Making a muscle

Teresa Audesirk

Physiologist Teresa Audesirk explores the reasons underlying performance disparities among elite athletes

AQA: 3.6.3 Skeletal muscles

Edexcel A: 7.2 Contraction of skeletal muscle; 7.10 Fast and slow twitch muscle

OCR A: 5.1.5 (l) Muscle and muscular contraction

OCR B: 4.1.2 Metabolism and exercise

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Key words

Skeletal muscle
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Elite sport
Endurance training
Resistance training
Gender differences

The world record speed for the 100 metre sprint stands at 37.58 km h^{-1}. The fastest marathon runner ran at only 20.6 km h^{-1}, but kept going for 42.2 km. The heaviest weight ever lifted overhead was 263 kg. All these records were achieved by men. World records for female runners are roughly 10% slower than those for men, and for weightlifters the gender difference is nearly 37%.

Muscle contraction is due to the shortening of spindle-shaped cells — muscle fibres — that make up the muscle (see Box 1). Muscles contain a mixture of two main fibre types — fast and slow — which are classified based on their speed of contraction and how they generate ATP to power contraction.

Although the muscles of the three male record holders listed above all work in the same way, and each used his muscles at close to the maximum possible, these men would never be competitive against each other. So what is it about their muscles that allows each to excel in his own sport?

Muscles, like athletes, have different abilities

Differences in fast and slow fibre composition of the muscles of different athletes help explain their differing achievements. The calf muscle (gastrocnemius) is used to maintain posture, for sprinting and for distance running. Most people have about half fast and half slow fibres in this multi-tasking muscle, but its composition can differ significantly between individuals (see Figure 1). Elite marathon runners have gastrocnemius muscles with roughly 80% slow fibres. Their calf muscles are a bit like those in turkey legs, packed with myoglobin that gives a dark colour, and used continuously over long periods. In contrast, the calf muscles of elite sprinters have about 80% fast fibres — more like the white meat of turkey breast muscles, which are used for brief bursts of wing-flapping to escape danger. Do the different training regimes of elite marathon runners and elite sprinters create these dramatic differences? No, there is no evidence that fast and slow fibres can be interconverted.

Clearly, to bring home Olympic gold, athletes must first win a genetic lottery that provides them with a certain ratio of fast to slow muscle fibres in specific muscles, predisposing them to excel at particular sports. But their muscle fibre composition is only part of the picture (see Box 2).

Fast and slow fibres respond differently to training

Marathon runners and sprinters tend to have distinct physiques. Some of this difference is due to training.

Elite marathon runners use endurance training. They may cover over 160 km each week, in runs of 8–38 km, at speeds that allow their muscles to respire aerobically. Endurance training targets slow and fast intermediate muscle fibres in the legs. Capillaries proliferate around them, bringing them more oxygenated blood. Endurance-trained muscle fibres produce more oxygen-storing myoglobin. The number of mitochondria in the fibres, which generate ATP by aerobic respiration, also increases.

The type of endurance training seems to be important. Studies involving cycling training regimes show increases in the volume of leg muscles and in the diameter of slow muscle fibres. In contrast, the diameter of both slow and fast-intermediate fibres decreased in college students training for their first marathon by running increasing distances. Slow and fast-intermediate fibres rely entirely or partly on a steady supply of oxygen provided by diffusion; a smaller fibre diameter allows more oxygen to reach the innermost myofibrils. In the college student study,

