Unit
2B

Chapter 23
Human biology and everyday life

Figure 23.1 A patient receiving a blood transfusion

Unit content

The relevance of human biology to everyday life
The rate of change in human biology means that there is a range of alternative treatments available. Each treatment has its risks, ethical concerns and benefits based on individual variations and the condition being treated. Health choices can be based on myths or misconceptions about human biology.

Had you been born in the days of ancient Greece or Rome, from 700 BC to 400 AD, your life expectancy would have been just 28 years. A major jump in life expectancy occurred with the introduction of sewers and other public health measures, as these greatly reduced the spread of infectious disease.

By the year 1900, newborn Australian males could expect to live for 55 years and females for 59 years. Over the past 100 years, major advances in the prevention and treatment of disease have resulted in dramatic increases in life expectancy. Today's Australian babies can look forward to a life span of 78–83 years.

In this chapter we look at just a few of the medical advances that have come about as a result of increasing human biological knowledge—advances that have led to people leading longer and healthier lives. We also look at some alternative treatments for disease and some of the choices that people have to make to stay healthy.

Antibiotics

On 12 February 1941 a policeman lay dying in hospital in Oxford, England. Albert Alexander had scratched his face on a rose thorn a few months earlier. The scratch had become infected with bacteria and the infection had spread throughout his body. His head was covered with weeping abscesses, which spread to his eyes, one of which had to be removed. He also had abscesses on his arms and in his lungs. On that fateful day he was given an injection of a new drug that had never before been tried on humans. His condition immediately began to improve and after a week of treatment Alexander was well on the way to recovery. Unfortunately, supplies of the new drug ran out and the treatment could not be continued. The bacteria took over once again and Albert Alexander died on 15 March.

The new drug was penicillin and the first trial on Albert Alexander showed just how powerful it could be in the treatment of bacterial infections. Later trials, when larger quantities of penicillin were available, met with outstanding success.

Penicillin is an **antibiotic**—a substance that kills or inhibits the growth of micro-organisms. Before the development of antibiotics in the 1930s and 1940s many people died when minor injuries became infected. There was nothing that could be done if the body's own resources could not fight the infection. Even a simple scratch from a rose thorn could prove to be fatal.

Penicillin was not the first antibiotic to be discovered. Sulfonamides, commonly known as sulfur drugs, were discovered in 1935 by a German doctor, Gerhard Domagk. Although still in use today for some infections, sulfonamides were quickly replaced by penicillin because it has wider applications and fewer side effects.

Figure 23.2 A photograph of Alexander Fleming's original bacterial culture. The penicillin mould is growing at the top. Bacterial colonies around the mould have died.

The discovery of penicillin

In 1928 a bacteriologist, Alexander Fleming, was working in London. He had been growing bacteria in Petri dishes and was checking the cultures before discarding them. Fleming noticed that one dish had become contaminated and had a mould growing in it. It was not unusual for bacterial cultures to become contaminated with mould but this one was different. Around the mould the bacteria that had been growing in the dish had been killed (Fig. 23.2). Fleming recognised that this mould could be the source of a substance that could be used to control bacterial infections.

Fleming identified the mould as *Penicillium notatum* and called the antibacterial substance **penicillin**. He published his findings in

1929 and continued working with the mould. Some crude preparations were actually used to treat eye infections. Penicillin proved very difficult to purify because it was unstable and Fleming's research made no further progress.

In 1935 an Australian, Howard Florey (Fig. 23.3a), was appointed Professor of Pathology at Oxford University. He was interested in searching for antibacterial chemicals and he employed a biochemist, Ernst Chain (Fig. 23.3b). Chain was a German Jew who had fled to England to escape the Nazis. In 1938 Chain read Fleming's 1929 report and he began work on cultures of *Penicillium notatum*. Chain succeeded in extracting relatively pure penicillin, which was tested on mice that had been infected with bacteria. Experiments on the mice were promising and led to successful testing on humans.

At this time Britain was at war with Germany, and Florey was unable to get enough British support to develop commercial production of penicillin. He travelled to the United States and commercial production was begun in Illinois in 1942. Penicillin saved the lives of countless soldiers whose war wounds became infected. Alexander Fleming, Howard Florey and Ernst Chain were awarded a Nobel Prize for their discoveries.

Penicillin works by preventing the synthesis of the walls of the bacterial cells. In this way it inhibits the reproduction of bacteria. About 30% of antibiotics used in Australia today are penicillin based. However, the value of penicillin has been reduced because many bacteria have evolved a resistance to it. Also, about 10% of people are allergic to penicillin so that there is a continuing search for new antibiotics.

Other antibiotics

Penicillin was so successful that it stimulated a search for other micro-organisms that produce antibiotic substances. In 1943 **streptomycin** was discovered. It was produced by an actinomycete that lives in soil. **Actinomycetes** are bacteria that produce branching filaments rather like the threads of moulds. Most of the antibiotics in use today, such as erythromycin, neomycin, tetracycline and vancomycin, have been developed from actinomycetes. These substances interfere with protein synthesis in the cells of the target bacteria.

(a)

(b)

Figure 23.3 **(a)** Howard Florey (right) with an assistant; **(b)** Ernst Chain worked with Florey to extract the active substance from the *Penicillium notatum* mould

Cephalosporin is an antibiotic that, like penicillin, is derived from a fungus. It has a similar action to penicillin, interfering with synthesis of the cell wall, but is much less likely to result in allergic reactions.

Use of antibiotics

Each antibiotic is effective for only certain types of bacterial infection and cannot be used to treat viral infections. This is why antibiotics are available only on prescription from a doctor. The doctor must assess the most likely cause of the infection and prescribe the most appropriate antibiotic. In some cases laboratory tests may be necessary to determine which drug will be most effective against the infecting bacterium.

