

TOPIC 7: RUN FOR YOUR LIFE

For the Edexcel Biology A Level (SNAB)

TOPICS COVERED

- Muscles and Joints
- Sliding Filament Theory
- Aerobic Respiration: Glycolysis, Link Reaction, Krebs Cycle and Electron Transport Chain
- Anaerobic Respiration and Fatigue
- Instant Energy and Balancing the Three Energy Systems
- Aerobic Capacity
- Control of Heart Rate
- Electrocardiograms
- Breathing
- Homeostasis
- Temperature Control
- Excessive Exercise and Immune Suppression
- Keyhole Surgery and Prosthesis
- Performance Enhancement



Muscles and Movement

Key Terminology

Term	Definition
Flexion	Muscles contract and relax to bend
Extension	Muscles straighten
Antagonistic	The way in which muscles work in pairs
Agonist / Flexor	Muscle which contracts to cause flexion
Antagonist / Extensor	Muscle which contracts to cause extension
Ligaments	Tough, flexible fibrous connective tissue which connects two bones
Joints	Where two bones connect and muscles bring about movement
Cartilage	Firm tissue which acts as a shock absorber at joints
Multinucleate	Multiple nuclei within a single cell due to the large size
Myofibril	Elongated contractile threads found in striated muscle cells
Sarcomere	Structural unit of a myofibril
Sarcoplasmic Reticulum	The endoplasmic reticulum of the sarcomere which releases calcium ions
Sarcoplasm	The cytoplasm of the sarcomere

Joints and Movement

Bones meet at joints – they are separated by a cavity filled with synovial fluid, enabling them to move as restricted by the ligaments. Tendons attach muscles to bones, and cartilage protects bones within joints.

Muscles work at a joint to control movement, working in antagonistic pairs as they can only pull. Muscles shorten, pulling on the bone and so moving the joint. A muscle that contracts to cause extension of a joint is called an extensor, while the corresponding flexor muscle contracts to bend a joint, reversing the movement.



The bones of the lower arm are attached to a biceps and triceps muscle by tendons; the bones themselves are attached by ligaments. The biceps is the agonist

muscle, which contracts to bend the arm as the bone is pulled, and the antagonist muscle, the triceps, contracts in order for extension, whilst the biceps relaxes.

Muscle Structure

Muscle is made up of bundles of muscle fibres, up to 2cm across. These are bound by connective tissue, continuous within the tendons. Each muscle fibre is a single long, multinucleated cell, up to several cm long but less than 0.1nm in diameter. The cytoplasm of muscle cells, the sarcoplasm, contains a specialised endoplasmic reticulum called the sarcoplasmic reticulum, which stores calcium. Each muscle cell contains myofibrils, each composed of contractile units called sarcomeres.

The sarcomere is made of two protein filaments, thin ones made of actin and thicker ones made of myosin. Contractions are brought about by sliding of these filaments. The proteins overlap to give the fibre its characteristic striated appearance. When muscle contracts, the actin moves between the myosin, shortening the length of the sarcomere and the muscle.

Sliding Filament Theory

• When a nerve impulse arrives at a neuromuscular junction, Ca²⁺ ions are released from the sarcoplasmic reticulum, before diffusing through the sarcoplasm to initiate movement of protein filaments

- Ca²⁺ attaches to troponin, causing it to move. Tropomyosin on the actin filament shifts, exposing myosin binding sites. Myosin heads bind with the exposed sites, forming cross-bridges
- When the myosin head binds to the actin, ADP and P_i on the myosin head are released as ATP is hydrolysed. The myosin changes shape, causing the myosin head to dip forward, resulting in actin moving over the myosin
- ATP binds to the myosin head, causing the myosin to detach from the actin. ATPase on the head hydrolyses the ATP, and the myosin head returns to its original position
- Ca²⁺ moves back into the sarcoplasmic reticulum by active transport, and my troponin and tropomyosin move back, blocking the binding sites

