Pearson Edexcel International Advanced Level

Thursday 4 June 2026

Morning (Time: 1 hour 45 minutes)

Paper reference **WBI15/01A**

Biology

International Advanced Level

UNIT 5: Respiration, Internal Environment, Coordination and Gene Technology

Question Paper

You must have:
Answer Book (sent separately)
Scientific article (enclosed), scientific calculator, ruler, HB pencil

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Pearson

Answer ALL questions.

1 Parkinson's disease is a neurodegenerative disease that involves the loss of neurones in parts of the brain.

1 (a) (i) Which neurotransmitter is released from these neurones?

(1)

A acetylcholine

B dopamine

C nicotine

D serotonin

1 (a) (ii) State the name of a drug that is used to increase the levels of this neurotransmitter.

(1)

1 (b) Imaging technology can be used to diagnose and track the progress of Parkinson's disease.

1 (b) (i) Which of the following uses X-rays to identify structures in the brain?

(1)

A computed tomography (CT)

B functional magnetic resonance imaging (fMRI)

C magnetic resonance imaging (MRI)

D positron emission tomography (PET)

1 (b) (ii) Which of the following pairs of scanning techniques can be used to detect active areas of the brain?

(1)

A CT and fMRI

B fMRI and MRI

C fMRI and PET

D MRI and PET

1 (c) Some other neurodegenerative conditions involve damage or loss of the myelin sheath.

Explain why this slows down the rate at which impulses are transmitted along neurones.

(2)

(Total for Question 1 = 6 marks)

2 Usher syndrome is a rare genetic condition that results in loss of hearing and loss of sight.

Individuals gradually develop night blindness and lose their peripheral vision over several years.

2 (a) There are three types of Usher syndrome.

2 (a) (i) In type I of this syndrome, individuals have severe problems with balance at birth.

Which part of the brain is involved in balance?

(1)

A cerebellum

B hypothalamus

C medulla oblongata

D pituitary gland

2 (a) (ii) In type II of this syndrome, neurones are unable to transmit impulses from the ear to the brain.

Which part of the reflex arc transmits impulses from the ear to the brain?

(1)

A motor neurone

B receptor

C relay neurone

D sensory neurone

2 (a) (iii) Type III of this syndrome is more common in Finland.

The population of Finland is 5.6 million.

In Finland, the frequency of this condition is 4 people per 100 000.

The ratio of boys to girls born in Finland is 1.06 : 1.

Calculate how many individuals in Finland have this condition and how many of these are males.

(2)

2 (b) In low light intensity, the eye adapts to maximise the amount of light entering.

Describe how this change occurs.

(2)

2 (c) Night blindness develops in individuals with Usher syndrome due to breakdown of the membranes of cells found in the retina.

Suggest why individuals with this syndrome struggle to see in dim light.

(3)

(Total for Question 2 = 9 marks)

3 Cold water swimming has been a long-standing tradition in many northern countries.

Some competitions occur in water colder than 6 °C and swimmers must be assessed before participating.

3 (a) Humans usually maintain a core body temperature between 36.5 °C and 37.5 °C.

Which term describes the process that maintains a constant core body temperature?

(1)