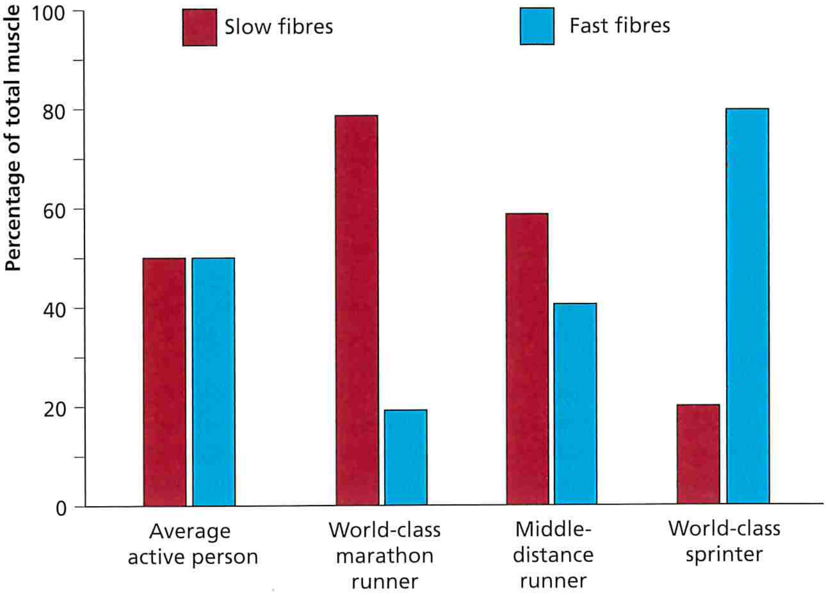


Figure 1 Relative numbers of fast and slow fibres in the calf muscle of legs predispose people to certain types of activity

Box 1 Composition of skeletal muscle

Figure 1.1 shows a typical skeletal muscle, encased in connective tissue that is modified at each end to form a tendon. Tendons attach to bones, enabling the muscles to move the body. The spindle-shaped cells of the muscle — muscle fibres — each contain many nuclei and are packed with myofibrils. Myofibrils consist of actin and myosin, the proteins that drive muscle contraction.

Most muscles contain a mixture of two main muscle fibre types — fast and slow. The smaller, slow fibres rely almost entirely on aerobic respiration to generate ATP. They respond more sluggishly and with less force than fast fibres but can contract repeatedly over long periods. Slow fibres are richly supplied with mitochondria and myoglobin — an oxygen-storing protein similar to haemoglobin that colours them red. They have a dense supply of capillaries. Postural muscles (such as the trapezius in the upper back) generally have more slow than fast fibres, allowing you to sit and study for hours without getting tired.

Fast fibres are larger, contain more myofibrils, and produce faster, more powerful contractions than slow fibres. They generate most of their ATP by anaerobic respiration (glycolysis), a process that yields much less ATP than aerobic respiration. These fibres have fewer capillaries and fewer mitochondria than slow fibres. Fast fibres hydrolyse ATP at an unsustainable rate during exercise, causing them to fatigue. Fast fibres are further divided into fast glycolytic fibres and fast intermediate fibres. Fast glycolytic fibres rely entirely on glycolysis and produce the fastest, strongest contractions. They are white because they lack myoglobin, and they fatigue very rapidly. Fast intermediate fibres use both aerobic and anaerobic respiration; their properties are intermediate between slow fibres and fast glycolytic fibres.