Respiration and Energy Systems



Term	Definition
Aerobic Respiration	Complete oxidation of glucose or other chemical stores in a series of enzyme-controlled
	reactions to form carbon dioxide and to transfer energy to ATP
Basal Metabolic Rate	BMR is the minimum energy requirement for a body to fuel basic metabolic processes
ATP	Adenosine Triphosphate – the energy currency of biology
Pyruvate	The 3C product of glycolysis
Coenzyme	Non-protein compounds which assist in enzyme-controlled reactions – in respiration, these are NAD and FAD
Glycolysis	The initial stage of carbohydrate breakdown, involving splitting of glucose
Link Reaction	The first step of aerobic respiration, where pyruvate is used to make acetyl (2C) CoA
Citric Acid Cycle	Series of decarboxylation, oxidation and phosphorylation reactions which reforms 4C
Electron Transport Chain	Final stage of aerobic respiration, where reduced coenzymes lose hydrogen ions and
	electrons, which are transported by chemiosmisis and
Phosphorylation	Addition of an inorganic phosphate group to ADP
Chemiosmosis	Diffusion of H ⁺ into the matrix down its electrochemical gradient, allowing ATP synthase to catalyse ATP synthesis
Oxygen Debt	After a period of exercise, there is excess oxygen requirement to remove lactate produced by anaerobic respiration
Creatine Phosphate	An energy store in muscles which can be rapidly hydrolysed for a short burst of exercise

BMR is the minimum energy requirement to fuel basic processes; it with age, gender and level of physical activity. Food is the source of energy for all animal activity, the main sources being fats and carbohydrates. Respiration is a series of enzyme-controlled reactions which produces ATP, the energy currency of biology; the overall equation for aerobic respiration is $C_6H_{12}O_6 + 6O_2 + 38ADP + 38P_i \rightarrow 6CO_2 + 6H_2O + 38ATP$. ATP is created by phosphorylation of ADP, and hydrolysis of ATP releases chemical potential energy.



glycogen store

The first stage of carbohydrate breakdown is glycolysis. This occurs in the cytoplasm, or the sarcoplasm of muscle cells. After the store of glucose, such as glycogen, is hydrolysed, an input of 2ATP is required. After this, the glucose can be split into 2 phosphorylated 3C intermediates. These are then oxidised to produce 3C pyruvate. Two hydrogens are removed during the reaction and taken up by the coenzyme NAD⁺ to form the reduced form NADH.

Glucose is at a higher energy level than the pyruvate, meaning this reaction is exothermic; the energy released is then used to direct creation of ATP. Phosphate from the intermediate is transferred directly to ADP, forming ATP by substrate-level phosphorylation.

Overall, glycolysis has a net gain of 2ATP, 2NADH and 2 pyruvates.



myosin binding sites





Aerobic Respiration

The pyruvate molecules pass into the mitochondria for the next stage, the link reaction. First, pyruvate is decarboxylated, losing a CO_2 group, and then dehydrogenated, losing 2 H atoms to NAD⁺. The resulting 2C molecule is used to from acetyl CoA. The coenzyme CoA is carried into the Citric Acid, or Krebs, cycle, which occurs in the mitochondrial matrix.

- Each CoA combines with a 4C compound to create 6C
- 6C is decarboxylated, releasing CO₂, and oxidised, releasing 2H to produce NADH
- Resulting 5C compound is also decarboxylated, then oxidised to remove 6H, producing 2NADH and 1FADH₂
- The decarboxylation of 5C also yields 1 ATP directly, by substrate-level phosphorylation

The reduced coenzymes NADH and FADH₂ transport H atoms to the mitochondrial inner membrane for the electron transport chain. Each hydrogen atom's proton and electron then separate during the electron transport chain:

- High-energy electrons are passed along the electron transport chain through protein carriers by a series of redox reaction; the carrier is reduced when accepting the electron then oxidised when it passes the particle on
- As these electrons move down the ETC, they release energy. This energy is used to pump protons across the membrane from the matrix to the intermembrane space. This establishes a steep electrochemical gradient with a high proton concentration in the intermembrane space and a low concentration in the matrix
- H⁺ flows through ATP synthase back to the matrix, by chemiosmosis. This is a proton-motive force



• Energy released by H⁺ movement through the ATP synthase allows oxidative phosphorylation of ADP, forming ATP

• Oxygen is the final electron acceptor; electrons and protons recombine to form H atoms which then react with O_2 to form water. If supply of O_2 stops, the ETC and ATP synthesis ceases

Since respiration is an enzyme-controlled reaction, rate is affected by enzyme and substrate concentrations, temperature and pH. ATP can also control reaction, as ATP presence can inhibit the enzyme in the first step of glycolysis, meaning respiration is stopped as the process cannot being – this is end point inhibition

Anaerobic Respiration

In the absence of oxygen, the electron transport chain ceases, and ² most respiration reactions cannot continue. However, in the cytoplasm, it is possible to oxidise the NADH formed during glycolysis. The pyruvate produced is reduced to lactate, oxidising the NADH to regenerate NAD⁺, allowing 2ATP to be produced.



