

BIOLOGY UNIT 5

VARIATION AND SELECTION

The genetic code is contained within a chemical in the nucleus of the cell, called DNA. DNA is organised into units called genes. The discovery of the structure of DNA in 1953 was the beginning of a new discipline called molecular biology. Molecular biology looks at how the genetic code in the DNA affects the function of the cell. Molecular biology is closely linked with genetics, which concerns the visible effects of the genes on living organisms. This Unit starts with a look at how DNA forms the genetic code, and how it is passed from cell to cell when a cell divides. Chapter 18 deals with genetics – how the DNA shows itself in organisms. Chapter 19 describes the theory that underpins all of biology – Darwin's theory of evolution by natural selection and the principles of selective breeding.



16 CHROMOSOMES, GENES AND DNA

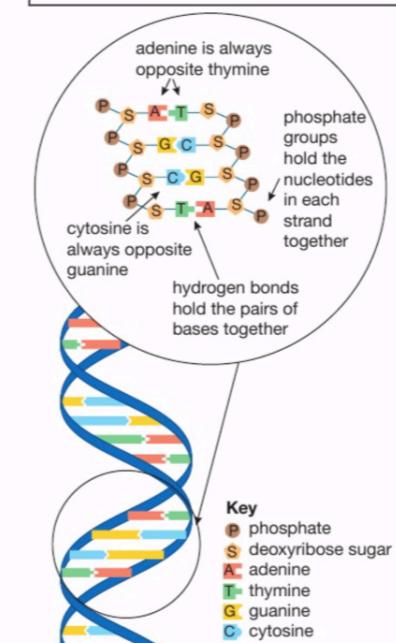
This chapter looks at the structure and organisation of genetic material, namely chromosomes, genes and DNA.

LEARNING OBJECTIVES

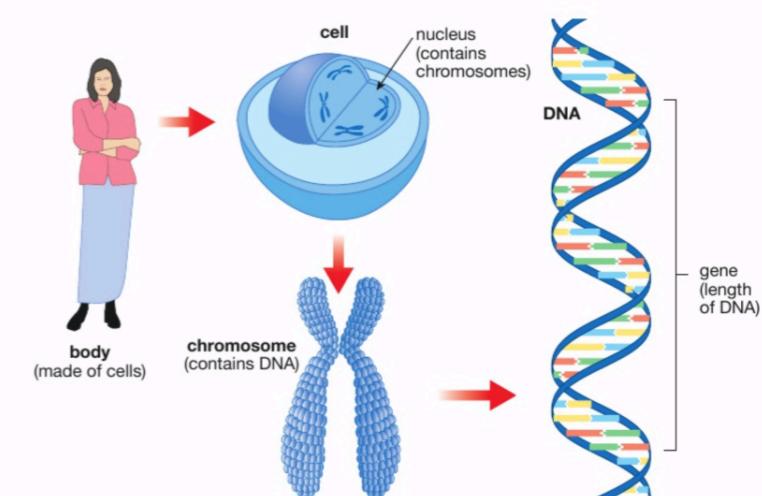
- Understand that the nucleus of a cell contains chromosomes on which genes are located
- Understand that a gene is a section of a molecule of DNA that codes for a specific protein
- Understand that mutation is a rare, random change in genetic material that can be inherited
- Know that in human cells the diploid number of chromosomes is 46 and the haploid number is 23
- Understand that the genome is the entire DNA of an organism
- Understand how genes exist in alternative forms called alleles, which give rise to different inherited characteristics

DID YOU KNOW?

DNA is short for **deoxyribonucleic acid**. It gets the 'deoxyribo' part of its name from the sugar in the DNA molecule – deoxyribose, a sugar containing five carbon atoms.



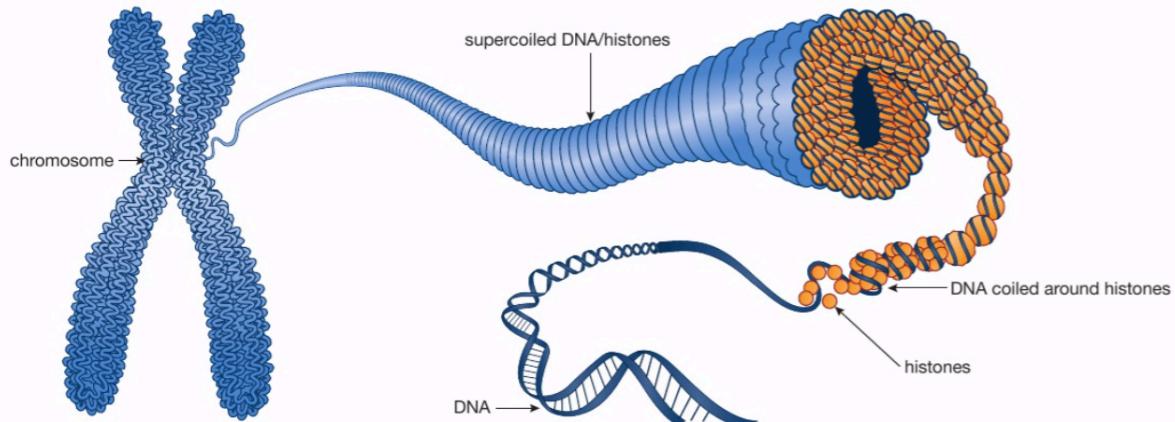
▲ Figure 16.2 Part of a molecule of DNA.



▲ Figure 16.1 Our genetic make-up

The chemical that is the basis of inheritance in nearly all organisms is **DNA**. DNA consists of two strands looking like a twisted ladder – the famous 'double helix'. The 'sides' of the ladder are made of alternating sugar and phosphate groups. The 'rungs' of the ladder are made of pairs of nitrogen-containing groups called bases (Figure 16.2). There are four bases called adenine, thymine, cytosine and guanine. The base adenine always pairs with thymine, and cytosine with guanine. These are called complementary base pairs. DNA is usually found in the nucleus of a cell, in the **chromosomes** (Figure 16.1). A small section of DNA that determines a particular feature is called a **gene**. Genes determine features by instructing cells to produce particular proteins which then lead to the development of the feature. So a gene can also be described as a section of DNA that codes for a particular protein.

DNA can replicate (make an exact copy of) itself. When a cell divides by mitosis (see Chapter 17), each new cell receives exactly the same type and amount of DNA. The cells formed are *genetically identical*.

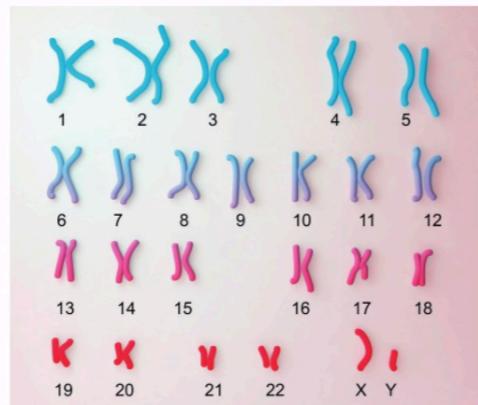


▲ Figure 16.3 The structure of a chromosome

Because a chromosome contains a particular DNA molecule, it will also contain the genes that make up that DNA molecule. Another chromosome will contain a different DNA molecule, and so will contain different genes.

HOW MANY CHROMOSOMES?

Nearly all human cells contain 46 chromosomes. The photographs in Figure 16.4 show the 46 chromosomes from the body cells of a human male.



▲ Figure 16.4 A man's chromosomes. One of each of the 22 homologous pairs are shown, along with the X and Y sex chromosomes. A woman's chromosomes are the same, except that she has two X chromosomes. A picture of all the chromosomes in a cell is called a karyotype.

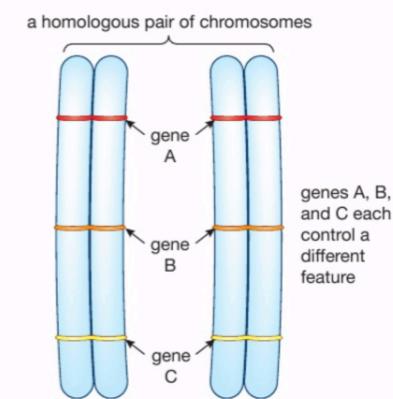
REMINDER

Red blood cells have no nucleus and therefore no chromosomes. (The lack of a nucleus means there is more room for carrying oxygen.)

KEY POINT

The X and the Y chromosomes are the **sex chromosomes**. They determine whether a person is male or female (see Chapter 18).

Pairs of matching chromosomes are called **homologous pairs**. They carry genes for the same features, and these genes are arranged at the same positions and sequence along the chromosome (Figure 16.5). Cells with chromosomes in pairs like this are **diploid** cells.



▲ Figure 16.5 Both chromosomes in a homologous pair have the same sequence of genes.

Not all human cells have 46 chromosomes. Red blood cells have no nucleus and so have none. Sex cells have only 23 – just half the number of other cells. They are formed by a cell division called **meiosis** (see Chapter 17). Each cell formed has one chromosome from each homologous pair, and one of the sex chromosomes. Cells with only half the normal diploid number of chromosomes, and therefore only half the DNA content of other cells, are **haploid** cells.

When two gametes fuse in **fertilisation**, the two nuclei join to form a single diploid cell (a **zygote**). This cell has, once again, all its chromosomes in homologous pairs and two copies of every gene. It has the normal DNA content.

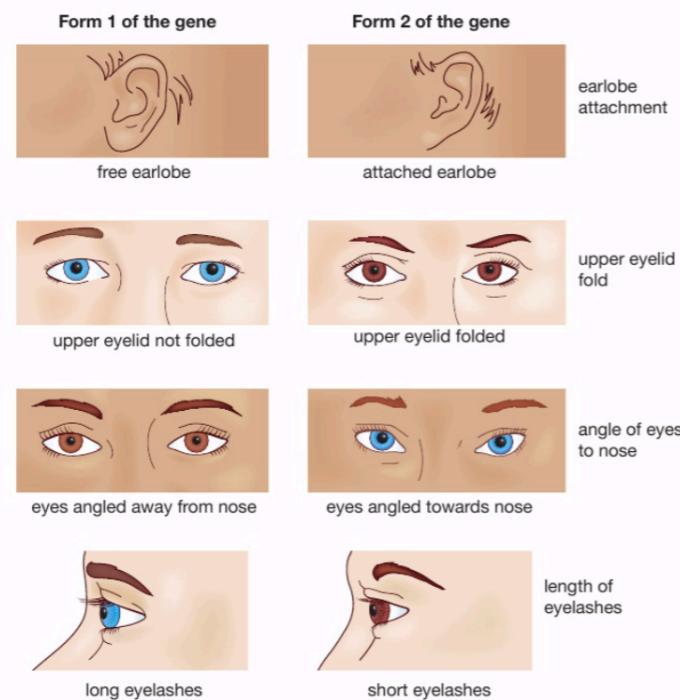
HOW MANY GENES?