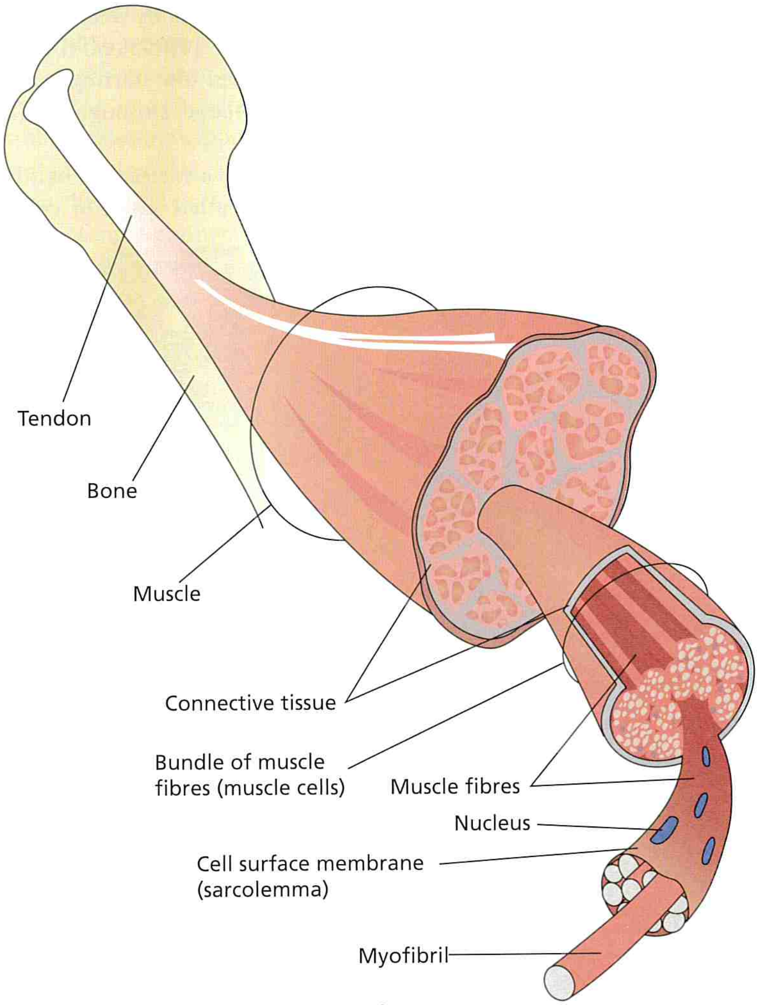


Figure 1.1 Structure of skeletal muscle

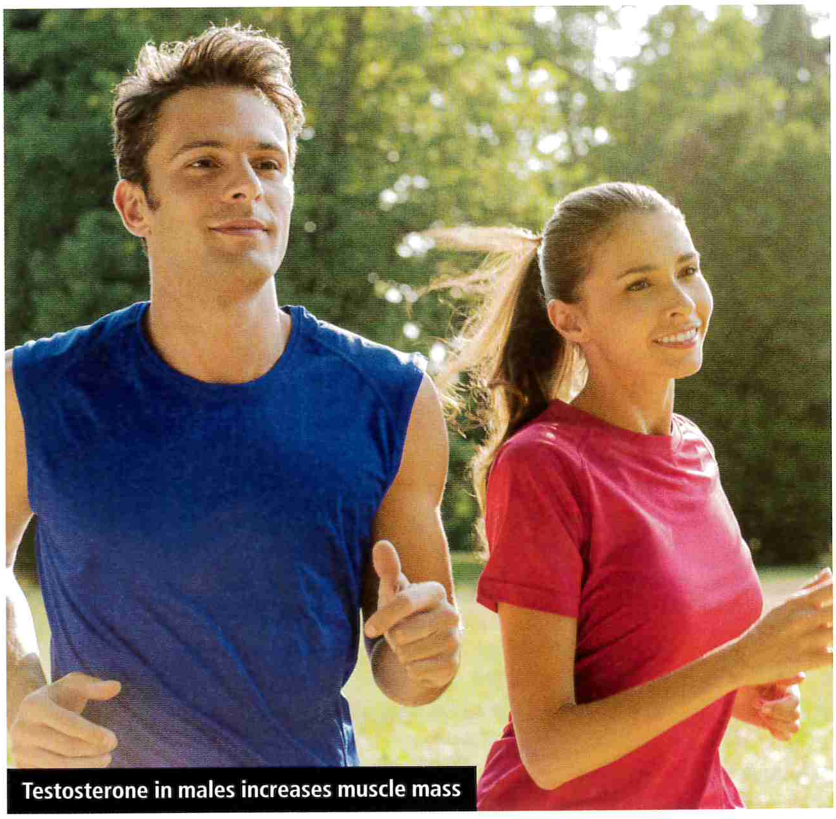
Box 2 Winning the genetic lottery

Elite runners need a combination of appropriate muscle composition, efficient cardiovascular and respiratory systems, strong tendons and ligaments, a suitable physique, and an optimal response to training. Each of these factors has a large genetic component. Understanding the genetic basis of elite performance in particular sports could direct aspiring athletes to appropriate sports at an early age, and identify those who would respond best to expensive training programmes and contribute most to team sports. This knowledge could also steer individuals away from activities likely to cause them injury or frustration. To date, genetic analyses of elite athletes have identified specific alleles of two genes (*ACE* and *ACTN3*) that are associated with prowess in power sports such as sprinting.

Unfortunately, possession of these alleles provides no guarantee of excelling in any sport because there are too many other interacting variables. Nonetheless, companies still lure people to pay for DNA testing with enticements such as 'Want to improve your athletic performance? Harness the power of your genetics!' For now, the winning combinations in the genetic lottery are only revealed when aspiring athletes work hard and long at the sports that come most naturally to them.

in which training involved running, both of these fibre types contracted significantly more powerfully after training, despite their reduced diameter.

Elite sprinters, in contrast, do more resistance training. They might spend 20 hours each week training for runs that last less than a minute. These workouts target fast fibres, exerting them against the resistance provided by weights, including body weight. Resistance training includes rapid, forceful moves such as lunges, skipping high, and skipping backwards. Upper body weight training is also important. Powerful arm pumping helps propel sprinters off the blocks and strong core muscles lift them smoothly from their low starting position. Resistance training causes all muscle fibre types to enlarge, although fast fibres enlarge the most.