Bacteria may be identified by staining techniques or by the appearance of the bacterial colonies when they are cultured in a Petri dish. Cultures can also be tested with a range of antibiotics to determine those to which they are most sensitive (Fig. 23.4).

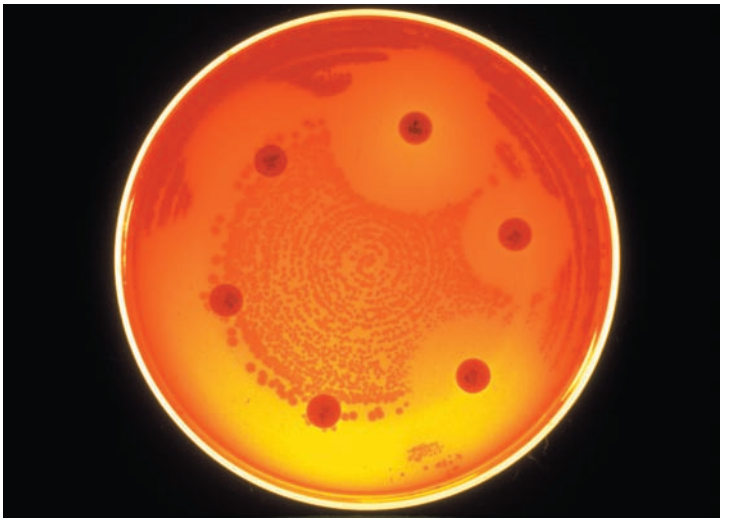

Figure 23.4 Comparing antibiotic sensitivity of bacteria using a culture plate with antibiotic assay discs. Notice the clear area where bacterial colonies have not grown around some of the discs. The antibiotic impregnated in each of those discs is effective against that species of bacteria

Antibiotic resistance

It was believed that the advent of antibiotics would result in the virtual elimination of bacterial infections. However, just four years after the mass production of penicillin began in 1943, strains of bacteria began to appear that were resistant to the drug.

Resistance develops by natural selection. Some bacteria will be more resistant to the antibiotic than others of the same species. If some of these resistant bacteria survive attack by the antibiotic, they will be able to reproduce and pass on their resistance to the next generation of bacteria. In this way resistant strains of certain bacteria may develop.

In the 1970s and 1980s doctors were likely to prescribe antibiotics as a precaution—just in case an infection occurred. Antibiotics are also widely used in agriculture. This wide exposure to them has hastened the development of resistance. Doctors are now much more careful about prescribing antibiotics and patients are advised to make sure that they complete the whole course of the drug to make sure all bacteria are eliminated.

Some strains of bacterial species have become **multiple drug resistant**; that is, they are resistant to many of the available antibiotics. Infections with these 'super bugs' are becoming increasingly difficult to treat.

Prevention of misuse and abuse of antibiotics will slow the development of resistance but there is no way of stopping it altogether. Strategies being used to overcome the problem are to develop new classes of antibiotics, to revive old antibiotics by using them in combination with other substances, and by genetic engineering of bacteria to disable antibiotic-resistant genes.

For more information on antibiotics go to:

- http://health.howstuffworks.com/question88.htm
- http://www.biotechnologyonline.gov.au/human/antibiotics.cfm

EXTENSION

Antibiotic-resistant strains of bacteria develop by natural selection, but how do bacteria acquire the genes for antibiotic resistance in the first place?

Research the various ways that bacterial genes can change so that the bacteria become resistant to antibiotics.

Antiviral drugs

Antibiotics are ineffective against viruses so there is still no treatment for common ailments such as colds and the 'flu. This has led to a hunt for chemicals that could be used to treat viral diseases.

Viruses enter a host cell and the virus DNA or RNA induces the cell to produce new virus particles. These particles can then leave the cell and infect new host cells.

The way in which viruses replicate makes it difficult to find drugs that will treat viral infections. Since the host cell produces the new virus particles, any drug that interferes with virus replication is likely to be toxic to the host. Early research involved culturing cells, infecting them with a virus and then trying different chemicals to see whether the amount of virus decreased. This time-consuming and hit-or-miss technique produced little result.

In the 1980s it became possible to determine the genetic sequences of viruses so that scientists could find out exactly how viruses work. Research today is aimed at identifying viral proteins that can be disabled by specially designed chemicals. If the proteins are very different from human proteins, there should be few side effects from the use of such a drug. The need to deal with the human immunodeficiency virus (HIV) has stimulated research into antiviral treatments. Quite a large number of antiviral drugs are now available and many more will be developed in the future.

Immunisation

Immunisation against infectious disease is one of the most significant public health measures that has ever been introduced. Epidemics of diseases like polio, diphtheria, tuberculosis, smallpox and many others were once feared because of the suffering and death that they caused. Today, especially in countries like Australia, these diseases are rare or unknown and the population do not live in fear of an outbreak of infectious disease.

Despite the outstanding success of immunisation programs, there are still many people who are opposed to immunisation. Their opposition arises because there are risks involved. A person can suffer a severe and debilitating reaction to a vaccine. However, such incidents are extremely rare and most people accept that the benefits of immunisation far outweigh any risks involved. In 2006, over 90% of Australian children up to the age of two years were fully immunised with all of the recommended vaccines. The recommended vaccination schedule for Australians is shown in Table 22.2 on page 299.

Immunity

Foreign molecules entering the body can trigger an immune response. Substances that are capable of stimulating an immune response are called **antigens**. They are large molecules of protein, lipid, carbohydrate or nucleic acid. Large molecules produced in a person's own body do not cause an immune response. These are called **self-antigens**. Foreign compounds that do trigger an immune response are **non-self-antigens**.