The entire DNA of an organism (the amount present in a diploid cell) is known as its **genome**. The human genome is made up of about 3.2 billion **base pairs**. One of the surprise discoveries of modern molecular biology is that only a small fraction of the genome consists of protein-coding genes. For example, the human genome contains about 20 000–25 000 genes coding for proteins, which is only about 1.5% of the total DNA. The rest have other functions, or functions yet to be discovered! (See the 'Looking ahead' feature at the end of this chapter.)

GENES AND ALLELES

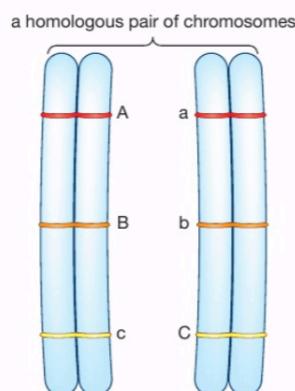
Genes are sections of DNA that control the production of proteins in a cell. Each protein contributes towards a particular body feature. Sometimes the feature is visible, such as eye colour or skin pigmentation. Sometimes the feature is not visible, such as the type of haemoglobin in red blood cells or the type of blood group antigen on the red blood cells.

Some genes have more than one form. For example, the genes controlling several facial features have alternative forms, which result in alternative forms of the feature (Figure 16.6).



▲ Figure 16.6 The alternate forms of four facial features

The gene for earlobe attachment has the forms 'attached earlobe' and 'free earlobe'. These different forms of the gene are called **alleles**. Homologous chromosomes carry genes for the same features in the same sequence, but the alleles of the genes may not be the same (Figure 16.7). The DNA in the two chromosomes is not quite identical.



▲ Figure 16.7 A and a, B and b, and C and c are different alleles of the same gene. They control the same feature but code for different expressions of that feature.

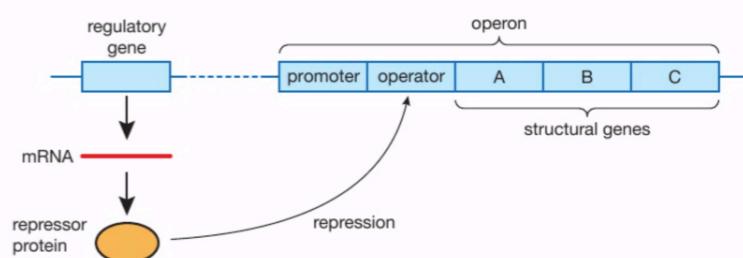
Each cell with two copies of a chromosome also has two copies of the genes on those chromosomes. Suppose that, for the gene controlling earlobe attachment, a person has one allele for attached earlobes and one for free earlobes. What happens? Is one ear free and the other attached? Are they

both partly attached? Neither. In this case, both earlobes are free. The 'free' allele is **dominant**. This means that it will show its effect, whether or not the allele for 'attached' is present. The allele for 'attached' is called **recessive**. The recessive allele will only show up in the appearance of the person if there is no dominant allele present. You will find out more about how genes work in Chapter 18.

LOOKING AHEAD – REGULATING GENES

You have seen how 'normal' gene coding for proteins (known as structural genes) make up only 1.5% of the genome. Some of the rest of the genome is DNA that regulates the action of the structural genes, switching them on and off. One way this happens is in regions of the DNA called **operons**.

An operon is a group of structural genes headed by a non-coding length of DNA called an operator, along with another sequence of DNA called a promoter. The promoter starts **transcription** by binding to an enzyme called RNA polymerase. Close to the promoter is a regulatory gene, which codes for a protein called a repressor. The repressor can bind with the operator, preventing the promoter from binding with RNA polymerase, and stopping transcription (Figure 16.8).



▲ Figure 16.8 An operon is a group of structural genes linked to an operator and a promoter. It is under the control of a regulatory gene.

The structural genes are in groups because they are related – e.g. they code for different enzymes in a metabolic pathway. Operons were first discovered in bacteria. At first we thought they only existed in prokaryotes, but molecular biologists have now found them in eukaryotic cells too.

CHAPTER QUESTIONS

SKILLS CRITICAL THINKING



More questions on DNA can be found at the end of Unit 5 on page 234.

- How many chromosomes are there in the body cells of a man?
 A 23 pairs + XX B 23 pairs + XY
 C 22 pairs + XX D 22 pairs + XY
- a What is:
 i a gene
 ii an allele?
 b Describe the structure of a chromosome.
 c How are the chromosomes in a woman's skin cells:
 i similar to
 ii different from those in a man's skin cells?



17 CELL DIVISION

Growth and reproduction are two characteristics of living things. Both involve cell division, which is the subject of this chapter.

LEARNING OBJECTIVES

- Understand how division of a diploid cell by mitosis produces two cells that contain identical sets of chromosomes
- Understand that mitosis occurs during growth, repair, cloning and asexual reproduction
- Understand how division of a cell by meiosis produces four cells, each with half the number of chromosomes, and that this results in the formation of genetically different haploid gametes
- Understand how random fertilisation produces genetic variation of offspring
- Understand that variation within a species can be genetic, environmental or a combination of both

In most parts of the body, cells need to divide so that organisms can grow and replace worn out or damaged cells. The cells that are produced in this type of cell division should be exactly the same as the cells they are replacing. This is the most common form of cell division.

Only in the sex organs is cell division different. Here, some cells divide to produce gametes (sex cells), which contain only half the original number of chromosomes. This is so that when male and female gametes fuse together (fertilisation) the resulting cell (called a **zygote**) will contain the full set of chromosomes and can then divide and grow into a new individual.

Human body cells are **diploid** – they have 46 chromosomes in 23 homologous pairs. The gametes, with 23 chromosomes (one copy of each homologous chromosome), are **haploid** cells.

There are two kinds of cell division: **mitosis** and **meiosis**. When cells divide by mitosis, two cells are formed. These have the same number and type of chromosomes as the original cell. Mitosis forms all the cells in our bodies except the gametes.

When cells divide by meiosis, four cells are formed. These have only half the number of chromosomes of the original cell. Meiosis forms gametes.

KEY POINT

Meiosis is sometimes called a *reduction division*. This is because it produces cells with only half the number of chromosomes of the original cell.

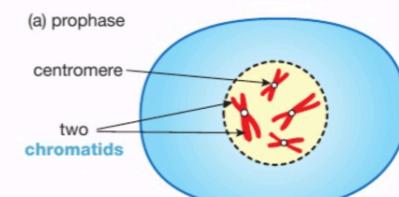
MITOSIS

When a 'parent' cell divides it produces 'daughter' cells. Mitosis produces two daughter cells that are genetically identical to the parent cell – both daughter cells have the same number and type of chromosomes as the parent cell.

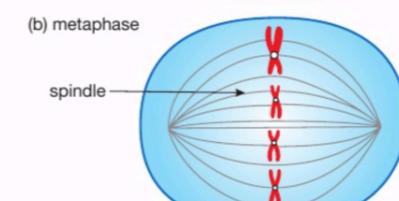
To achieve this, the dividing cell must do two things.

- It must copy each chromosome before it divides. This involves the DNA replicating and more proteins being added to the structure. Each daughter cell will then be able to receive a copy of each chromosome (and each molecule of DNA) when the cell divides.

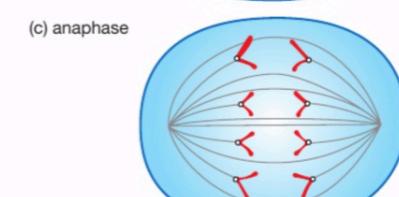
- It must divide in such a way that each daughter cell receives one copy of every chromosome. If it does not do this, both daughter cells will not contain all the genes.



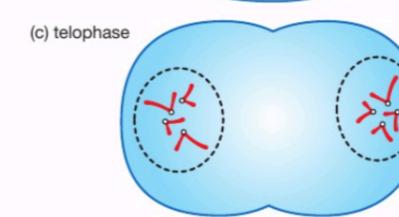
Before mitosis the DNA replicates and the chromosomes form two exact copies called chromatids. During the first stage of mitosis (prophase) the chromatids become visible, joined at a centromere. The nuclear membrane breaks down.



During metaphase a structure called the spindle forms. The chromosomes line up at the 'equator' of the spindle, attached to it by their centromeres.



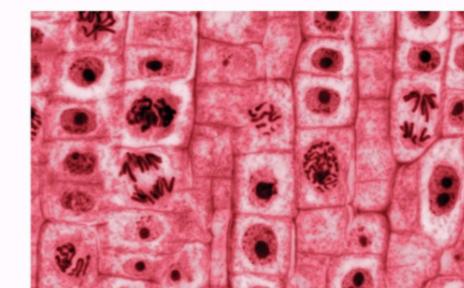
During anaphase, the spindle fibres shorten and pull the chromatids to the opposite ends ('poles') of the cell. The chromatids separate to become the chromosomes of the two daughter cells.



In the last stage (telophase) two new nuclei form at the poles of the cell. The cytoplasm starts to divide to produce two daughter cells. Both daughter cells have a copy of each chromosome from the parent cell.

▲ Figure 17.1 The stages of mitosis. For simplicity the cell shown contains two homologous pairs of chromosomes (one long pair, one short). (You do not need to remember the names of the stages.)

A number of distinct stages occur when a cell divides by mitosis. These are shown in Figure 17.1. Figure 17.2 is a photograph of some cells from the root tip of an onion. Cells in this region of the root divide by mitosis to allow growth of the root.



▲ Figure 17.2 Cells in the root tip of an onion dividing by mitosis. Can you identify any of the stages shown in Figure 17.1?

Each daughter cell formed by mitosis receives a copy of every chromosome, and therefore every gene, from the parent cell. Each daughter cell is genetically identical to the others. All the cells in our body (except the gametes) are formed by mitosis from the zygote (single cell formed at fertilisation). They all, therefore, contain copies of all the chromosomes and genes of that zygote. They are all genetically identical.

Whenever cells need to be replaced in our bodies, cells divide by mitosis to make them. This happens more frequently in some regions than in others.

- The skin loses thousands of cells every time we touch something. This adds up to millions every day that need replacing. A layer of cells beneath the surface is constantly dividing to produce replacements.
- Cells are scraped off the lining of the gut as food passes along. Again, a layer of cells beneath the gut lining is constantly dividing to produce replacement cells.
- Cells in our spleen destroy worn out red blood cells at the rate of 100 000 000 000 per day! These are replaced by cells in the bone marrow dividing by mitosis. In addition, the bone marrow forms all our new white blood cells and **platelets**.
- Cancer cells also divide by mitosis. The cells formed are exact copies of the parent cell, including the mutation in the genes that makes the cells divide uncontrollably.

