreeding is the mating between the of different species.

Intergeneric outbreeding is the mating between the of different genera.

The hybrids are stronger, heavier and vigourous than their parents.

The superiority of the hybrid is called hybrid vigour or heterosis.

Hybrid vigour brings the following effects in plants and animals.

It increases the height, viability, fertility, resistance to environmental factors, disease and pests.

It increases the size of the fruits, seeds and leaves.

It increases the yield of the crop.

It causes better germination and growth rate.

It initiates early flowering and fruit setting.

It increases milk production.

It increases number of eggs in poultry.

It produces better beef and pork.

It increases in silk production.

Mule is a hybrid. Mule is born for a male donkey and a female horse.

Mule is superior to a horse in strength, ability to and resistance to disease.

Mule is more intelligent than their parents.

Thus mule exhibits hybrid vigour.

However mule is sterile and cannot produce another mule.

Mule is employed in Indian army in Himalayan mountain.