An **antibody** is a specialised protein that is produced in response to a non-self-antigen. Antibodies belong to a group of proteins known as **immunoglobulins**. They are Y-shaped molecules, and the two tips of the Y are active sites where the antibody can combine with a specific antigen. The antibody produced in response to an antigen can combine with that antigen to form an **antigen–antibody complex**. The active site on the antigen and the active part of the antibody fit together something

like a key in a lock. Each antibody can combine with only one particular antigen, in the same way that a key will only open a particular lock (Fig. 23.5).

On combining with the antigen, the antibodies inactivate it so that symptoms of infection do not develop. At the same time special cells are formed called memory cells. **Memory cells** allow the response to occur more rapidly if the same antigen should again enter the body.

On the first exposure to an antigen, the body's immune system usually responds fairly slowly. Several days may be required to build up large amounts of antibodies. With a second or subsequent exposure to the same antigen, the response is much faster because of the activity of the memory cells (Fig. 23.6).

This is the principle on which vaccination works. The first exposure to the antigen is the encounter with the vaccine. Memory cells for that antigen are formed. If exposure to the antigen (the infecting micro-organism) occurs again, its effects can be neutralised quickly before symptoms of disease develop. Some diseases require up to three inoculations of vaccine in order to build up enough memory cells to be effective against an attack by the pathogen.

Figure 23.5 **(a)** Antibodies combine with antigens at specific sites; **(b)** antigen molecules combine with an antigen to form an antigen–antibody complex

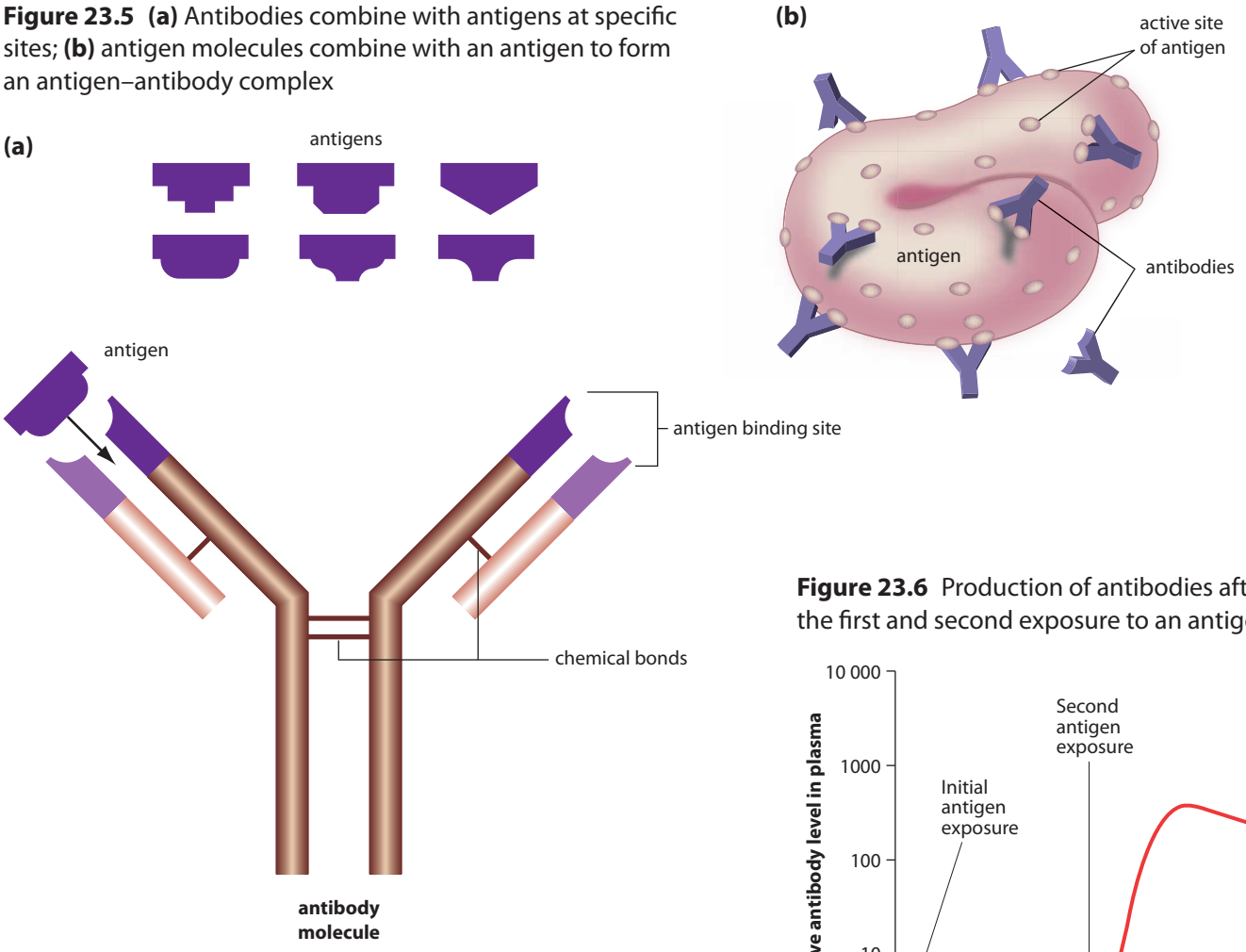

Figure 23.6 Production of antibodies after the first and second exposure to an antigen

Types of immunity

A person is immune to a disease if the body is able to respond quickly enough to prevent symptoms occurring when the body is invaded by pathogenic micro-organisms. Such an ability to respond rapidly may be natural or it may be artificial. **Natural immunity** occurs without any human intervention; **artificial immunity** results from giving people an antibody or an antigen that triggers off the immune response so that they will make their own antibodies.