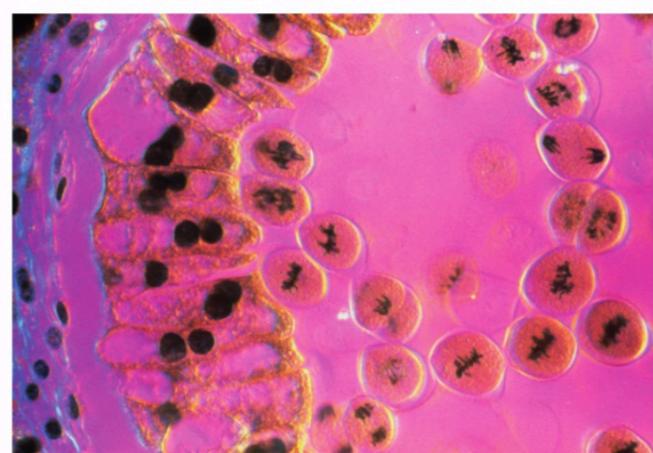
MEIOSIS

Meiosis forms gametes. It is a more complex process than mitosis and takes place in two stages called meiosis I and meiosis II, resulting in four haploid cells. Each daughter cell is genetically different from the other three and from the parent cell.

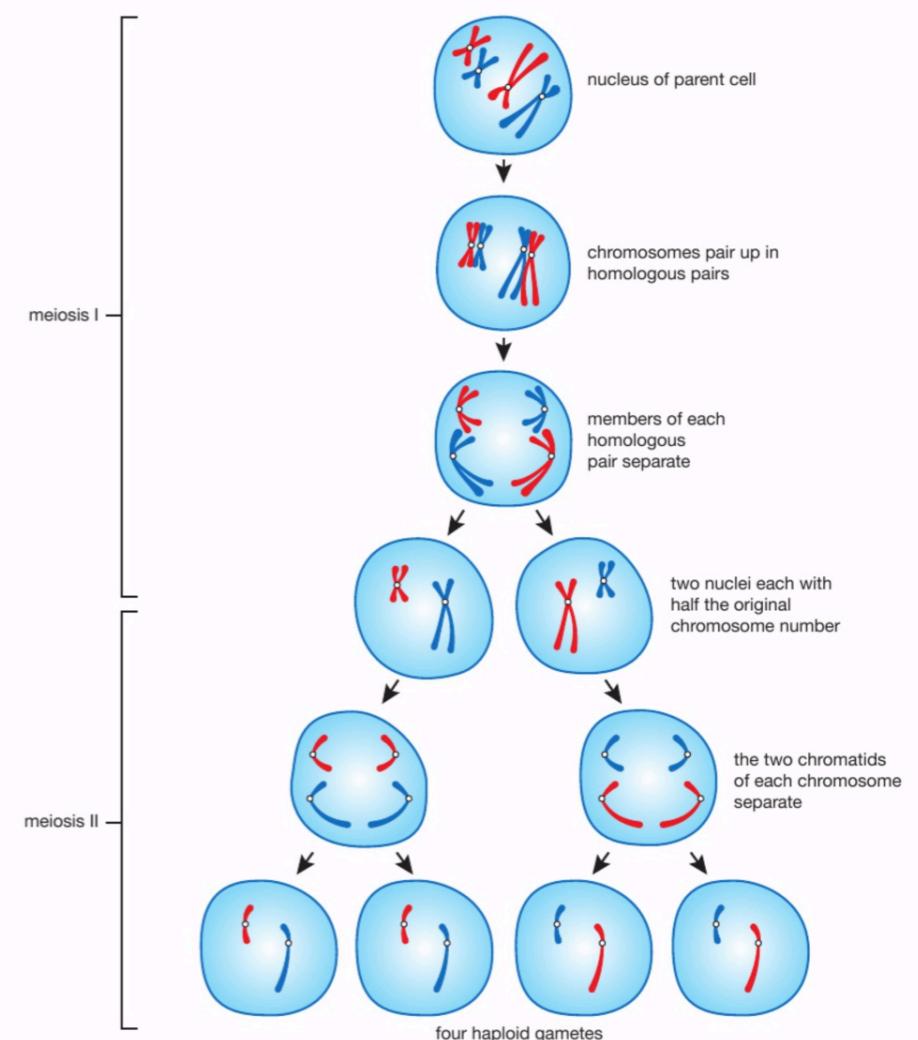
During meiosis the parent cell must do two things:

- It must copy each chromosome so that there is enough genetic material to be shared between the four daughter cells
- It must divide twice, in such a way that each daughter cell receives just one chromosome from each homologous pair.

These processes are summarised in Figure 17.4. Figure 17.3 shows cells in the anther of a flower dividing by meiosis.



▲ Figure 17.3 Photomicrograph of an anther showing cells dividing by meiosis.

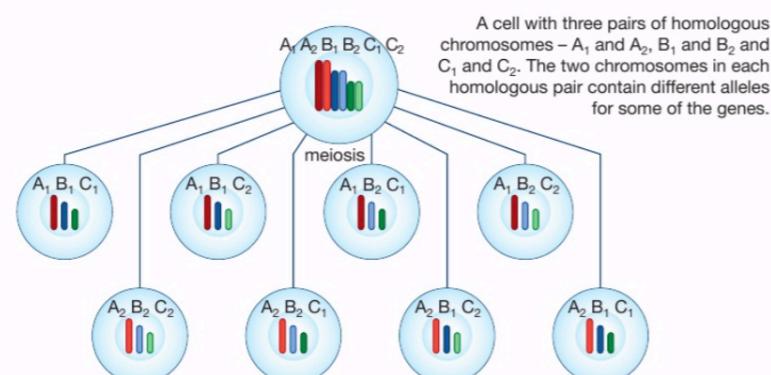


▲ Figure 17.4: The stages of meiosis. For simplicity the parent cell contains only two homologous pairs of chromosomes (one long pair, one short). To help you to see what happens, one member of each pair is coloured red and one blue. The cell membrane is shown, but the nuclear membrane has been omitted. A spindle forms during each division, but these have also been omitted for clarity.

There are two main events during meiosis:

- during the first division, one chromosome from each homologous pair goes into each daughter cell
- during the second division, the chromosome separates into two parts. One part goes into each daughter cell.

The gametes formed by meiosis don't all have the same combinations of alleles – there is *genetic variation* in the cells. During the two cell divisions of meiosis, the chromosomes of each homologous pair are shared between the two daughter cells independently of each of the other homologous pairs. This allows for much possible genetic variation in the daughter cells (Figure 17.5).



EXTENSION WORK

There is a mathematical rule for predicting how many combinations of chromosomes there can be. The rule is: number of possible combinations = 2ⁿ where n = number of pairs of chromosomes.

With two pairs of chromosomes, the number of possible combinations = 2² = 4. With three pairs of chromosomes, the number of possible combinations = 2³ = 8. With the 23 pairs of chromosomes in human cells, the number of possible combinations = 2²³ = 8 388 608!

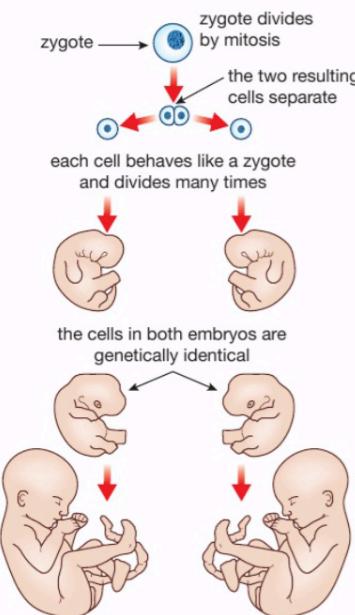
As a result of the two divisions of meiosis, each sex cell formed contains one chromosome from each homologous pair. This gives eight combinations. As A₁ and A₂ contain different alleles (as do B₁ and B₂, and C₁ and C₂) the eight possible sex cells will be genetically different.

▲ Figure 17.5 How meiosis produces variation

Table 17.1 summarises the similarities and differences between mitosis and meiosis.

Table 17.1 Comparison of meiosis and mitosis.

Feature of the process	Mitosis	Meiosis
Chromosomes are copied before division begins	Yes	Yes
Number of cell divisions	One	Two
Number of daughter cells produced	Two	Four
Daughter cells are haploid or diploid	Diploid	Haploid
Genetic variation in the daughter cells	No	Yes



▲ Figure 17.6 How identical twins are formed

SEXUAL REPRODUCTION AND VARIATION

Sexual reproduction in any multicellular organism involves the fusion of two gametes to form a zygote. The offspring from sexual reproduction vary genetically for a number of reasons. One reason is because of the huge variation in the gametes. Another reason is because of the random way in which fertilisation takes place. In humans, any one of the billions of sperm formed by a male during his life could, potentially, fertilise any one of the thousands of ova formed by a female.

This variation applies to both male and female gametes. So, just using our 'low' estimate of about 8.5 million different types of human gametes means that there can be 8.5 million different types of sperm and 8.5 million different types of ova. When fertilisation takes place, any sperm could fertilise any ovum. The number of possible combinations of chromosomes (and genes) in the zygote is 8.5 million × 8.5 million = 7.2 × 10¹³, or 72 trillion! And remember, this is using our 'low' number!

This means that every individual is likely to be genetically unique. The only exceptions are identical twins. Identical twins are formed from the same zygote – they are sometimes called monozygotic twins. When the zygote divides by mitosis, the two *genetically identical* cells formed do not 'stay together'. Instead, they separate and each cell behaves as though it were an individual zygote, dividing and developing into an embryo (Figure 17.6). Because they have developed from genetically identical cells (and, originally, from the same

zygote), the embryos (and, later, the children and the adults they become) will be genetically identical.

Non-identical twins develop from different zygotes and so are not genetically identical.

Seeds are made by sexual reproduction in plants. Each seed contains an embryo, which results from a pollen grain nucleus fusing with an egg cell nucleus. Embryos from the same plant will vary genetically because they are formed by different pollen grains fertilising different egg cells and so contain different combinations of genes.

ASEXUAL REPRODUCTION AND CLONING

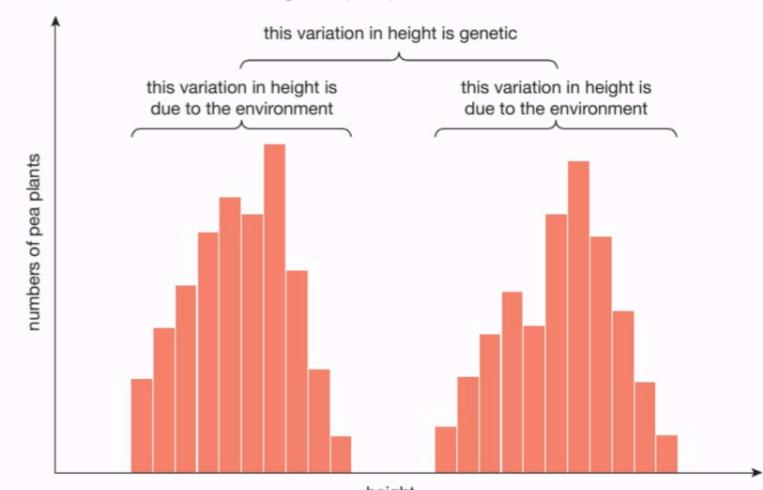
Plant breeders have known for a long time that sexual reproduction produces variation. They realised that if a plant had some desirable feature, the best way to get more of that plant was not to collect and plant its seeds, but to **clone** it in some way. Modern plant-breeding techniques allow the production of many thousands of identical plants from just a few cells of the original (see Chapter 19).