The end product of lactate must be disposed of, as in the aqueous conditions of the cytoplasm, lactic acid is formed. pH falls, inhibiting the enzymes that catalyse glycolysis, meaning activity cannot continue, as protons from the lactic acid accumulate and neutralise the -ve groups on the active site, removing the attraction between charged groups on the substrate and on the active site.

After a period of anaerobic respiration, most of the lactate is reoxidsed to pyruvate or converted to glycogen in the liver. This is the cause of oxygen-debt after exercise.





Instant Energy and the Three Energy Systems PAPERS PRACTICE

At the start of exercise, the immediate regeneration of ATP is achieved by rapid hydrolysis of creatine phosphate in the ATP/PC system; creatine phosphate + ADP \rightarrow creatine + ATP. This reaction provides energy for roughly 6-10s of intense exercise. Later, creatine phosphate stores can be regenerated from ATP when the body is at rest.

Exercise involves all three energy systems: aerobic and anaerobic respiration, and the ATP/PC reaction. At the start of any exercise, aerobic respiration cannot meet the demands for energy because the supply of oxygen to the muscles from circulation is insufficient, meaning ATP will be regenerated without using oxygen, first by the ATP/PC system, then anaerobic respiration. In endurance-type exercise, an increase in blood supply to the muscles ensures higher oxygen supply; aerobic respiration can regenerate ATP as quickly as it is broken down.

Performance Control

Key Terminology and Equations

Term	Definition		
Aerobic Capacity	The ability to take in, transport and use oxygen		
Cardiac Output	Volume of blood pumped by the heart in a minute		
Electrocardiogram	ECGs are graphical records of electrical activity during the cardiac cycle		
Myogenic	Able to contract without external nervous stimulation		
Autonomic Nervous System	Regulates the internal environment by controlling smooth and cardiac muscle and the		
	organs of the cardiovascular, excretory and endocrine systems		
Sympathetic Division	Division Fight or flight control, preparing for high-energy activity		
Parasympathetic Division	Rest and digest control, conserving energy stores and enhancing digestion		
Negative Feedback	Counteraction of an effect by its own influence on the process giving rise to it, as when a		
	high level of a particular hormone in blood may inhibit further secretion of that hormone		
Tidal Volume	Volume of air breathed in and out in one breath		
Vital Capacity	Maximum volume of air that can be inhaled and exhaled		
Minute Ventilation	Volume of air taken into the lungs in one minute		
Myoglobin	A protein which acts as an oxygen store in slow twitch fibres, with a high affinity for oxygen meaning oxygen is only released when levels fall very low		

Cardiac Output = Stroke Volume × Heart Rate

Minute Ventilation = Tidal Volume × Breathing Rate

Aerobic capacity is the ability to take in, transport and use oxygen. At rest, an average person consumes 0.2-0.3 litres of oxygen, known as VO₂. This increased to 3-6 litres during maximum aerobic exercise, known as VO₂(max), measured in ml min⁻¹ kg⁻¹. VO₂(max) depends on the efficiency of uptake and delivery of oxygen by the respiratory and circulatory systems, and the efficiency of O₂ use by muscle fibres. VO₂ is much higher as, during exercise, cardiac output is higher, breathing rate is higher, and breathing is deeper.

Cardiac Output and Control of Heart Rate

During exercise, more blood is travels back to the heart in venous return. During diastole, the heart therefore fills with a larger volume of blood and the cardiac muscle is stretched, causing stronger contraction, meaning stroke volume *and therefore cardiac output* is much higher.

Cardiac muscle is myogenic, meaning it can contract without external nervous stimulation: this means that stimulations from within the heart cause depolarisation.