You can play the Immune System Defender Game at http://nobelprize.org/educational_games/medicine/immunity

Both natural and artificial immunity can be either passive or active. **Passive immunity** is when a person is given antibodies produced by someone else. The individual's body plays no part in the production of antibodies. This can occur naturally when antibodies from the mother pass across the placenta to a developing foetus or when the mother's antibodies are passed to the baby in breast milk. It can also be gained artificially when a person is injected with antibodies to combat a particular infection. This is often done when a person is exposed to pathogens that cause serious diseases, such as tetanus, diphtheria and rabies. Antibodies are given so that immunity is established immediately. Passive immunity is short-lived; it lasts only as long as the antibodies are in the body.

Active immunity results when the body is exposed to a foreign antigen and manufactures antibodies in response to that antigen. This type of immunity is prolonged because, although the amount of the antibody produced gradually decreases, the 'memory' of that antigen persists through the memory cells once the antigen has been dealt with. Should a subsequent infection involving the same antigen occur, the appropriate antibodies can be produced very quickly before the infection can produce any disease symptoms. Such immunity lasts for many years, sometimes for life. Active immunity to a disease can result from an actual attack of the disease (natural active immunity) or from an injection of the antigens associated with the disease (artificial active immunity). Table 23.1 summarises the types of immunity.

Figure 23.7 Smallpox causes a rash with raised pustules filled with pus-like fluid

History of vaccination

Smallpox was a highly contagious disease caused by a virus. It was characterised by a rash that turned into raised pustules filled with pus-like fluid. This stage was accompanied by a high fever and death was quite common. Over time the pustules dried out and formed scabs. When the scabs fell off they often left permanent scars, or pock marks, on the skin.

Lady Mary Wortley Montagu was the wife of the British ambassador to Turkey in the early 1700s. While living in Turkey she noticed that many Turks deliberately infected themselves with smallpox. They used a needle to prick the skin and introduce powdered scabs from a smallpox victim into their blood. The Chinese had, for centuries, been using

Table 23.1 Types of immunity

Type	Natural	Artificial
Passive	Antibodies enter the bloodstream across the placenta or in breast milk	Antibodies are injected into the bloodstream
Active	Ability to manufacture antibodies results from an attack of the disease	Ability to manufacture antibodies results from an injection of an antigen (a vaccine)

a similar practice; they inhaled the scab powder. Deliberately infecting themselves with smallpox usually resulted in a mild illness, although there was a risk that it could be serious and death could even occur. The big advantage was that the person became immune to smallpox.

On her return to England, Lady Mary introduced the procedure in 1724. It was strongly opposed by doctors but the idea caught on and many people, including the royal family, were inoculated.

An English rural doctor, Edward Jenner (Fig. 23.8), had been inoculated as a young boy. One of his patients, a milkmaid, told him that she could not catch smallpox because she had previously had cowpox. Cowpox was similar to smallpox but the symptoms were very mild and death never occurred. Jenner became interested and noted that many people who worked with cows never got smallpox, even when repeatedly exposed to infection.

Figure 23.8 A painting of Edward Jenner infecting a young boy with cowpox

In 1796 Jenner took the bold step of deliberately infecting a young boy with cowpox. He allowed the boy to recover from the cowpox and then infected him with smallpox by injecting pus from smallpox under the boy's skin. The boy did not get smallpox. Jenner's method caught on and was called vaccination after the Latin name for cow—*vacca*.

It is interesting to note that under the ethical standards of today, Jenner would never have been able to perform these experiments. There was no assessment and approval by an ethics committee, no informed consent and the risk of harming the subject was high.

It was not until the 1880s that further advances in the vaccination of humans were made. Louis Pasteur (Fig. 23.9), the great French scientist, developed vaccines for rabies and cholera. Pasteur's great advance was that his vaccines consisted of artificially weakened pathogens, whereas Jenner had used a naturally weak form of the disease organism.

Figure 23.9 Painting of Louis Pasteur working in his laboratory in Paris

Vaccines have now been introduced for a large number of infectious diseases (see Table 23.2) and, as a result, much human suffering has been alleviated. A worldwide effort, coordinated by the **World Health Organization (WHO)**, has completely eliminated smallpox. Mass vaccinations and surveillance to detect new cases led to the world being declared free of smallpox in 1980—the first infectious disease to be completely eliminated.

The WHO targeted polio for global eradication by 2000. Polio has now been eliminated from most parts of the world, but it still exists in a few places where armed conflict makes it difficult to run immunisation programs.

Types of vaccine

A **vaccine** is an antigen preparation used in immunisation. Vaccines are of four types:

1. Vaccines containing live **attenuated** micro-organisms—micro-organisms of reduced **virulence**. That is, micro-organisms with a reduced ability to produce disease

Table 23.2 Dates of introduction of human vaccines

Year	Disease	Year	Disease
1798	Smallpox	1935	Yellow fever
1885	Rabies	1955	Injectable polio vaccine
1897	Plague	1962	Oral polio vaccine
1923	Diphtheria	1964	Measles
1926	Pertussis (whooping cough)	1967	Mumps
1927	Tuberculosis	1970	Rubella
1927	Tetanus	1981	Hepatitis B

Source: WHO

symptoms so that the immunised person does not contract the disease, but does manufacture antibodies against the antigen. Pasteur first used this method to immunise chickens against fowl cholera, and sheep and cattle against anthrax. He attenuated the bacteria by exposing them to a temperature of 42 °C (5 °C higher than normal body temperature) for about a week. Some vaccines containing live attenuated micro-organisms are those for immunisation against polio, tuberculosis, rubella (German measles), measles, mumps, yellow fever and chickenpox.