When organisms reproduce asexually, there is no fusion of gametes. A part of the organism grows and somehow breaks away from the parent organism. The cells it contains were formed by mitosis, so contain exactly the same genes as the parent. Asexual reproduction produces offspring that are genetically identical to the parent, and genetically identical to each other.

Asexual reproduction is common in plants (see Chapter 13). For example, flower bulbs grow and divide asexually each season to produce more bulbs. Asexual reproduction also occurs in some animals (see Chapter 9).

GENES AND ENVIRONMENT BOTH PRODUCE VARIATION

There are two varieties of pea plants that are either tall or short. This difference in height is due to the genes they inherit. There are no 'intermediate height' pea plants. However, all the tall pea plants are not *exactly* the same height and neither are all the short pea plants *exactly* the same height. Figure 17.7 illustrates the variation in height of pea plants.



▲ Figure 17.7 Bar chart showing variation in height of pea plants.

SKILLS CRITICAL THINKING, REASONING



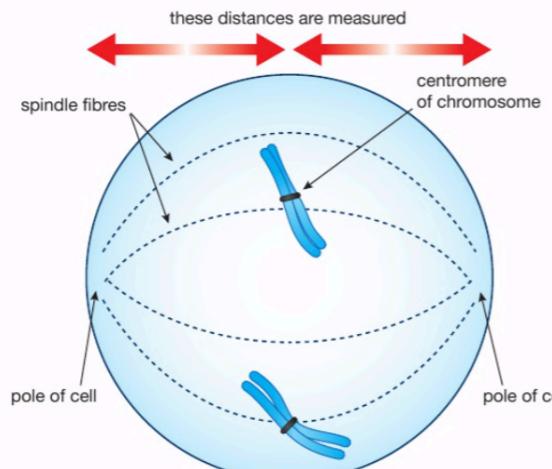
d Some pea plants are tall; others are dwarf. However, the tall plants are not exactly the same height and neither are all the dwarf plants the same height.

e People in some families are more at risk of heart disease than people in other families. However, not every member of the 'high risk' families have a heart attack and some members of the 'low risk' families do.

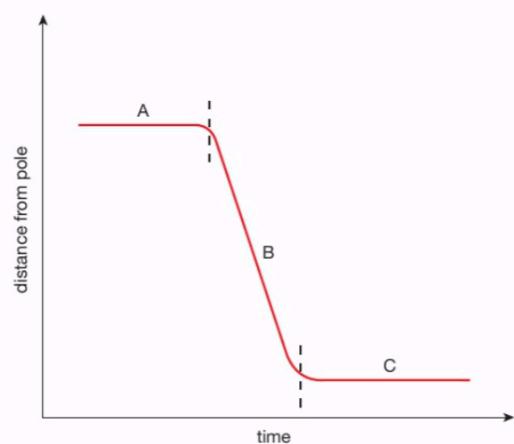
SKILLS ANALYSIS, INTERPRETATION



10 In an investigation into mitosis, the distance between a chromosome and the pole (end) of a cell was measured.



The graph shows how these distances changed during mitosis.



a Describe two events that occur during stage A.
 b Explain what is happening during stage B.
 c Describe two events that occur during stage C.

18 GENES AND INHERITANCE

How and why do we inherit features from our parents? This chapter answers these questions by looking at the work of Gregor Mendel and how he has helped us to understand the mysteries of inheritance.

LEARNING OBJECTIVES

- Understand that genes exist in alternative forms called alleles which give rise to differences in inherited characteristics
- Understand the meaning of the terms dominant, recessive, homozygous, heterozygous, phenotype and genotype
- Describe patterns of monohybrid inheritance using genetic diagrams
- Predict probabilities of outcomes from monohybrid crosses
- Understand how to interpret family pedigrees
- Understand how the sex of a person is controlled by one pair of chromosomes, XX in a female and XY in a male
- Describe the determination of the sex of offspring at fertilisation, using a genetic diagram
- Understand that most phenotypic features are the result of polygenic inheritance rather than single genes

The groundbreaking research that uncovered the rules of how genes are inherited was carried out by Gregor Mendel and published in 1865. The rules of inheritance are now known as 'Mendelian genetics' in his honour.

GREGOR MENDEL

Gregor Mendel was a monk who lived in a monastery in Brno in what is now the Czech Republic (Figure 18.1). He became interested in the science of heredity, and carried out hundreds of breeding experiments using pea plants. From his research Mendel was able to explain the laws governing inheritance.

Mendel found that for every feature or 'character' he investigated:

- a 'heritable unit' (what we now call a **gene**) is passed from one generation to the next
- the heritable unit (gene) can have alternative forms (we now call these different forms **alleles**)
- each individual must have two alternative forms (alleles) per feature
- the gametes only have one of the alternative forms (allele) per feature
- one allele can be dominant over the other.

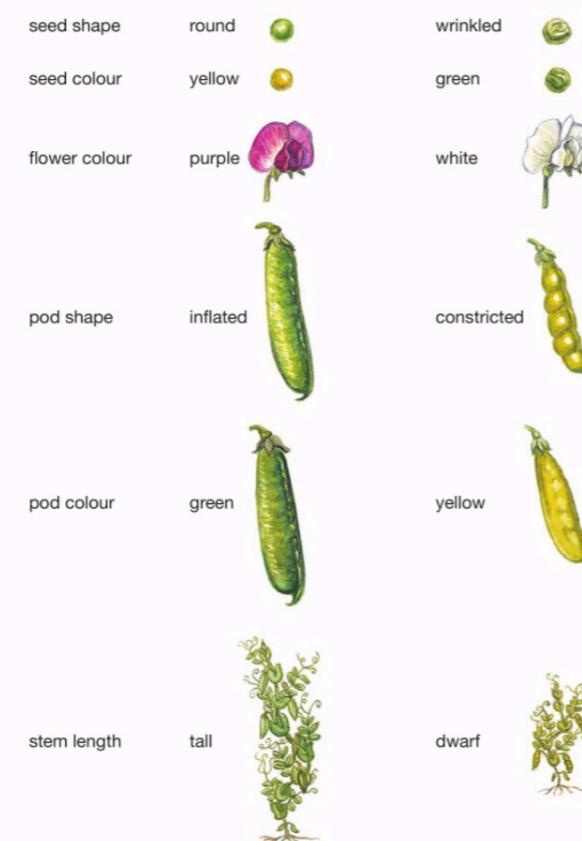


▲ Figure 18.1 Gregor Mendel.

Mendel used these ideas to predict outcomes of cross-breeding or 'crosses' between plants, which he tested in his breeding experiments. He published his results and ideas in 1865 but few people took any notice, and his work went unrecognised for many years. It wasn't until 1900 that other biologists working on inheritance rediscovered Mendel's work and realised its importance. In 1903, the connection between the behaviour of genes in Mendelian genetics and the behaviour of chromosomes in meiosis was noticed and the science of genetics was established.

MENDEL'S EXPERIMENTS ON INHERITANCE

Mendel noticed that many of the features of pea plants had two alternative forms. For example, plants were either tall or very short (called a 'dwarf' variety); they either had purple or white flowers; they produced yellow seeds or green seeds. There were no intermediate forms, no pale purple flowers or green/yellow seeds or intermediate height plants. Figure 18.2 shows some of the contrasting features of pea plants that Mendel used in his breeding experiments.



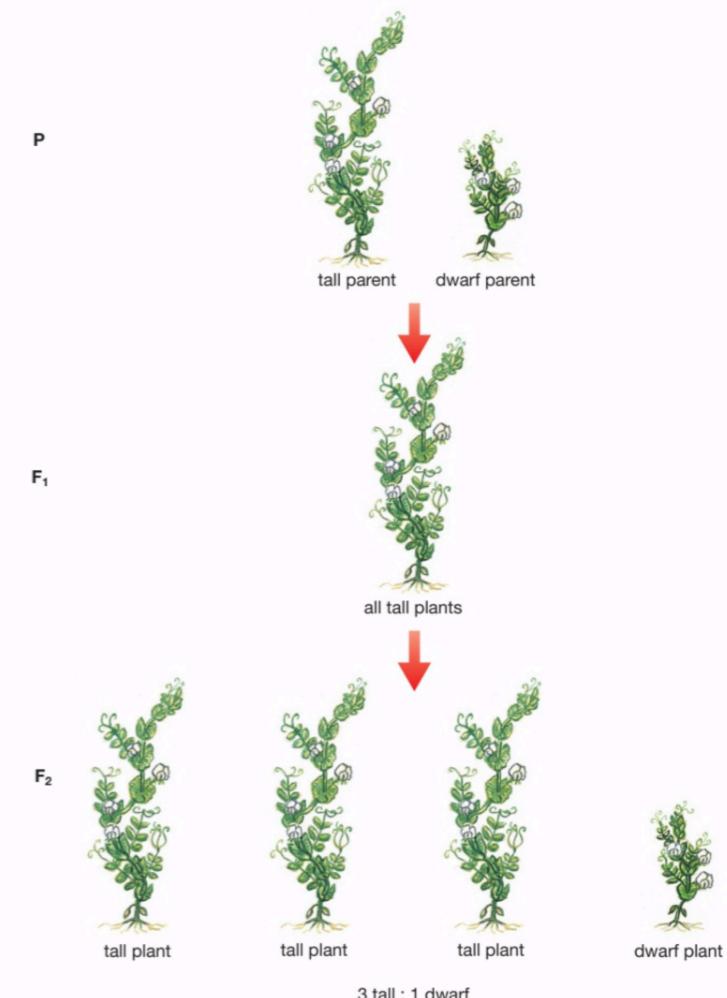
KEY POINT

In his breeding experiments, Mendel initially used only plants that had 'bred true' for several generations. For example, any tall pea plants he used came from generations of pea plants that had all been tall.

▲ Figure 18.2 Some features of pea plants used by Mendel in his breeding experiments.

Mendel decided to investigate, systematically, the results of cross breeding plants that had contrasting features. These were the 'parent plants', referred to as 'P' in genetic diagrams. He transferred pollen from one experimental plant to another. He also made sure that the plants could not be self-fertilised.

He collected all the seeds formed, grew them and noted the features that each plant developed. These plants were the first generation of offspring, called the **F₁ generation**. He did not cross-pollinate these plants, but allowed them to self-fertilise. Again, he collected the seeds, grew them and noted the features that each plant developed. These plants formed the second generation of offspring or **F₂ generation**. When Mendel used pure-breeding tall and pure-breeding dwarf plants as his parents, he obtained the results shown in Figure 18.3.