- 1. Depolarisation starts at the sinoatrial node (SAN), a small area of fibre near the opening of the vena cava.
- 2. Impulses from the SAN spread across the atria walls, causing contraction, before continuing to the atrioventricular node (AVN).
- 3. Impulses pause for 0.13s to allow for atrial systole to be completed before passing down the Purkyne fibres to the heart apex. These are large muscle fibres that together form the bundle of His
- 4. Impulses spread up through the ventricle walls causing contraction from the apex upwards, squeezing blood into the arteries





Electrocardiograms record electrical activity during the cardiac cycle. In an ECG, electrodes are attached to the chest and limbs, and a small current can be detected at the skin's surface.



ECG Segment	Description		
P Wave	Atrial depolarisation, leading to atrial systole		
PQ	Atrial Systole		
PR Interval	Time taken for impulses to be conducted from SAN to the		
	ventricles via the AVN		
QRS Complex	Ventricular waves of depolarisation, leading to contraction		
ST	Ventricular Systole		
T Wave	Repolarisation of ventricles during diastole		

To calculate heart rate, divide 300 by the number of large squares per beat.

Syr	npathetic Stimulation
•	Higher cardiac output

- Bronchi dilate
- Breathing rate increased
- Digestion inhibited
- Hydrolysis of glycogen
- Adrenaline secreted

If heart rate <60 a person has bradycardia, common in fit athletes but a potential symptom of heart problems like hypothermia, heart disease or drugs. If heart rate exceeds 100, a person is tachycardic, often a result of anxiety, fever or exercise. It may also be a symptom of CHD or fluid loss.

During ischaemia, heart tissue is deprived of oxygen due to blockage of coronary arteries. Arrhythmias can be detected in ECG abnormalities, and traces also show areas of damage and inadequate blood flow.

Decreased breathing rate

Decreased cardiac output

Enhanced digestion

Nervous Control

The autonomic nervous system regulates the internal environment by controlling smooth and cardiac muscle and the organs of the cardiovascular, excretory and endocrine systems. It has two divisions: sympathetic (fight or flight), and parasympathetic (rest and digest).

Heart rate is controlled in a negative feedback loop:

•



Adrenaline is secreted by the adrenal glands. It has a direct effect on the SAN, increasing heart rate and contractility. Adrenaline also causes dilation of arterioles supplying skeletal muscles, and constriction of arteries to non-essential organs, maximising blood flow to muscles.

Breathing

Tidal volume is the volume of air breathed in and out in one breath, and the maximum volume of air we can inhale and exhale is our vital capacity. Minute ventilation (dm³ min⁻¹) is the volume of air taken into the lungs in one minute, found by multiplying the tidal volume by breathing rate. These lung volumes can be measured using a spirometer (CP17).





A spirometer trace allows calculation of all the volumes and rates above, as shown to the right. To calculate breathing rate, find the average length of a breath, then scale up to 60s. Oxygen consumption is found by finding the change in volume between two troughs of tidal volume measurements e.g. between A and B.

The ventilation centre in the medulla oblongata controls breathing. The centre sends impulses every 2-3 seconds to the external intercostal muscles and diaphragm muscles, which contract to cause inhalation as pressure in the lungs drops. During deep inhalation, neck and upper chest muscles are also used. As the lungs inflate, stretch receptors in the bronchioles are stimulated, sending inhibitory impulses to the ventilation centre, stopping impulses to the muscles. As the diaphragm and intercostals relax, exhalation is allowed. Exhalation is caused by the elastic recoil of the lungs and the lowering of the ribs. Internal intercostal muscles only contract during deep exhalation.

At rest, the most important stimulus controlling breathing rate and depth is concentration of CO₂ in arterial blood via pH. A small decrease in pH leads to a large increase in ventilation. Faster and deeper breathing maintains the steep concentration gradient between alveolar air and the bloodstream. This is also negative feedback control.



Muscle Fibres

There are two types of muscle fibre: slow twitch, darker muscle specialised for slower, sustained contraction and long period of exercise; and fast twitch, lighter muscle specialised for rapid, intense contractions with ATP produced almost entirely from anaerobic glycolysis. Slow twitch fibres contain much myoglobin, an oxygen store as the dark-red protein has a high O₂ affinity, meaning they can respire aerobically for long periods of time. Fast twitch fibres, however, have rapid lactate build-up as they respire anaerobically, meaning they fatigue quickly. With aerobic training, fast twitch fibres take on some slow twitch characteristics, for example increased mitochondria frequency.