2. Vaccines containing dead or **inactivated** micro-organisms. Immunity produced in this way is not usually as prolonged as it would be with immunisation using live attenuated micro-organisms. Examples of vaccines of this type include cholera, typhoid, whooping cough and influenza vaccines.
3. Vaccines containing a toxin produced by the bacterium or virus. In cases where the pathogen produces an effect in humans by liberating toxins, it is not necessary to use living or dead micro-organisms for immunisation. The toxins produced by the pathogen can be inactivated so that when they are injected into someone they do not make the person ill. Such inactivated toxins are called **toxoids**. Injections of toxoids are used to immunise against diphtheria and tetanus.
4. Vaccines containing synthetic 'human-made' substances, or **biosynthetic** vaccines. The immune system responds to the synthetic substance as if it were an antigen. *Haemophilus influenzae* type B (HiB) vaccine is biosynthetic.

The vaccines available today may be injected or taken by mouth as a liquid or a tablet. In the future they may be delivered in food. Scientists are trying to genetically engineer foods such as bananas, potatoes, rice or peas so that the food will contain part of a pathogenic organism. This would be of great benefit in developing countries. There would be no need for the special transport or storage that vaccines require and trained staff would not be needed to give the vaccines.

EXTENSION

Most vaccines are still injected but a lot of the current research effort is aimed at producing non-injectable vaccines.

- Find out some of the ways in which vaccines may be delivered in the future.
- What would be the advantages of non-injectable vaccines?

Blood groups and transfusions

A blood **transfusion** can be given to a person suffering from excessive blood loss, some types of anaemia, leukaemia, haemophilia or other conditions. It involves blood, or a blood product, from a donor being injected directly into the patient's bloodstream.

Some of the earliest, and unsuccessful, transfusions took place in the seventeenth century and involved the transfer of animal blood to humans. Early attempts at transfusions of whole blood between humans met with either spectacular success or the death of the patient.

In 1901, Karl Landsteiner (Fig. 23.10), an Austrian doctor, experimented by mixing samples of blood taken from different people. His research led to the discovery of what is now called the **ABO blood group system**. Thirty-nine years later, Landsteiner, by then a citizen of the United States, discovered the **Rh blood group system**. A number of additional blood group systems have been discovered, by Landsteiner and others, but the ABO and Rh groupings are of particular importance in blood transfusions.

ABO blood groups

As we saw earlier, an **antigen** is a substance that is capable of stimulating the formation of a specific protein called an **antibody**. **Antibodies** are produced in response to an antigen and are able to combine with the antigen that initiated the response.

The surfaces of red blood cells contain particular antigens that are able to react with appropriate antibodies in the plasma. Antigen–antibody reactions are the basis for the various classifications of blood groups.

There are two antigens involved in the ABO classification of blood groups: antigen A and antigen B. On the surface of the red blood cells a person may have either antigen A, antigen B, both antigens, or neither. These four possibilities correspond to the four groups of the ABO system—group A (antigen A), group B (antigen B), group AB (both antigens) and group O (neither antigen). The body's ability to make the antigens, and hence a person's ABO blood group, is inherited.

The antibody that reacts against antigen A is called anti-A, and that which reacts against antigen B is called anti-B. A person is not normally able to produce antibodies that react against his or her own red blood cells. Thus, a group A person can produce only the antibody anti-B, a group B person can produce only anti-A, a group AB person cannot produce either antibody, and a group O person can produce both. This is summarised in Table 23.3.

Figure 23.10 Karl Landsteiner discovered the ABO and Rh human blood systems

Table 23.3 A summary of the ABO blood groups

Blood group	Antigens on red blood cells	Antibodies in plasma
A	Antigen A	Anti-B
B	Antigen B	Anti-A
AB	Antigen A and antigen B	Neither anti-A nor anti-B
O	Neither antigen A nor antigen B	Both anti-A and anti-B

Rh blood groups

The Rh blood group system is so named because Landsteiner used the blood of rhesus monkeys in his initial investigations. Like the ABO system, it is based on antigens that occur on the surface of the red blood cells.

A person with Rh antigens is said to be Rh positive; a person without these antigens is Rh negative. An individual without the Rh antigens is able to produce an anti-Rh antibody that reacts against those antigens. Rh-positive individuals cannot produce anti-Rh antibody.

For more on blood groups go to:

- http://www.betterhealth.vic.gov.au/BHCV2/bhcarticles.nsf/pages/Blood_groups
- http://nobelprize.org/educational_games/medicine/landsteiner/readmore.html (where you can also play the Blood Typing Game)

Transfusions

As indicated previously, a transfusion transfers blood, or one of the components of blood, from one person to another. For most transfusions it is necessary to match the blood groups of the donor and the recipient, although the use of some blood products, such as clotting factors, may not require matching of blood groups.

The mixing of blood types that are incompatible can cause the erythrocytes to clump together, or **agglutinate** (Fig. 23.11). If the receiver's blood contains, or is able to make, antibodies against the antigens on the donor's red cells, the foreign cells will clump together and disintegrate. It is therefore essential that the blood group of the receiver and donor be the same. The ABO blood group of the donor is always matched to that of the receiver when transfusions are given.

Rh blood groups are also matched for transfusion purposes. The anti-Rh antibody is not normally present in the plasma of Rh-negative people but it is produced on exposure to the Rh antigen. The first transfusion of Rh-positive blood to an Rh-negative patient does not usually cause problems because the antibodies are produced slowly. However, that first exposure sensitises the person, so that any subsequent exposure results in very rapid production of antibodies. Clumping of the red cells results in a manner similar to ABO incompatibility.