▲ Figure 18.3 A summary of Mendel's results from breeding tall pea plants with dwarf pea plants.

Mendel obtained similar results when he carried out breeding experiments using plants with other pairs of contrasting characters (Figure 18.4). He noticed two things in particular.

- All the plants of the F₁ generation were of one type. This type was not a blend of the two parental features, but one or the other. For example, when tall and dwarf parents were crossed, all the F₁ plants were tall.
- There was always a 3:1 ratio of types in the F₂ generation. Three-quarters of the plants in the F₂ generation were of the type that appeared in the F₁ generation. One-quarter showed the other parental feature. For example, when tall F₁ plants were crossed, three-quarters of the F₂ plants were tall and one-quarter were dwarf.

Mendel was able to use his findings to work out how features were inherited, despite having no knowledge of chromosomes, genes or meiosis. Nowadays we can use our understanding of these ideas to explain Mendel's results.

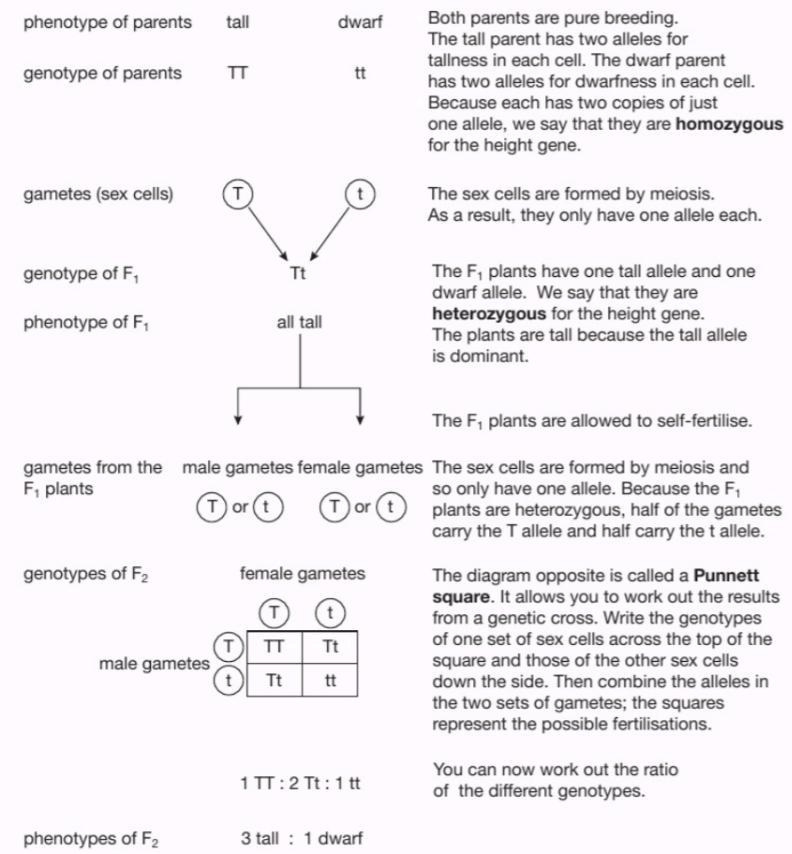
EXPLAINING MENDEL'S RESULTS

- Each feature is controlled by a gene, which is found on a chromosome.
- There are two copies of each chromosome and each gene in all body cells, except the gametes.
- The gametes have only one copy of each chromosome and each gene (i.e. one allele).
- There are two alleles of each gene.
- One allele is **dominant** over the other allele, which is **recessive**.
- When two different alleles (one dominant and one recessive) are in the same cell, only the dominant allele is expressed (shown in the appearance of the organism).
- An individual can have two dominant alleles, two recessive alleles or a dominant allele and a recessive allele in each cell.

KEY POINT

Normally, we use the first letter of the dominant feature to represent the gene, with a capital letter indicating the dominant allele and a lower case letter the recessive allele. Tall is dominant to dwarf in pea plants, so we use T for the allele for tall and t for dwarf.

We can use the cross between tall and dwarf pea plants as an example (Figure 18.4). In pea plants, there are tall and dwarf alleles of the gene for height. We will use the symbol T for the tall allele and t for the dwarf allele. The term **genotype** describes the alleles each cell has for a certain feature (e.g. TT). The **phenotype** is the feature that results from the genotype (e.g. a tall plant).



▲ Figure 18.4 Results of crosses using true-breeding tall and dwarf pea plants.

KEY POINT

Mendel's experiments described above, all involve single genes (e.g. the gene for height, or the gene for flower colour). The name given for inheritance involving one gene is **monohybrid** inheritance. It is possible to draw genetic diagrams involving two or more genes (e.g. height and flower colour together), but for International GCSE you only need to interpret monohybrid crosses.

It is important to remember that in genetic crosses, ratios such as 3:1 are *predicted* ratios. In breeding experiments the *actual* numbers of offspring are unlikely to exactly fit a 3:1 ratio.

Imagine you flip a coin 20 times. The most likely outcome is that you will get 10 heads and 10 tails. However, you wouldn't be surprised to get, by chance, 11 heads and 9 tails, or 8 heads and 12 tails. The same principle applies to the outcome of a breeding experiment.

For example, one of Mendel's experiments produced 787 tall plants and 277 dwarf plants. This is a ratio of 2.84:1, not quite the expected 3:1. The reason for this is that there are a number of factors that affect survival of the plants – some pollen may not fertilise some ova, some seedlings may die before they mature, and so on. These are unpredictable or 'chance' events. The numbers that Mendel found were statistically close enough to the expected 3:1 ratio, and he found the same thing when he repeated his experiments with other characteristics.

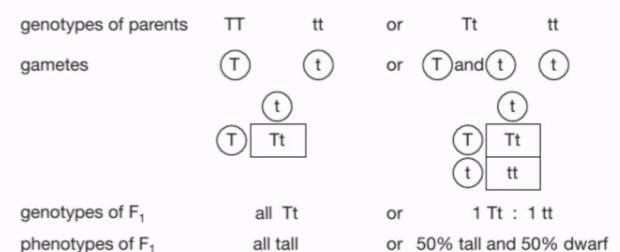
WORKING OUT GENOTYPES – THE TEST CROSS

You cannot tell just by looking at a tall pea plant whether it is **homozygous** (TT) or **heterozygous** (Tt). Both these genotypes would appear equally tall because the tall allele is dominant.

It would help if you knew the genotypes of its parents. You could then write out a genetic cross and perhaps work out the genotype of your tall plant. If you don't know the genotypes of the parents, the only way you can find out is by carrying out a breeding experiment called a **test cross**.

In a test cross, the factor under investigation is the unknown genotype of an organism showing the dominant phenotype. A tall pea plant could have the genotype TT or Tt. You must control every other possible variable *including the genotype of the plant you breed it with*. The only genotype you can be *certain* of is the genotype of plants showing the recessive phenotype (in this case dwarf plants). They *must* have the genotype tt.

In this example, you must breed the 'unknown' tall pea plant (TT or Tt) with a dwarf pea plant (tt). You can write out a genetic cross for both possibilities (TT ' tt and Tt ' tt) and *predict* the outcome for each (Figure 18.5). You can then compare the result of the breeding experiment with the predicted outcome, to see which result matches the prediction most closely.



▲ Figure 18.5 A test cross

From our crosses we would expect:

- all* the offspring to be tall if the tall parent was homozygous (TT)
- half* the offspring to be tall and *half* to be dwarf if the tall parent was heterozygous (Tt).

WAYS OF PRESENTING GENETIC INFORMATION

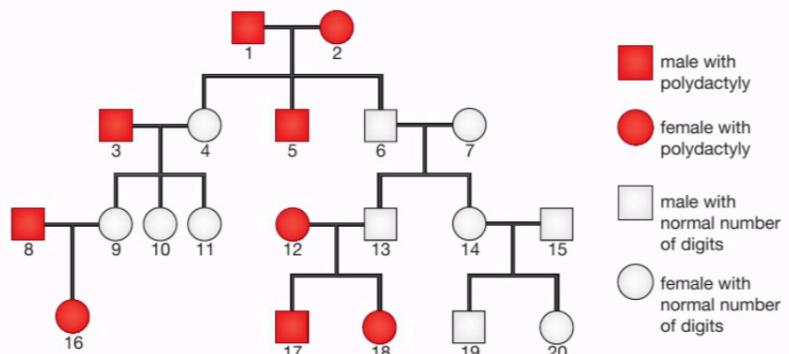
Writing out a genetic cross is a useful way of showing how genes are passed through one or two generations, starting from the parents. To show a family history of a genetic condition requires more than this. We can use a diagram called a **pedigree**.

Polydactyly is an inherited condition in which a person develops an extra digit (finger or toe) on the hands and feet. It is determined by a dominant allele. The recessive allele causes the normal number of digits to develop.

If we use the symbol D for the polydactyly allele and d for the normal-number allele, the possible genotypes and phenotypes are:

- DD – person has polydactyly (has two dominant polydactyly alleles)
- Dd – person has polydactyly (has a dominant polydactyly allele and a recessive normal allele)
- dd – person has the normal number of digits (has two recessive, normal-number alleles).

We don't use P and p to represent the alleles as you would expect, because the letters P and p look very similar and could easily be confused. The pedigree for polydactyly is shown in Figure 18.6.



▲ Figure 18.6 A pedigree showing the inheritance of polydactyly in a family.

We can extract a lot of information from a pedigree. In this case:

- there are four generations shown (individuals are arranged in four horizontal lines)
- individuals 4, 5 and 6 are children of individuals 1 and 2 (a family line connects each one directly to 1 and 2)
- individual 4 is the first-born child of 1 and 2 (the first-born child is shown to the left, then second born to the right of this, then the third born and so on)
- individuals 3 and 7 are not children of 1 and 2 (no family line connects them directly to 1 and 2)
- individuals 3 and 4 are father and mother of the same children – as are 1 and 2, 6 and 7, 8 and 9, 12 and 13, 14 and 15 (a horizontal line joins them).

It is usually possible to work out which allele is dominant from a pedigree. You look for a situation where two parents show the same feature and at least one child shows the contrasting feature. In Figure 18.6, individuals 1 and 2 both have polydactyly, but children 4 and 6 do not. There is only one way to explain this:

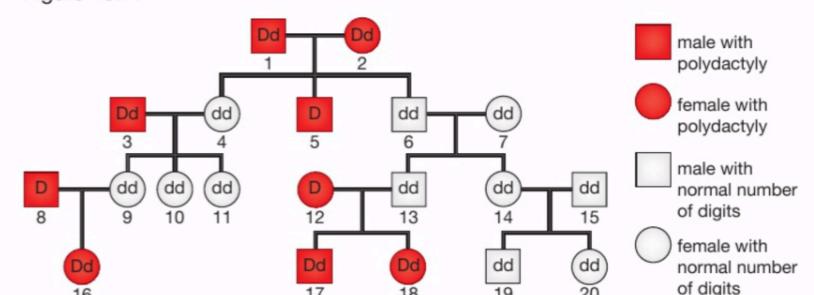
- the normal alleles in 4 and 6 can only have come from their parents (1 and 2), so 1 and 2 must both carry normal alleles
- 1 and 2 show polydactyly, so they must have polydactyly alleles as well
- if they have both polydactyly alleles *and* normal alleles but show polydactyly, the polydactyly allele must be the dominant allele.