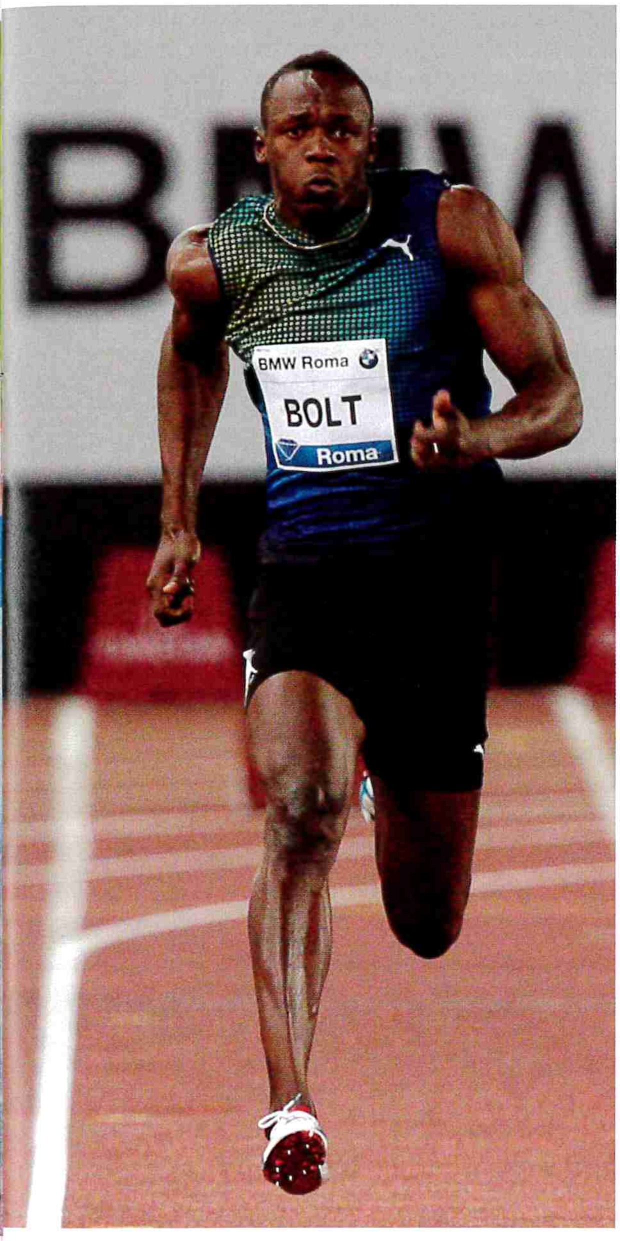

Testosterone in males increases muscle mass

Typical musculature of a long distance runner (left) and a sprinter (right)

Muscle fibres slightly damaged by training release a variety of chemicals. These chemicals stimulate tiny, adjacent cells, called satellite cells, to proliferate and merge with muscle fibres, particularly fast muscle fibres, making them larger. Within these enlarged fibres, new myofibrils are produced and expanded with more contractile proteins, increasing muscle fibre diameter and strength. Fast fibres are enlarged further by more stored glycogen and glycolytic enzymes. The fast-intermediate fibres also produce more mitochondria and myoglobin, supporting aerobic respiration as a source of more ATP.

Why do muscles get sore?

If you are serious about endurance sports or bulking up your muscles, you must 'feel the burn'. Contrary to popular belief, the pain experienced during intense exercise is not due to build-up of lactate. Instead, very rapid ATP hydrolysis in your fast glycolytic muscle fibres produces proton (H^+) concentrations that exceed the buffering capacity



of the cells. The pain will limit the weight you can lift or abruptly halt your run, while your panting restores enough oxygen for your muscles to resume respiration.

Although you may feel fine directly after a new or unusually challenging workout, 1–3 days later you will likely experience soreness. Your training will have damaged muscle fibres and their surrounding connective tissue. The result is swelling and inflammation, which hurts. But the damage also triggers complex biochemical changes that help muscles adapt and strengthen as they recover.