Types of transfusions

Whole blood (Fig. 23.12) is blood as it is taken from the donor but with a chemical added to prevent clotting. Transfusions of whole blood are used mainly in cases of severe blood loss.

Red cell concentrates are the most widely used component of blood. They are produced by spinning blood at very high speed in a centrifuge. The heavier cells sink to the bottom, leaving the lighter plasma on top. The concentrate may or may not have platelets and white blood cells (leucocytes) removed. Transfusions of red cell concentrates are used for patients suffering from heart disease or severe anaemia.

Figure 23.11 Mixing of blood: (left) with incompatible plasma; red cells are clumped together. (right) with compatible plasma; no clumping of red cells

Plasma, the liquid part of the blood, may be given to patients requiring extra clotting factors for control of severe bleeding, or to patients with liver disease.

Platelet concentrates are given to patients who have abnormal platelets or a reduced number of platelets.

Cryoprecipitate is obtained by freezing the plasma and thawing it slowly. When the plasma is thawed the cryoprecipitate remains solid. It contains many of the substances necessary for blood clotting. Cryoprecipitate may be used to treat some forms of haemophilia, but it is most often used for severe bleeding.

Immunoglobulins are a group of proteins that act as antibodies. They are extracted from the blood and used

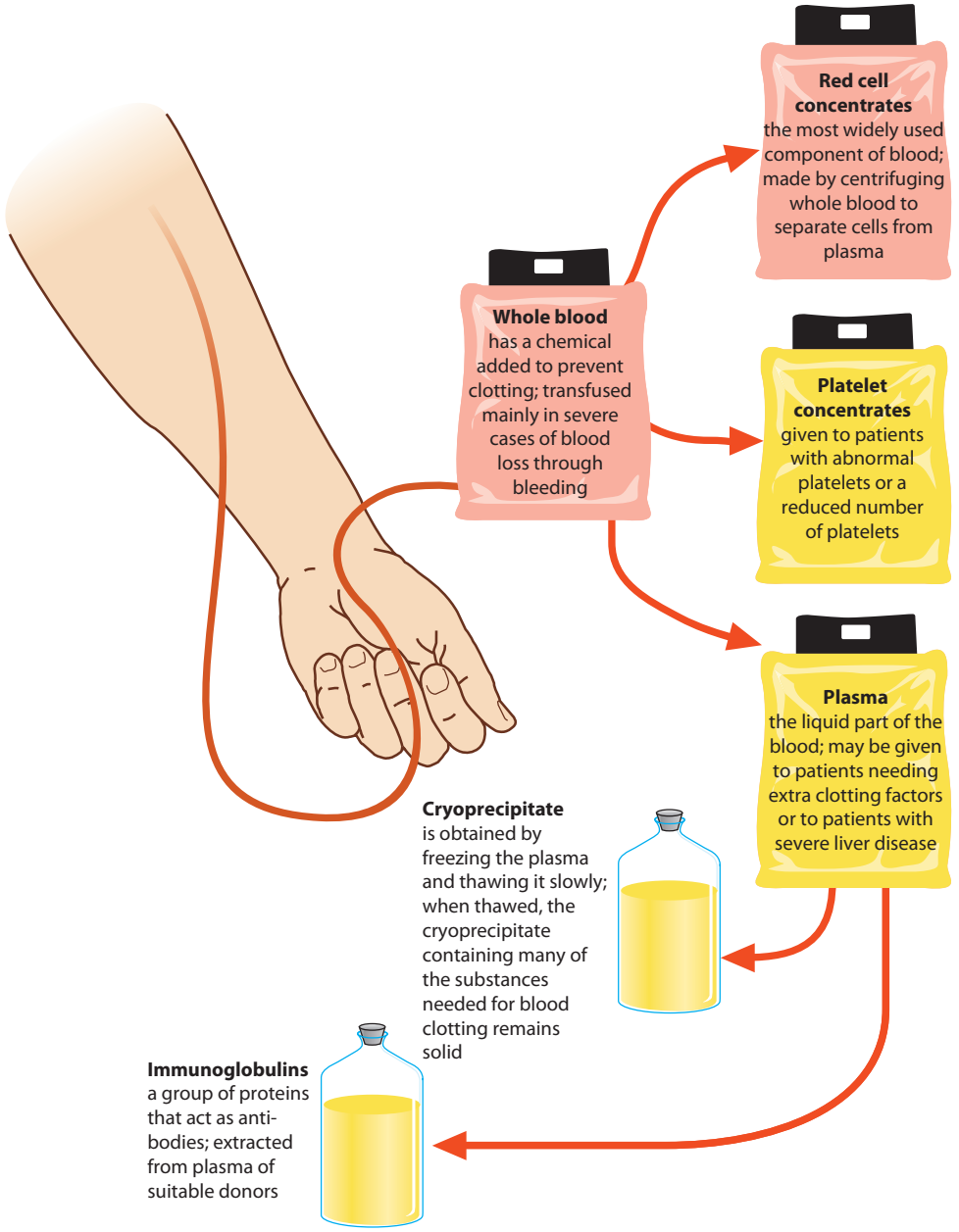

Figure 23.12 Blood and blood products used for transfusion

for patients who are deficient in antibodies. Particular immunoglobulins from certain donors are used to treat patients who have no immunity to a particular disease. For example, tetanus immunoglobulin may be used to treat tetanus.

An **autologous transfusion** is when the patient's own blood is used. The blood is collected from the patient prior to an operation that may require a transfusion. Such transfusions are often used for elective surgery and the blood is collected about four weeks before the operation. Autologous transfusions eliminate the risk of transmission of disease and most possible side effects of the usual transfusions.

Becoming a blood donor

In Australia blood is free. It is collected from donors by the Red Cross Blood Transfusion Service and no charge is made for the blood or for blood products. Most states and territories in Australia have laws that prohibit payment for blood donations or charges for blood supplied to patients.