Now that we know which allele is dominant, we can work out most of the genotypes in the pedigree. All the people with the normal number of digits *must* have the genotype dd (if they had even one D allele, they would show polydactyly). All the people with polydactyly must have at least one polydactyly allele (they must be either DD or Dd).

From here, we can begin to work out the genotypes of the people with polydactyly. To do this we need to remember that people with the normal number of digits must inherit one 'normal-number' allele from each parent, and also that people with the normal number of digits will pass on one 'normal-number' allele to each of their children.

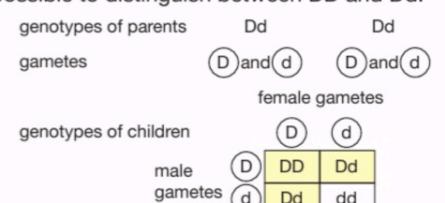
From this we can say that any person with polydactyly who has children with the normal number of digits must be heterozygous (the child must have inherited one of their two 'normal-number' alleles from this parent), and also that any person with polydactyly who has one parent with the normal number of digits must also be heterozygous (the 'normal-number' parent can only have passed on a 'normal-number' allele). Individuals 1, 2, 3, 16, 17 and 18 fall into one or other of these categories and must be heterozygous.

We can now add this genetic information to the pedigree. This is shown in Figure 18.7.



▲ Figure 18.7 A pedigree showing the inheritance of polydactyly in a family, with details of genotypes added.

We are still uncertain about individuals 5, 8 and 12. They could be homozygous or heterozygous. For example, individuals 1 and 2 are both heterozygous. Figure 18.8 shows the possible outcomes from a genetic cross between them. Individual 5 could be any of the outcomes indicated by the shading. It is impossible to distinguish between DD and Dd.

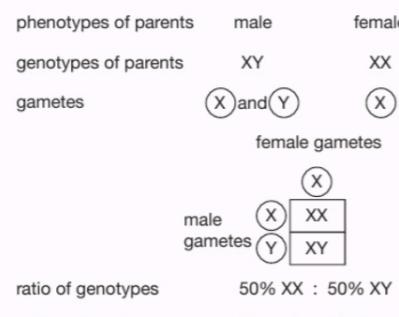


▲ Figure 18.8 Possible outcomes from a genetic cross between two parents, both heterozygous for polydactyly.

SEX DETERMINATION

Our sex – whether we are male or female – is not under the control of a single gene. It is determined by the X and Y chromosomes – the **sex chromosomes**. As well as the 44 non-sex chromosomes, there are two X chromosomes in all cells of females (except the egg cells) and one X and one Y chromosome in all cells of males (except the sperm). Our sex is effectively determined by the presence or absence of the Y chromosome. The full chromosome complement of male and female is shown in Figure 16.4 on page 198.

DID YOU KNOW?
Because the Y chromosome, when present, causes a zygote to develop into a male, some people describe it as 'dominant'. This is incorrect: dominant and recessive are terms that are only applied to individual alleles.



▲ Figure 18.9 Determination of sex in humans

POLYGENIC INHERITANCE

All of the genetic crosses that you have seen in this chapter have been examples of inheritance involving single genes. The reason for this is that it is easier to draw genetics diagrams and explain what is happening if we start by considering alleles of a single gene. However, many characteristics are controlled by two or more genes working together. This is called **polygenic inheritance**.

DID YOU KNOW?
Melanin protects the skin against the harmful effects of ultraviolet radiation, which is a mutagen that can cause skin cancer.



◀ Figure 18.10 Skin colour depends on the amount of melanin in the skin. It is a result of polygenic inheritance.

Other human characteristics determined by several genes (**polygenes**) are human height and body mass (weight).

CHAPTER QUESTIONS

SKILLS CRITICAL THINKING



More questions on chromosomes, genes and inheritance can be found at the end of Unit 5 on page 234.

1 Which of the following is true of dominant alleles?

- A they are only expressed if present as a pair
- B they determine the most favourable of a pair of alternative features
- C they are inherited in preference to recessive alleles
- D a dominant allele is expressed if present with a recessive allele

2 In pea plants, the allele for purple petals is dominant to the allele for white petals. A plant heterozygous for petal colour was crossed with a plant with white petals. What would be the ratio of genotypes in the offspring?

A 1:1 B 2:1 C 1:0 D 3:1



3 The allele for yellow coat colour in mice (Y) is dominant to the allele for non-yellow coat colour (y).

Mice with the genotype yy have non-yellow coats.

Mice with the genotype Yy have yellow coats.

Mice with the genotype YY die as embryos.

Two heterozygous mice were crossed. What is the probability that a surviving mouse in the F1 generation will be yellow?

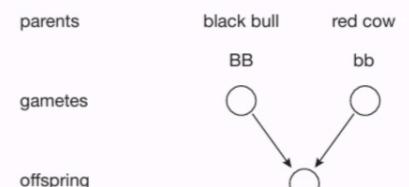
A 0.00 B 0.25 C 0.50 D 0.67

4 Predict the ratios of offspring from the following crosses between tall/dwarf pea plants.

- a TT x TT
- b TT x Tt
- c TT x tt
- d Tt x Tt
- e Tt x tt
- f tt x tt.



5 In cattle, a pair of alleles controls coat colour. The allele for black coat colour is dominant over the allele for red coat colour. The genetic diagram represents a cross between a pure-breeding black bull and a pure-breeding red cow. B = dominant allele for black coat colour; b = recessive allele for red coat colour.



a i What term describes the genotypes of the pure-breeding parents?
ii Explain the terms dominant and recessive.



SKILLS CRITICAL THINKING



b **i** What are the genotypes of the gametes of each parent?
ii What is the genotype of the offspring?
c Cows with the same genotype as the offspring were bred with bulls with the same genotype.
i What genetic term describes this genotype?
ii Draw a genetic diagram to work out the ratios of:
 the genotypes of the offspring
 the phenotypes of the offspring.

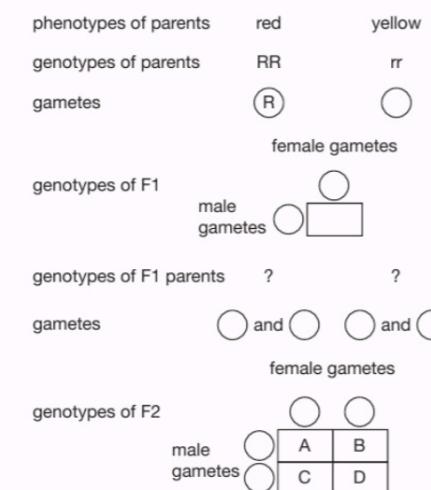
SKILLS INTERPRETATION, PROBLEM SOLVING



SKILLS INTERPRETATION

6 In nasturtiums, a single pair of alleles controls flower colour.

The allele for red flower colour is dominant over the allele for yellow flower colour. The diagram represents the results of a cross between a pure-breeding red-flowered nasturtium and a pure-breeding yellow-flowered nasturtium. R = dominant allele for red flower colour; r = recessive allele for yellow flower colour.



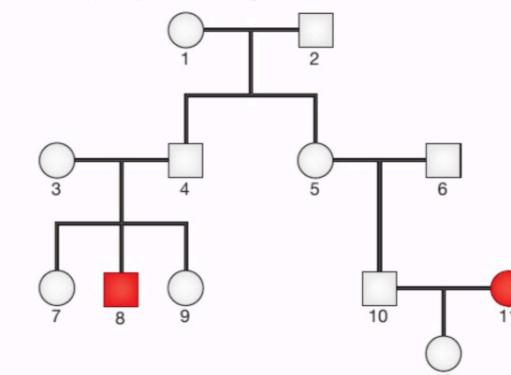
a Copy and complete the genetic diagram.
b What are the colours of the flowers of A, B, C and D?

SKILLS ANALYSIS

SKILLS ANALYSIS



7 Cystic fibrosis is an inherited condition. The diagram shows the incidence of cystic fibrosis in a family over four generations.



Legend:
 ■ affected male
 □ unaffected male
 ● affected female
 ○ unaffected female

SKILLS INTERPRETATION

a What evidence in the pedigree suggests that cystic fibrosis is determined by a recessive allele?
b What are the genotypes of individuals 3, 4 and 11? Explain your answers.
c Draw genetic diagrams to work out the probability that the next child born to individuals 10 and 11 will
i be male,
ii suffer from cystic fibrosis.

SKILLS REASONING

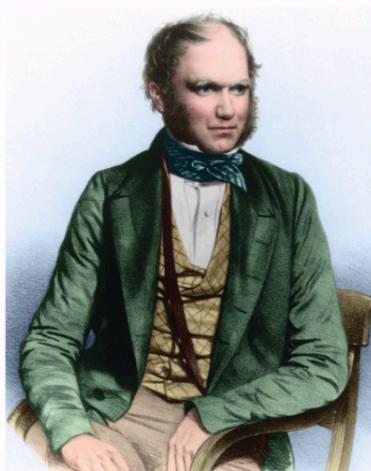
8 In guinea pigs, the allele for short hair is dominant to that for long hair.
a Two short-haired guinea pigs were bred and their offspring included some long-haired guinea pigs. Explain these results.
b How could you find out if a short-haired guinea pig was homozygous or heterozygous for hair length?

19 NATURAL SELECTION, EVOLUTION AND SELECTIVE BREEDING

Over millions of years, life on this planet has evolved from its simple beginnings into the vast range of organisms present today. This has happened by a process called natural selection. This chapter looks at natural selection, as well as traditional methods of selective breeding and modern developments involving cloned organisms.

LEARNING OBJECTIVES

- Explain Darwin's theory of evolution by natural selection
- Understand how resistance to antibiotics can increase in bacterial populations
- Understand how selective breeding can be used to produce plants with desired characteristics
- Understand how selective breeding can be used to produce animals with desirable characteristics



▲ Figure 19.1 Charles Darwin (1809–1882).

The meaning of '**evolution**' is that species of animals and plants are not fixed in their form, but change over time. It is not a new idea. For thousands of years philosophers have discussed this theory. By the beginning of the nineteenth century there was overwhelming evidence for evolution, and many scientists had accepted that it had taken place. What was missing was an understanding of the *mechanism* by which evolution could have occurred.

The person who proposed the mechanism for evolution that is widely accepted today was the English biologist Charles Darwin (Figure 19.1). He called the mechanism **natural selection**.