BiologicalSciencesReview*Extras*

Go online for a worksheet on muscles: www.hoddereducation.co.uk/bioreviewextras

Further reading

An explanation of factors that contribute to delayed muscle soreness: **https://tinyurl.com/yajqj6ob**

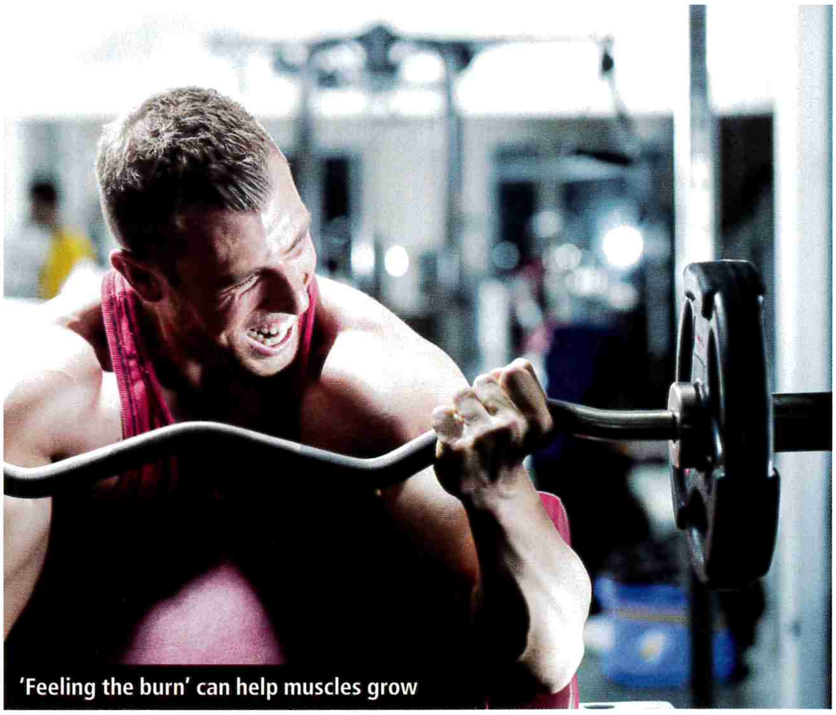
A ground-breaking transgender cyclist: **https://tinyurl.com/yd2naj97**

Why are the fastest world records held by men?

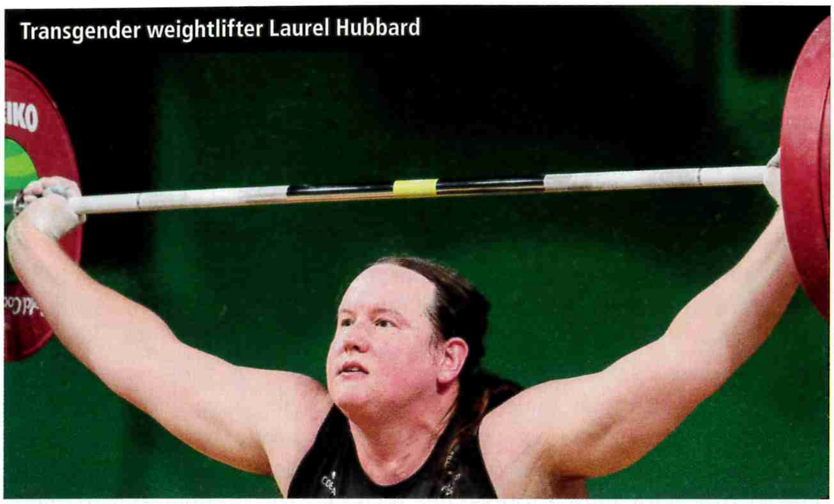
Although elite female athletes can outperform most fit men in their chosen sports, world records are essentially all held by males. On average, the gap between the genders in their sports records is around 10%. Scientists have uncovered several contributing factors but the most important is testosterone. Although male and female muscles have about the same number of fibres, the dramatic increase in testosterone that starts at puberty in males stimulates muscle fibre growth. Testosterone activates satellite cells to fuse with muscle fibres and increases the number and diameter of myofibrils, swelling muscle mass rather like resistance training does. Testosterone also reduces fat storage, so larger muscles carrying less fat increase the male advantage. Testosterone stimulates an increase in the concentration of haemoglobin in blood, which delivers oxygen to muscles. Not surprisingly, the synthetic steroids often abused by competitive athletes are close chemical relatives of testosterone.