	Respire	Colour	Mitochondria	Sarcoplasmic Reticulum	Myoglobin & Glycogen	Fatigue	Capillaries
Slow Twitch	Aerobic	Red	Many	Little	High	Resistant	Many
Fast Twitch	Anaerobic	White	Few	Extensive	Low	Quickly	Few
Homeostasis Key Terminology							

Homeostasis

Term	Definition
Homeostasis	Maintenance of a stable internal environment
Norm Value	The value at which a condition is controlled, usually the optimum
Negative Feedback	A deviation from the norm results in a change in the opposite direction, back to the norm;
	this means that the actual value fluctuated in a narrow range around the norm
Positive Feedback	The output from a control centre moves a change in a condition further from the norm
Thermoregulation	Control of body temperature at 37.5°c

Feedback Control Mechanisms

In order for cells to function properly, internal body conditions must be maintained within a narrow range by homeostasis. By controlling blood glucose, ion and CO₂ concentrations in addition to water potential, pH and temperature, in turn tissue fluid and therefore cell environment conditions are also controlled

Negative feedback is where a deviation from the norm results in a change in the opposite direction, back to the norm; this means that the actual value fluctuated in a narrow range around the norm. When a change from the norm is detected by receptors, effectors act to return the condition to the set point. Examples include control of heart and ventilation rates, control of enzyme activity by end-point inhibition (e.g. ATP binds to an enzyme involved in glycolysis and inhibits its activity), control of hormone levels, and control of population size (predation and competition limit size).

Positive feedback is where output from a control centre moves a change in a condition further from the norm. Examples include increasing oxytocin levels during childbirth to increase uterus contraction rate, and also coagulation.

Temperature Control

Temperature is controlled at 37.5°c by cyclical and dynamic thermoregulatory mechanisms in order for enzymecatalysed reactions to occur at a reasonable rate.

Temperature is controlled by the hypothalamus, which receives impulses from receptors in the skin, and then causes effectors to bring about a change.

				keratinised layer		ope	ning of	
Boo	dy Temperature	High Body Temperature		(cells dead)	capillaries	swe	at duct	
•	Vasoconstriction: constriction of arterioles and capillaries near the skin and dilation of the shunt vessel means blood flow is diverted away from the skin	 Vasodilation: relaxing of arterioles and capillaries near the skin and constriction of the shunt vessel means blood flows 		epidermis	\square	57	F	temperature receptor
•	meaning less energy is lost Erector muscles contract and behavioural changes cause trapping warm air and reducing	 close to the surface and energy is lost be radiation, convection and conduction Sweat glands secrete sweat, 		dermis		The second secon		
•	energy loss by convection Liver stimulated to raise	which is released via the sweat duct. Sweat						—— sweat gland
•	metabolic rate Skeletal muscles rapidly contract and relax to release energy through shivering	 evaporates and transfers energy to the surroundings Liver inhibited which reduces metabolic rate 		1		hypodermis	5/-	subcutaneous fat
			1	vein artery shu	unt ef	ifector	sensory	

Overdoing It

Key Terminology

Term	Definition
Natural Killer Cells	A type of lymphocyte found in the blood and lymph which provide non-specific immunity against
	cells invaded by viruses and cancerous cells
Keyhole Surgery	Minimally invasive surgery carried out through a very small incision using fibre optics and minute
	video cameras. Arthroscopy is keyhole surgery on joints
Prosthesis	Artificial body part used by someone with a disability to allow them to regain some normal function

Excessive Exercise and Immune Suppression 70/2 (1997)

Athletes engaged in heavy training programmes see more prone to infection than normal. There is a U-shaped relationship between risk of infection and amount of exercise. Increased exposure to pathogens and suppressed immunity with hard exercise may increase infection rates.

Moderate Exercise increases number of natural killer cells, which are activated by cytokines and interferons, and appear to target cells that do not display self-markers. The killer cells release perforin, which makes pores in the targeted cell membrane, allowing other molecules such as proteases to enter and cause apoptosis.

During recovery after *vigorous exercise*, the number and activity of natural killer cells, phagocytes, B cells and T helper cells, fall. The decrease in T helper cells reduces the number of cytokines available to activate lymphocytes, in turn reducing the quantity of antibody produced. An inflammatory response may also occur in muscles due to damage to muscle fibres caused by heavy exercise, reducing the availability of non-specific immune response against respiratory infection. Intense exercise may also cause psychological stress and therefore secretion of hormones such as adrenaline and cortisol – both hormones are known to suppress the immune system.