THE WORK OF CHARLES DARWIN

Charles Darwin was the son of a country doctor. He did not do particularly well at school or university and was unable to decide on a profession. His father is supposed to have said: 'you're good for nothing but shooting guns and rat-catching ... you'll be a disgrace to yourself and all of your family'. He was wrong – Darwin went on to become one of the most famous scientists of all time!

At the age of 22, Charles Darwin became the unpaid biologist aboard the survey ship HMS *Beagle*, which left England for a five-year voyage in 1831 (Figure 19.2).



Figure 19.2 The five-year journey of HMS *Beagle* ►

DID YOU KNOW?

A fossil is the remains of an animal or plant that lived thousands or millions of years ago, preserved in sedimentary rocks. Fossils are formed when minerals replace the materials in bone and tissue, creating a replica of the original organism in the rock.

During the voyage, Darwin collected hundreds of specimens and made many observations about the variety of organisms and the ways in which they were adapted to their environments. He gained much information, in particular, from the variety of life forms in South America and the Galapagos Islands. Darwin was influenced by the work of Charles Lyell who was, at the time, laying the foundations of modern geology. Lyell was using the evidence of rock layers to suggest that the surface of the Earth was constantly changing. The layers of sediments in rocks represented different time periods. Darwin noticed that the fossils found in successive layers of rocks often changed slightly through the layers. He suggested that life forms were continually changing – evolving.

On his return to England, Darwin began to evaluate his data and wrote several essays, introducing the ideas of natural selection. He arrived at his theory of natural selection from observations made during his voyage on HMS *Beagle* and from deductions made from those observations. Darwin's observations were that:

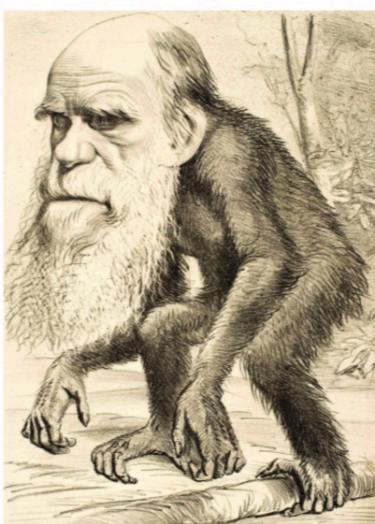
- organisms generally produce more offspring than are needed to replace them – a single female salmon can release 5 million eggs per year; a giant puffball fungus produces 40 million spores
- despite this over-reproduction, stable, established populations of organisms generally remain the same size – the seas are not overflowing with salmon, and we are not surrounded by lots of giant puffball fungi!
- members of the same species are not identical – they show variation.

He made two important deductions from these observations.

- From the first two observations he deduced that there is a 'struggle for existence'. Many offspring are produced, yet the population stays the same size. There must be competition for resources and many individuals must die.
- From the third observation he deduced that, if some offspring survive while others die, those organisms best suited to their environment would survive to reproduce. Those less suited will die. This gave rise to the phrase 'survival of the fittest'.

Notice a key phrase in the second deduction – the best-suited organisms *survive to reproduce*. This means that those characteristics that give the organism a better chance of surviving will be passed on to the next generation. Fewer of the individuals that are less suited to the environment survive to reproduce. The next generation will have more of the type that is better adapted and fewer of the less well adapted type. This will be repeated in each generation.

Another naturalist, Alfred Russell Wallace, had also studied life forms in South America and Indonesia and had reached the same conclusions as Darwin. Darwin and Wallace published a scientific paper on natural selection jointly, although it was Darwin who went on to develop the ideas further. In 1859, he published his now famous book *On the Origin of Species by Means of Natural Selection* (usually shortened to *The Origin of Species*).



▲ Figure 19.3 Darwin's ideas were unpopular and many newspapers of the time made fun of them.

This book changed forever the way in which biologists think about how species originate. Darwin went on to suggest that humans could have evolved from ape-like ancestors. For this he was ridiculed, largely by people who had misunderstood his ideas (Figure 19.3). He also carried out considerable research in other areas of biology, such as plant tropisms (see Chapter 12).

KEY POINT

When Darwin proposed his theory of natural selection, he did not know about genes and how they control characteristics. Gregor Mendel had yet to publish his work on inheritance, and as you have seen, the significance of Mendel's work was not recognised until 1903.

The theory of natural selection proposes that some factor in the environment 'selects' which forms of a species will survive to reproduce. Forms that are not well adapted will not survive.

The following is a summary of how we think natural selection works:

1. there is variation within the species
2. changing conditions in the environment (called a selection pressure) favours one particular form of the species (which has a selective advantage)
3. the frequency of the favoured form increases (it is selected for) under these conditions (survival of the fittest)
4. the frequency of the less well adapted form decreases under these conditions (it is selected against).

As you have seen, many gene mutations are harmful, and cells that carry them will not usually survive. Some mutations are 'neutral' and if they arise in the gametes, may be passed on without affecting the survival of the offspring. However, a few mutations can actually be beneficial to an organism. Beneficial mutations are the 'raw material' that are ultimately the source of new inherited variation.

SOME EXAMPLES OF HOW NATURAL SELECTION MIGHT HAVE WORKED

THE HOVERFLY



▲ Figure 19.4 Two insects showing 'warning colouration'. (a) A wasp, which has a sting. (b) A harmless hoverfly.

Figure 19.4 shows two species of insect: a wasp and a hoverfly. Wasps can defend themselves against predators using a sting. They also have a body with yellow and black stripes. This is called a 'warning colouration'. Predators such as birds soon learn that these colours mean that wasps have the sting, and they avoid attacking them.

Hoverflies do not have a sting. However, they have an appearance that is very like a wasp, with similar yellow and black stripes – they are 'mimics' of wasps. Predators treat hoverflies as if they do have a sting.

Clearly, mimicking a wasp is an advantage to the hoverfly. How could they have evolved this appearance? We can explain how it could have happened by natural selection.

The selection pressure was predation by birds and other animals. Among the ancestors of present-day hoverflies there would have been variation in colours. As a result of mutations, some hoverflies gained genes that produced stripes on their bodies. These insects were less likely to be eaten by predators than hoverflies without the stripes – they had a *selective advantage*.

Since the hoverflies with stripes were more likely to survive being eaten, they were more likely to reproduce, and would pass on the genes for stripes to their offspring. This process continued over many generations. Gradually more mutations and selection for 'better' stripes took place, until the hoverflies evolved the excellent warning colouration that they have today.

KEY POINT

Note that perfect stripes didn't have to appear straight away. Even a slight stripy appearance could give a small selective advantage over hoverflies without stripes. This would be enough to result in an increase in stripy hoverflies in the next generation.

THE POLAR BEAR



▲ Figure 19.5 A polar bear hunting on the Arctic sea ice.

The polar bear lives in the Arctic, inhabiting landmasses and sea ice covering the waters within the Arctic Circle (Figure 19.5). It is a large predatory carnivore, mainly hunting seals. One way the bear hunts is to wait near holes in the ice where seals come up to breathe. It also silently approaches and attacks seals that are resting on the ice.

Polar bears have many **adaptations** that suit them to their habitat. These include:

- a thick layer of white fur, which reduces heat loss and acts as camouflage in the snow
- wide, large paws. These help with walking in the snow, and are used for swimming
- strong, muscular legs – a bear can swim continuously in the cold Arctic waters for days
- nostrils that close when the bear is swimming under water
- a large body mass. Polar bears are the largest bears on Earth. An adult male averages 350 to 550 kilograms, and the record is over 1000 kilograms. This large size results in the animal having a small surface area to volume ratio, which reduces heat loss
- a 10 centimetre thick layer of insulating fat under the skin
- a well developed sense of smell – used to detect the bear's prey
- bumps on the pads of the paws to provide grip on the ice
- short, powerful claws, which also provide grip, and are needed for holding the heavy prey.

The polar bear is thought to have evolved from a smaller species, the brown bear, about 150,000 years ago. How did it evolve its adaptations for life in the Arctic? Let's consider just one of the adaptations, the thick white fur.

There are two main selection pressures in favour of thick white fur. The first is the need for insulation to reduce heat loss. The polar bear often has to survive temperatures of -30°C , and temperatures in the Arctic can fall as low as -70°C . The second is camouflage; white fur camouflages the animal against the snow so that it can approach its prey unseen and then attack it.

Among the brown bears that were the ancestors of the polar bear there would have been variations in fur length and colour. When some of these bears came to live in colder, more northerly habitats, those individuals with longer and paler fur would have had a selective advantage over others with shorter, darker fur. Any gene mutations that produced long, pale fur increased this advantage. Bears with these genes were less likely to die from the cold, or from lack of food. As a result, well-adapted bears were more likely to reproduce and pass on their genes. Over many thousands of years more mutations and selection for long, white fur produced the adaptation we see in the polar bear today. The same process of natural selection is thought to have happened to bring about the other adaptations shown by the polar bear.

CAN WE OBSERVE NATURAL SELECTION IN ACTION?

Most animals and plants reproduce slowly, so it takes a long time for natural selection to have an observable effect. To observe natural selection happening we can study organisms that reproduce quickly, such as bacteria or insects.

ANTIBIOTIC RESISTANCE IN BACTERIA



▲ Figure 19.6 This photo shows a colony of bacteria growing on a petri dish of nutrient agar. The circular discs contain different antibiotics. The discs have clear areas around them, where the bacteria have been killed by the antibiotic diffusing out from the discs.

Antibiotics are chemicals that kill or reduce the growth of microorganisms (Figure 19.6). They are used in medicine mainly to treat bacterial infections, although a few antibiotics are effective against fungal pathogens. Antibiotics do not work on viruses, so they are no use in treating any disease caused by a virus.

Natural antibiotics are produced by bacteria and fungi. They give a microorganism an advantage over other microorganisms when competing for nutrients and other resources, since the antibiotic kills the competing organisms.

Alexander Fleming discovered the first antibiotic in 1929. It is made by the mould *Penicillium*, and is called **penicillin**. Penicillin kills bacteria, and was first used to treat bacterial infections in the 1940s. Since then other natural antibiotics have been discovered, and many more have been chemically synthesised in laboratories. The use of antibiotics has increased dramatically, particularly over the last 20 years. We now almost expect to be given an antibiotic for even the most minor of ailments. This can be dangerous, as it leads to the development of bacterial resistance to antibiotics, so that the antibiotics are no longer effective in preventing bacterial infection.

DID YOU KNOW?

A particularly worrying example of a resistant bacterium is **MRSA**. MRSA stands for methicillin-resistant *Staphylococcus aureus*. It is sometimes called a 'super bug' because it is resistant to many antibiotics including methicillin (a type of penicillin). It is a particular problem in hospitals where it is responsible for many difficult-to-treat infections.