The Red Cross is always in need of blood donors and at times there are critical shortages of certain types of blood. Any healthy person aged 18–65 years may be a donor and in some states the minimum age is 16 years with parental permission. Donating blood is a worthwhile way of contributing to society. It costs nothing and gives satisfaction by helping others.

Screening blood and blood products

Donated blood is checked carefully to make sure that it does not contain any disease-causing micro-organisms. When a person is infected with a pathogen the body produces antibodies against the foreign organism. These antibodies will appear in the blood of the infected person. **Blood screening** tests a sample of each blood donation for antibodies to make sure that diseases such as HIV and hepatitis are not passed on to patients who receive donated blood.

Unfortunately, there is a brief 'window period'—a short time between infection and the development of antibodies. During this period screening for antibodies would not detect the presence of a pathogen. For HIV and hepatitis the window period can be up to three months.

A technique called nucleic acid testing can now be used to detect the presence of viral RNA in blood. This test looks for the virus itself rather than the antibodies produced in response to the virus. Nucleic acid testing, introduced in Australia in 2000, has significantly reduced the window period but there is always a small risk that blood used for transfusions may be infected.

Genetic engineering

Genetic engineering, also known as **recombinant DNA technology**, involves the introduction into cells of DNA that is foreign to the organism or that has been modified in some way. In Chapter 22 the potential for curing genetic disorders by replacing faulty genes with healthy ones was discussed. However, genetic engineering of organisms other than humans has already produced great health benefits for humankind.

In 1982 insulin produced by genetically engineered bacteria was approved for the treatment of diabetes. Until that time insulin was extracted from the pancreas of pigs and cattle. Using genetic engineering techniques the human gene that has the code for insulin production was introduced into bacterial cells. The bacteria became insulin factories and are cultured in vats where they produce insulin used to treat diabetes. The insulin produced by the bacteria is identical to human insulin because the human gene was engineered into the bacteria. This insulin does not produce the side effects suffered by some people when insulin from cattle or pigs was used.

Human growth hormone (hGH) used to be extracted from human bodies. Use of the hormone involved the risk of transmission of viral and other diseases. hGH is now made by genetically engineered *Escherichia coli* (*E. coli*) bacteria without the risk of side effects when used in humans. The first genetically engineered vaccine for use in humans, the hepatitis B vaccine, was introduced in 1986. Since those early days of the technology, many other hormones, drugs and vaccines have been developed using genetic modification.

Complementary and alternative medicine

Alternative medicine is treatment that is used in place of conventional medical care; for example, when cancer is treated with a special diet rather than with methods recommended by a cancer specialist. Examples of alternative therapies are herbal

medicine, Chinese medicine, chiropractic, osteopathy, naturopathy, iridology, reflexology, aromatherapy, acupuncture, massage and meditation.

Complementary medicine is when one or more of the alternative methods is used at the same time as standard medical treatment; for example, using acupuncture (Fig. 23.13) to alleviate some of the side effects of cancer treatment.

Figure 23.13
Acupuncture is an ancient Chinese medical tradition

Many alternative therapies have been in use for hundreds of years and some, such as Chinese medicine, for thousands of years. With the advent of vaccination and antibiotics, infectious diseases were no longer major killers and natural therapies fell out of favour. It is only in the past 20 or 30 years that alternative therapies have become popular again. More than 60% of Australians now use alternative therapies at some time. A number of factors have contributed to this rise in popularity, such as the negative side effects of some standard treatments, a less personal doctor–patient relationship, and increasing ethnic and cultural diversity.

Many alternative therapies are increasingly being seen by the medical profession as having value, particularly when used to complement conventional medical practices. About one-fifth of Australian doctors now practice or study complementary medicine. Australian universities and colleges are also recognising some alternative therapies and offer courses in areas such as acupuncture, chiropractic and herbal medicine.

It is important to realise that many alternative therapies have not yet undergone rigorous scientific evaluation. They may appear to work for various reasons. Some diseases are self-limiting; they get better over time no matter what treatment is given. The placebo effect is very powerful, where the patient's belief in the value of the treatment leads to improvement even if the treatment has no real effect. Some conditions such as allergies and arthritis are cyclical and spontaneous remission may make it seem as if the alternative treatment is working.

Other problems with alternative therapies are that some of the chemical substances used may not be regulated by the Therapeutic Goods Administration, so that their purity and safety cannot be guaranteed. There may also be little regulation of the practitioners of alternative therapies.

Alternative therapies have their place and each of us has to make up our own mind about their use. Information is readily available from health departments and through reputable Internet sites about the reliability and pitfalls of the various treatments. You should find out about the advantages and disadvantages before embarking on an alternative treatment. If you are using alternative therapies, it is important to tell your doctor. Some alternative therapies may have adverse effects on conventional procedures, such as when herbal medicines interfere with the activity of prescribed drugs. St John's wort is an example; it disrupts immunosuppressive drugs and warfarin, a drug that reduces the risk of blood clots.

Working scientifically

Activity 23.1 Testing penicillin

Howard Florey's team at Oxford succeeded in isolating penicillin. The crucial experiment to test the effectiveness of penicillin as an antibiotic was carried out on 25 May 1940. At 11.00 am on that day eight mice, all the same weight and age, were each injected with 100 million streptococci, a type of bacterium. Previous experiments had shown that an injection of that size would kill all mice injected.

After the injection of streptococci four mice were put back in their cages and given no further treatment. The other four mice were divided into two pairs—pair A and pair B. One hour after the injection of the streptococci the A mice were injected with 10 mg of penicillin and the B mice were given 5 mg of penicillin. No further treatment was given to the A pair, but the B pair were given four more injections of penicillin each of 5 mg over a period of 12 hours.

