

SUPPLEMENTARY MATERIAL

UNIT VI: CHAPTER 1; 1.1, PAGE 6 AT THE END OF FIRST PARA

Under unfavourable condition the *Amoeba* withdraws its pseudopodia and secretes a three-layered hard covering or cyst around itself. This phenomenon is termed as **encystation**. When favourable conditions return, the encysted *Amoeba* divides by multiple fission and produces many minute amoeba or pseudopodiospores; the cyst wall bursts out, and the spores are liberated in the surrounding medium to grow up into many amoebae. This phenomenon is known as **sporulation**.

UNIT VI: CHAPTER 1; 1.1, PAGE 7 AT THE END OF SECOND PARA

In some organisms, if the body breaks into distinct pieces (fragments) each fragment grows into an adult capable of producing offspring (e.g., *Hydra*). This is also a mode of asexual reproduction called **fragmentation**.

UNIT VII: CHAPTER 5; PAGE 85 (TO BE GIVEN AS SECTION 5.4)

POLYGENIC INHERITANCE

Mendel's studies mainly described those traits that have distinct alternate forms such as flower colour which are either purple or white. But if you look around you will find that there are many traits which are not so distinct in their occurrence and are spread across a gradient. For example, in humans we don't just have tall or short people as two distinct alternatives but a whole range of possible heights. Such traits are generally controlled by three or more genes and are thus called as polygenic traits.

Besides the involvement of multiple genes polygenic inheritance also takes into account the influence of environment. Human skin colour is another classic example for this. In a polygenic trait the phenotype reflects the contribution of each allele, i.e., the effect of each allele is additive. To understand this better let us assume that three genes A, B, C control skin colour in human with the dominant forms A, B and C responsible for dark skin colour and the recessive forms a, b and c for light skin colour. The genotype with all the dominant alleles (AABBCC) will have the darkest skin colour and that with all the recessive alleles (aabbcc) will have the lightest skin colour. As expected the genotype with three dominant alleles and three recessive alleles will have an intermediate skin colour. In this manner the number of each type of alleles in the genotype would determine the darkness or lightness of the skin in an individual.

UNIT VII: CHAPTER 5; PAGE 85 (TO BE GIVEN AS SECTION 5.5)

PLEIOTROPY

We have so far seen the effect of a gene on a single phenotype or trait. There are however instances where a single gene can exhibit multiple phenotypic expression. Such a gene is called a pleiotropic gene. The underlying mechanism of pleiotropy in most cases is the effect of a gene on metabolic pathways which contributes towards different phenotypes. An example of this is the disease phenylketonuria, which occurs in humans. The disease is caused by mutation in the gene that codes for the enzyme phenyl alanine hydroxylase (single gene mutation). This manifests itself through phenotypic expression characterised by mental retardation and a reduction in hair and skin pigmentation.

UNIT VII: CHAPTER 5; 5.4, PAGE 86 AFTER SECOND PARA

SEX DETERMINATION IN HONEY BEE

The sex determination in honey bee is based on the number of sets of chromosomes an individual receives. An offspring formed from the union of a sperm and an egg develops as a female (queen or worker), and an unfertilised egg develops as a male (drone) by means of parthenogenesis. This means that the males have half the number of chromosomes than that of a female. The females are diploid having 32 chromosomes and males are haploid, i.e., having 16 chromosomes. This is called as haplodiploid sex-determination system and has special characteristic features such as the males produce sperms by mitosis (Figure 5.13), they do not have father and thus cannot have sons, but have a grandfather and can have grandsons.

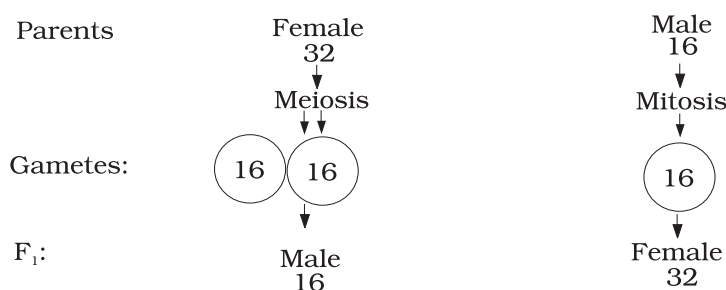


Fig.5.13 Sex determination in honey bee

**UNIT VII: CHAPTER 5; 5.6.2, PAGE 89 AFTER FIRST PARA****COLOUR BLINDNESS**

It is a sex-linked recessive disorder due to defect in either red or green cone of eye resulting in failure to discriminate between red and green colour. This defect is due to mutation in certain genes present in the X chromosome. It occurs in about 8 per cent of males and only about 0.4 per cent of females. This is because the genes that lead to red-green colour blindness are on the X chromosome. Males have only one X chromosome and females have two. The son of a woman who carries the gene has a 50 per cent chance of being colour blind. The mother is not herself colour blind because the gene is recessive. That means that its effect is suppressed by her matching dominant normal gene. A daughter will not normally be colour blind, unless her mother is a carrier and her father is colour blind.

UNIT VII: CHAPTER 5; 5.6.2, PAGE 90 AFTER SECOND PARA**THALASSEMIA**

This is also an autosome-linked recessive blood disease transmitted from parents to the offspring when both the partners are unaffected carrier for the gene (or heterozygous). The defect could be due to either mutation or deletion which ultimately results in reduced rate of synthesis of one of the globin chains (α and β chains) that make up haemoglobin. This causes the formation of abnormal haemoglobin molecules resulting into anaemia which is characteristic of the disease. Thalassaemia can be classified according to which chain of the haemoglobin molecule is affected. In α Thalassaemia, production of α globin chain is affected while in β Thalassaemia, production of β globin chain is affected. α Thalassaemia is controlled by two closely linked genes HBA1 and HBA2 on chromosome 16 of each parent and it is observed due to mutation or deletion of one or more of the four genes. The more genes affected, the less alpha globin molecules produced. While β Thalassaemia is controlled by a single gene HBB on chromosome 11 of each parent and occurs due to mutation of one or both the genes. Thalassaemia differs from sickle-cell anaemia in that the former is a quantitative problem of synthesising too few globin molecules while the latter is a qualitative problem of synthesising an incorrectly functioning globin.

UNIT VII: CHAPTER 7; 7.3, PAGE 129 AFTER SECOND PARA

Embryological support for evolution was also proposed by Ernst Haeckel based upon the observation of certain features during embryonic stage common to all vertebrates that are absent in adult. For example, the embryos of all vertebrates including human develop a row of vestigial gill slit just behind the head but it is a functional organ only in fish and not found in any other adult vertebrates. However, this proposal was disapproved on careful study performed by Karl Ernst von Baer. He noted that embryos never pass through the adult stages of other animals.

**UNIT X: CHAPTER 13; 13.1, PAGE 221 AT THE END OF FIRST
PARA**

Each organism has an invariably defined range of conditions that it can tolerate, diversity in the resources it utilises and a distinct functional role in the ecological system, all these together comprise its **niche**.

**UNIT X: CHAPTER 15; PAGE 266 IN FOURTH LINE OF 15.2.2
AFTER THE WORD THREATENED**

(organisms facing a very high risk of extinction in the wild in the near future)