Resistance starts when a random mutation gives a bacterium resistance to a particular antibiotic. In a situation where the antibiotic is widely used, the new resistant bacterium has an advantage over non-resistant bacteria of the same type. The resistant strain of bacterium will survive and multiply in greater numbers than the non-resistant type. Bacteria reproduce very quickly – the generation time of a bacterium (the time it takes to divide into two daughter cells) can be as short as 20 minutes. This means that there could be 72 generations in a single day, producing a population of millions of resistant bacteria.

Resistant bacteria will not be killed by the antibiotic, meaning the antibiotic is no longer effective in controlling the disease.

Bacterial resistance to antibiotics was first noticed in hospitals in the 1950s, and has grown to be a major problem today. The resistant bacteria have a selective advantage over non-resistant bacteria – they are 'fitter'. In effect, the bacteria have evolved as a result of natural selection.

Doctors are now more reluctant to prescribe antibiotics. They know that by using them less, the bacteria with resistance have less of an advantage and will not become as widespread.

HINT

Some people talk about bacteria becoming *immune* to antibiotics. This is a misunderstanding. Immunity happens in people – we become immune to microorganisms that infect us, as a result of the immune response.

Bacteria become *resistant* to antibiotics.

PESTICIDE RESISTANCE IN INSECTS

Just as pathogenic bacteria can become resistant to antibiotics, insect pests can develop resistance to insecticides. The powerful insecticide DDT was first used in the 1940s (see Chapter 15). By the 1950s many species of insect (e.g. mosquitoes) appeared to be resistant to DDT. The resistant insects had developed a gene mutation that stopped them being killed by the insecticide.

While DDT continued to be used, the resistant insects had a selective advantage over the non-resistant ones. They survived to breed, so that with each generation the numbers of resistant insects in the population increased.

The same thing has happened with modern insecticides. There are now hundreds of examples of insect pests that have developed resistance to different insecticides.

SELECTIVE BREEDING

About 12 000 years ago, the human way of life changed significantly. Humans began to grow plants and keep animals for milk and meat. They became farmers rather than hunters. This change first took place in the Middle East. Similar changes took place a little later in the Americas (where potatoes and maize were being grown) and in the Far East (where rice was first cultivated).

In the Middle East, humans first grew the cereal plants wheat and barley, and kept sheep and goats. Later, their livestock included cattle and pigs. Cultivating crops and keeping stock animals made it possible for permanent settlements to appear – life in villages began. Because of the more certain food supply, there was spare time, for the first time ever, for some people to do things other than hunt for food.

Ever since the cultivation of the first wheat and barley and the domestication of the first stock animals, humans have tried to obtain bigger yields from them. They cross-bred different maize plants (and barley plants) to obtain strains that produced more grain. They bred sheep and goats to give more milk and meat – selective breeding had begun. Today, animals and plants are bred for more than just food. For example, animals are used to produce a range of medicines and for research into the action of drugs.

Selective breeding is best described as the breeding of only those individuals with desirable features. It is sometimes called '**artificial selection**', as human choice, rather than environmental factors, is providing the selection pressure (compare this with natural selection, described above).

The methods used today for selective breeding are very different from those used only 50 years ago. Modern gene technology makes it possible to create a new strain of plant within weeks, rather than years.

TRADITIONAL SELECTIVE BREEDING

PLANTS



- 1 About 11 000 years ago, two strains of wild wheat were cultivated by farmers. Initially, all attempts at cross-breeding to produce wheats with a better yield gave only sterile offspring.
- 2 About 8000 years ago, a fertile hybrid wheat appeared from these two wild wheats. This was called emmer wheat and had a much higher yield than either of the original wheats.

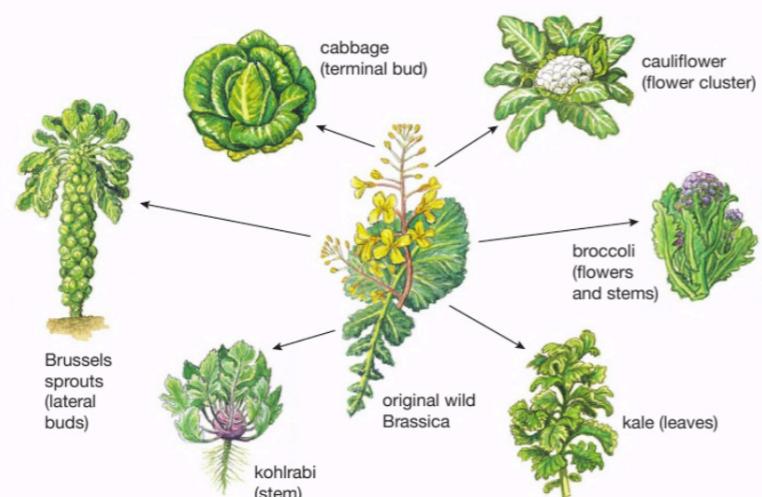
▲ Figure 19.7 Modern wheat is the result of selective breeding by early farmers.

- 3 The emmer wheat was cross-bred with another wild wheat to produce wheat very similar to the wheats used today to make bread. This new wheat had an even bigger yield and was much easier to 'process' to make flour.

DID YOU KNOW?

The production of modern bread wheats by selective breeding is probably one of the earliest examples of producing genetically modified food. Each original wild wheat species had 14 chromosomes per cell. The wild emmer hybrid had 28 chromosomes per cell. Modern bread wheat has 42 chromosomes per cell. Selective breeding has modified the genetic make-up of wheat.

Other plants have been selectively bred for certain characteristics. *Brassica* is a genus of cabbage-like plants. One species of wild *brassica* (*Brassica olera*) was selectively bred to give several strains, each with specific features (see Figure 19.8). Some of the strains had large leaves, others had large flower heads, and others produced large buds.



▲ Figure 19.8 Selectively breeding the original wild *brassica* plants to enhance certain features has produced several familiar vegetables.

Selective breeding has produced many familiar vegetables. Besides the ones produced from *Brassica*, selective breeding of wild *Solanum* plants has produced the many strains of potatoes that are eaten today. Carrots and parsnips are also the result of selective breeding programmes. Crop plants are bred to produce strains that:

- give higher yields
- are resistant to certain diseases (the diseases would reduce the yields)
- are resistant to certain insect pest damage (the damage would reduce the yield)
- are hardier (so that they survive in harsher climates or are productive for longer periods of the year)
- have a better balance of nutrients in the crop (for example, plants that contain more of the types of amino acids needed by humans)

DID YOU KNOW?

Plant breeders do not just breed plants for food. Nearly all garden flowers are the result of selective breeding. Breeders have selected flowers to have a particular size, shape, colour and fragrance. Roses and orchids are among the most selectively bred of our garden plants.

Figure 19.9 shows a field of potato plants. Some have been bred to be resistant to insect pests, while others were not selectively bred in this way.



▲ Figure 19.9 Selective breeding can reduce damage by pests. The plants on the right have been bred to be resistant to a fungal pest. Plants on the left are not resistant to the pest.

ANIMALS

Farmers have bred stock animals for similar reasons to the breeding of crops. They have selected for animals that:

- produce more meat, milk or eggs
- produce more fur or better quality fur
- produce more offspring
- show increased resistance to diseases and parasites

Again, like crop breeding, breeding animals for increased productivity has been practised for thousands of years. A stone tablet found in Iran appears to record the results of breeding domesticated donkeys. It was dated at over 5000 years old.

For many thousands of years, the only way to improve livestock was to mate a male and a female with the features that were desired in the offspring. In cattle, milk yield is an important factor and so high-yielding cows would be bred with bulls from other high-yielding cows.

Since about 1950, the technique of **artificial insemination (AI)** has become widely available. Bulls with many desirable features are kept and semen is obtained from them. The semen is diluted, frozen and stored. Farmers can buy quantities of this semen to inseminate their cows. The semen is transferred into the cow's uterus using a syringe. AI makes it possible for the semen from one prize bull to be used to fertilise many thousands of cows.

Modern sheep are domesticated wild sheep, and cows have been derived from wild aurochs. Just think of all the varieties of dogs that now exist. All these have been derived from one ancestral type. This original 'dog' was a domesticated wolf (Figure 19.10). In domesticating the wolf, humans gained an animal that

UNIT QUESTIONS

SKILLS → REASONING



1

For natural selection to operate, some factor has to exert a 'selection pressure'. In each of the following situations, identify both the selection pressure and the likely result of this selection pressure.

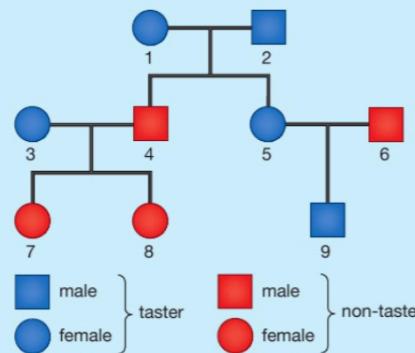
- Near old copper mines, the soil becomes polluted with copper ions that are toxic to most plants. (2)
- In the Serengeti of Africa, wildebeest are hunted by lions. (2)
- A farmer uses a pesticide to try to eliminate pests of a potato crop. (2)

(Total 6 marks)

SKILLS → ANALYSIS

2

PTC (phenylthiocarbamide) is a chemical that to some people has a very bitter taste, while other people cannot taste it at all. The diagram shows the inheritance of PTC tasting in a family.



- What evidence in the diagram suggests that the allele for PTC tasting is dominant? (2)
- Using T to represent the tasting allele and t to represent the non-tasting allele, give the genotypes of individuals 3 and 7. Explain how you arrived at your answers. (4)
- Why can we not be sure of the genotype of individual 5? (2)
- If individuals 3 and 4 had another child, what is the chance that the child would be able to taste PTC? Construct a genetic diagram to show how you arrived at your answer. (4)

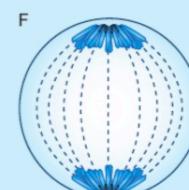
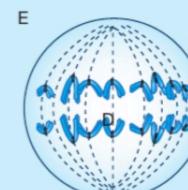
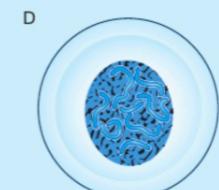
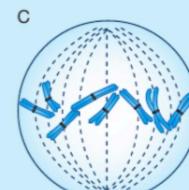
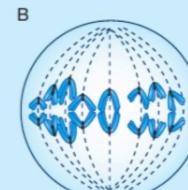
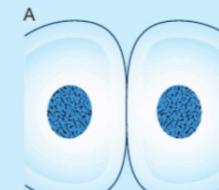
(Total 12 marks)

SKILLS → ANALYSIS



3

The diagrams A to F show an animal cell during cell division. The diploid number of this cell is eight.



- Put the pictures in the correct order. (2)
- Is the cell going through mitosis or meiosis? Explain your answer. (2)
- What is the diploid number of a human cell? (1)
- Describe two differences between mitosis and meiosis. (2)

SKILLS → CRITICAL THINKING



(Total 7 marks)

SKILLS → REASONING



SKILLS → ANALYSIS

SKILLS → INTERPRETATION