The results of this first experiment to test penicillin are shown in Table 23.4.

Table 23.4 Result of testing mice injected with streptococci

Treatment	Mouse								
Penicillin	A1	10 mg							survived 4 days
	A2	10 mg							survived 6 days
	B3	5 mg	5 mg	5 mg	5 mg	5 mg			survived 13 days
	B4	5 mg	5 mg	5 mg	5 mg	5 mg			survived 6 weeks+
No penicillin	5					died			
	6						died		
	7						died		
	8							died	
Hours after infection		2	4	6	8	10	12	14	16

Use the description of Florey's experiment and the data in Table 23.4 to answer the following questions.

1. What was the independent variable in this experiment?
2. What was the dependent variable?
3. List the variables that were controlled in the experiment.
4. Explain why four of the mice were given a lethal dose of streptococci but no penicillin.
5. What would Florey have been able to conclude from this experiment?
6. Over the following weeks Florey carried out many similar experiments. Why would it be necessary to repeat the same experiment a number of times?

Activity 23.2 A model of genetic engineering

Go to the web page http://www3.iptv.org/exploremore/ge/what/insulin.cfm#1. Use the model provided to work through the steps involved in isolating the gene for insulin production in humans and splicing the gene into the DNA of a bacterium.

REVIEW QUESTIONS

1. List three advances in public health that have led to increased life expectancy.
2. **(a)** What is an antibiotic?
   (b) Explain how some antibiotics work.
3. **(a)** Explain how strains of bacteria can become resistant to an antibiotic.
   (b) What are some of the problems that arise from bacterial resistance?
4. Why has it been difficult to develop drugs that are effective against viral infections?
5. People who inhaled or injected material from smallpox scabs ran the risk of severe disease and perhaps death. Why did people run the risk?
6. What was the big advance in disease prevention that was achieved by Edward Jenner?
7. Louis Pasteur took Jenner's work a step further. What was Pasteur's contribution to disease prevention?
8. **(a)** What methods were used to totally eradicate smallpox from the world?
   (b) Why has it not yet been possible to eradicate polio in the same way?
9. **(a)** What is an antigen?
   (b) Explain the difference between self-antigens and non-self-antigens.
10. **(a)** What is an antibody?
    (b) Explain how it is that an antibody is specific to a particular antigen.
11. Explain the importance of memory cells in immunity.
12. What is the difference between:
    (a) natural and artificial immunity?
    (b) active and passive immunity?
13. **(a)** How could passive immunity be gained artificially?
    (b) How could active immunity be acquired naturally?
14. **(a)** What is a vaccine?
    (b) Describe four ways in which vaccines may be produced.
15. Why is it rare to get a disease such as measles or chickenpox more than once?
16. Explain why early attempts at blood transfusion were sometimes successful but, more often, led to the death of the patient. What would have caused the death of those patients who died?
17. **(a)** What antigens and antibodies are involved in the ABO blood group system?
    (b) What determines the ABO blood group to which a person belongs?
18. Why is donated blood screened before use?
19. **(a)** Why is genetic engineering also known as recombinant DNA technology?
    (b) Explain what happens when an organism is genetically engineered.

APPLY YOUR KNOWLEDGE

1. Typhoid is a disease caused by a bacterium. To make a positive diagnosis of typhoid, a sample of the patient's blood is taken and mixed with typhoid bacteria. If the bacilli agglutinate (clump together) the patient has typhoid.
   (a) Why is this result a positive diagnosis for the disease?
   (b) Could the person be suffering from some other disease?
2. Judged by today's ethical standards Edward Jenner should not have trialled the effect of inoculating people with cowpox in the way that he did. Do you think that ethics committees are necessary to safeguard people's rights and interests? Is the growth of scientific knowledge being restricted because researchers have to meet ethical standards?

   List the advantages and disadvantages of imposing ethical requirements on scientific research. (Chapter 2 describes the ethical principles that must be met for scientific research.)
3. Haemolytic disease of the newborn may occur if a mother has Rh-negative blood and her developing foetus has Rh-positive blood. Some of the foetus's blood may leak across the placenta and mix with the mother's blood. The mother will then produce anti-Rh antibodies, which can move back across the placenta and destroy the baby's red cells. The cause of haemolytic disease of the newborn was discovered in 1940. A vital clue that enabled scientists to determine the cause was that first babies were never affected by the condition unless the mother had previously received a transfusion of Rh-positive blood.
   (a) Why would first babies rarely be affected by this condition?
   (b) Why would the condition be more likely to occur if a mother had previously received a transfusion of Rh-positive blood?
4. **(a)** A person was prescribed an antibiotic for a bacterial infection. After taking half of the tablets the infection cleared up, so the patient stopped taking them. Explain how this person's actions could contribute to the development of antibiotic resistance in the bacteria.
   (b) A person was prescribed an antibiotic for a bacterial infection of the throat. While taking the antibiotic tablets the patient developed a bacterial infection of the big toe. Explain why the antibiotics that the patient was taking for the sore throat did not prevent the growth of bacteria in the toe.
5. When blood plasma is given in a transfusion, would the donor of the plasma have to be the same ABO blood group as the receiver? Explain your answer.
6. Table 23.5 shows the result of testing blood samples from three different individuals with group A plasma and group B plasma. What is the blood group of each of the individuals D, E and F?

Table 23.5

Individual	D	E	F
Group A serum	Clumping	No clumping	Clumping
Group B serum	No clumping	No clumping	Clumping

7. Write a letter to a fictitious friend urging them to become a blood donor. In your letter explain the advantages of blood donorship to the individual and to society.