'Biological Male Dominates Women's Cycling Competition'

This headline highlights the intense controversy surrounding a small number of elite transgender athletes who have transitioned from male to female. Does an individual whose muscles matured under the influence of testosterone have an unfair advantage in women's sports? There is no hard scientific evidence to support this. In a study of 19 individuals transitioning from male to female, a year of testosterone suppression resulted in haemoglobin concentrations typical of biological females, along with a significant reduction in muscle mass. But data on the actual performance of transgender athletes are extremely limited. A study of eight runners who competed as both male and female — that is, before and after transition — recorded slower times as females. An elite male cyclist experienced a performance decline of 11.4% as a transgender female,



'Feeling the burn' can help muscles grow


Transgender weightlifter Laurel Hubbard

matching the typical gender gap. She failed to finish in the top 30 women in a recent professional race. Weightlifters show the largest advantage following male to female transition. In 2017, a transgender female weightlifter, Laurel Hubbard, won gold at the Australasian Championships and two silvers at the world weightlifting championships.

Since 2016, the International Olympic Committee requires that transgender female competitors maintain their testosterone at or below a specified level for at least a year. Two British transgender females who met this criterion and qualified for the Olympic Games would not reveal their sports or identities for fear of public backlash. The controversy continues.

Teresa Audesirk is a retired professor of biology from the University of Colorado at Denver, where she researched and taught neurobiology. She is a co-author of the introductory college textbook *Biology: Life on Earth*, now in its 11th edition.

Key points

- Fast and slow skeletal muscle fibres are adapted for bursts of speed (fast) or slower prolonged activity (slow).
- Genes endow elite sprinters with more fast fibres, and elite marathon runners with more slow fibres, in their calf muscles.
- Training causes minor damage to muscles that stimulates them to adapt to the specific exercise involved.
- Testosterone provides an advantage to athletes. Testosterone suppression in transgender female athletes may eliminate this advantage.

Antibody protein structure

Recently, two A-level biology students asked me to settle an argument about whether an antibody molecule had a tertiary protein structure or a quaternary protein structure.

My initial reaction was pleasure that they had remembered that an antibody is a protein molecule. I was even more pleased when they correctly added that:

- antibodies of only one specific type are secreted by a plasma cell
- a plasma cell is one of a clone of cells produced by the mitotic divisions of a single activated B lymphocyte

We then explored their question using Figures 1 and 2.

Figure 1 is a diagrammatic representation of an antibody molecule. It shows that an antibody molecule comprises four polypeptides held together by disulfide bridges. Two of the chains are long chains (termed 'heavy' because they have a molecular mass of about 150 kDa) and two are short (termed 'light' because their molecular mass is about 25 kDa).

Since it is a chain of amino acids linked together by peptide bonds, each polypeptide shows the **primary protein structure** of the antibody molecule. The two variable regions that form the sites at which the antibody binds to its complementary antigen show the **tertiary protein structure** of the antibody molecule. Finally, the presence of four polypeptide chains shows the **quaternary protein structure** of the antibody molecule.

'But what about secondary protein structure?', one student asked. This is not shown by the simple diagram in Figure 1. The computer model shown in Figure 2 shows that the polypeptides are themselves coiled — this is the **secondary protein structure** of the antibody molecule.

So, an antibody molecule has a primary, secondary, tertiary and quaternary structure.

Martin Rowland

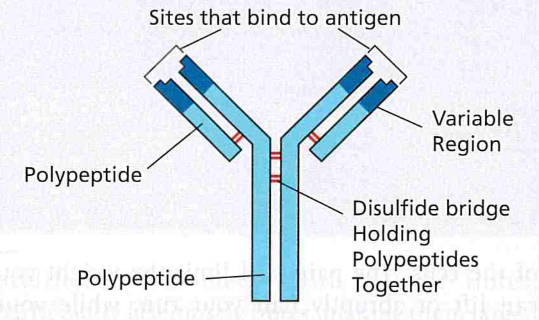


Figure 1 An antibody molecule

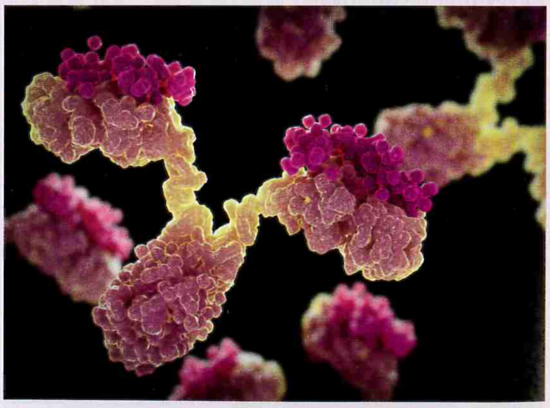


Figure 2 A computer model of an antibody molecule attached to two antigen molecules (pink), forming an antigen–antibody complex (×300 0000)