Damage to Joints during Exercise

Professional athletes risk developing joint injuries due to high forces the sports generate on their joints. Repeated force causes wear and tear of joints. Treatments include RICE, anti-inflammatory painkillers and surgical repair. Issues with the knee include: damage to articular cartilage meaning bones grind, causing arthritis and inflammation; the kneecap does not glide smoothly across the femur due to damage of the cartilage; fluid sacs at points of contact; sudden twisting causing damage to ligaments.



Keyhole Surgery

Keyhole surgery has allowed repair of damaged joints or removal of diseased organs through small holes. During an arthroscopic procedure, a surgeon makes two small incisions before observation or surgery can occur. Damage to the anterior cruciate ligament – which usually prevents the knee bending too far back – can be tackled using keyhole surgery. The ligament can be repaired and the knee joint stabilised to reduce risk of further injury. Advantages of keyhole surgery are: no blood transfusion; small incision means less pain and shorter recovery time; reduced risk of infection. The only disadvantage is the difficult perspective and limited range of motion means there is a risk of accidental damage.

Prostheses

Prosthetics are used to help people with a disability to become more physically active. Prosthetics may also be used to replace damaged joints that have not responded to other therapy, either from injury or disease. Replacing knee joints is particularly successful: once the kneecap is moved, the ends of the femur and tibia are trimmed, then a bone cement is used to attach the prosthetic, whilst the original ligaments are returned.

Benefits of Moderate Exercise

- Increasing arterial vasodilation, lowering blood pressure and CHD and stroke risk, and HDL levels rise
- Balance between energy input and output helps to maintain weight
- Increased muscle cell sensitivity to insulin, improving glucose regulation and reducing type II diabetes risk
- Increased bone density, delayed onset of osteoporosis, reduced risk of cancer, and improved mental wellbeing

Performance Enhancement

Key Terminology

Doping

Use of drugs to enhance performance in sport

Hormones are secreted by endocrine glands, and each type only affects specific target cells after travelling in the blood.

- Peptide hormones are protein chains which bind to a receptor on the cell membrane, which activates second messengers in the cytoplasm. The functional second messenger activates enzymes or transcription factors.
- Steroid hormones are formed from lipids and have complex ring structures. These pass through the membrane and bind to receptors in the cytoplasm. The activated hormone-receptor complex acts as a transcription factor.

Transcription is initiated by a complex of RNA polymerase and protein transcription factors binding to DNA at the promotor region. Some transcription factors are present in cells; others are synthesised only at certain stages. Most are only active in the presence of regulatory molecules. The gene remains switched off until all transcription factors are active. Transcription can be prevented if a repressor molecule, e.g. an inactive factor, is attached to the promotor.

Doping (defined above) using EPO and testosterone is banned by the World Anti-Doping Agency.

Hormone	Effect	Risks
Erythropoietin	Peptide hormone produced by the kidney, which stimulates the formation of new red blood cells in bone marrow, increase oxygen-carrying capacity. It can be difficult to detect whether high EPO levels are natural or not	High risk of thrombosis – possible heart attacks or strokes
Testosterone	Steroid hormone which causes development of secondary sexua characteristics, and also increases muscle size and strength. Injections help increase muscle development. Anabolic steroids are produced from modification of testosterone but can be detected by mass spec of urine	High blood pressure Liver damage and kidney failure Increased adrenaline
Creatine	Creatine is legal, and is absorbed and transported to muscle. Higher CF storage would allow improved performance during repeated rapid high intensity exercise. Using creatine with weights sees increased muscle mass	Nausea and vomiting High blood pressure Kidney damage and muscle cramp

Pressure to succeed in competitive sport is ever increasing due to financial rewards and media interest; in combination with desire to succeed, some athletes are prepared to take drugs that will enhance performance.

Arguments For Doping		Arguments Against Doping		
٠	Athletes should be able to decide for themselves whether	٠	Athletes are not making an informed decision, lacking	
	to take a drug or not		information about health risks and under pressure	
•	Right to achieve the best they can	•	Unfair for the many who will not dope	
•	Already inequality from resources	•	Cheating violates the right to a fair competition	