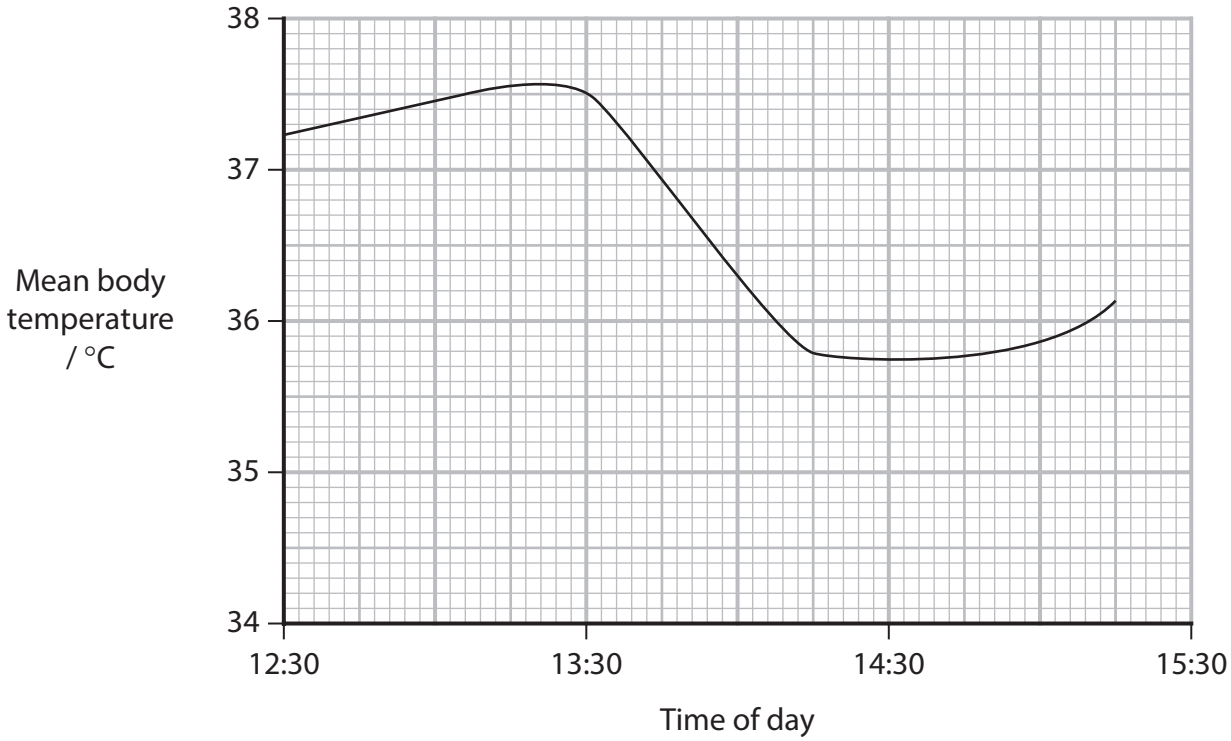
A habituation

B homeostasis

C osmoregulation

D positive feedback

3 (b) The graph shows changes in core body temperature of a swimmer recorded before, during and after swimming in cold water.

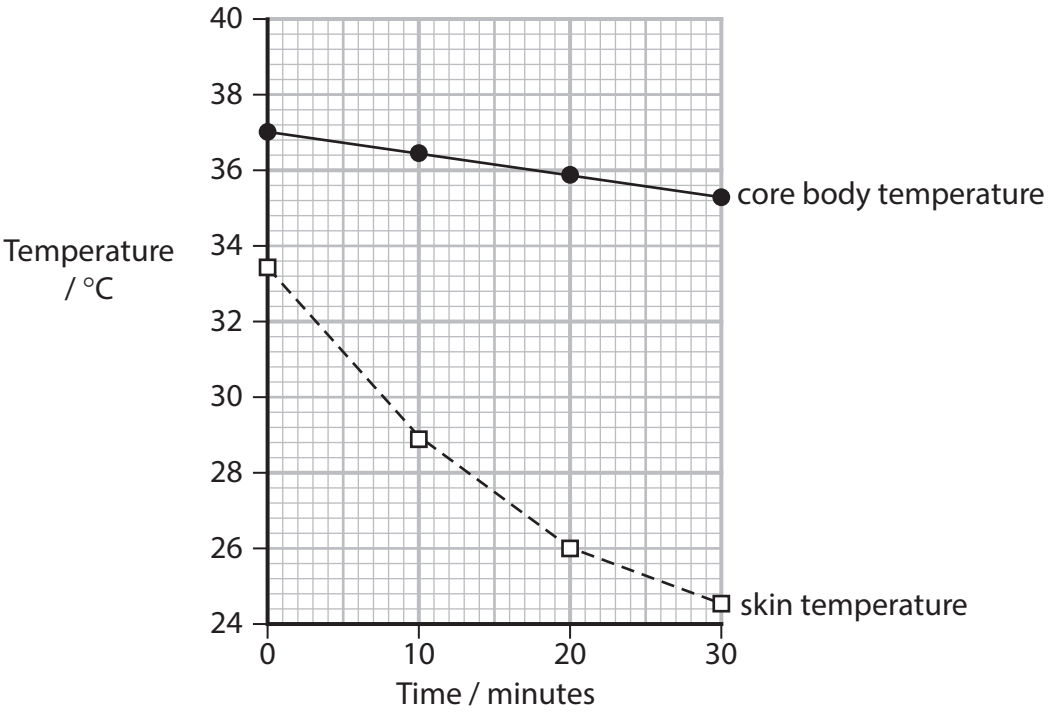


Calculate the rate of cooling of the core body temperature from 13:30 to 14:00.

Give your answer with appropriate units.

(2)

3 (c) The core body temperature and skin temperature of this swimmer were measured over a 30 minute period.



Explain the changes in the swimmer's core body temperature and skin temperature.

(4)

3 (d) The table shows the core body temperature of a student during 30 minutes of exercise in warm conditions.

Duration of exercise / minutes	Core body temperature / °C
0	37.0
5	37.3
10	37.6
15	37.9
20	38.2
25	38.4
30	38.5

Calculate the percentage increase in core body temperature from 10 to 25 minutes.

Give your answer to **two** decimal places.

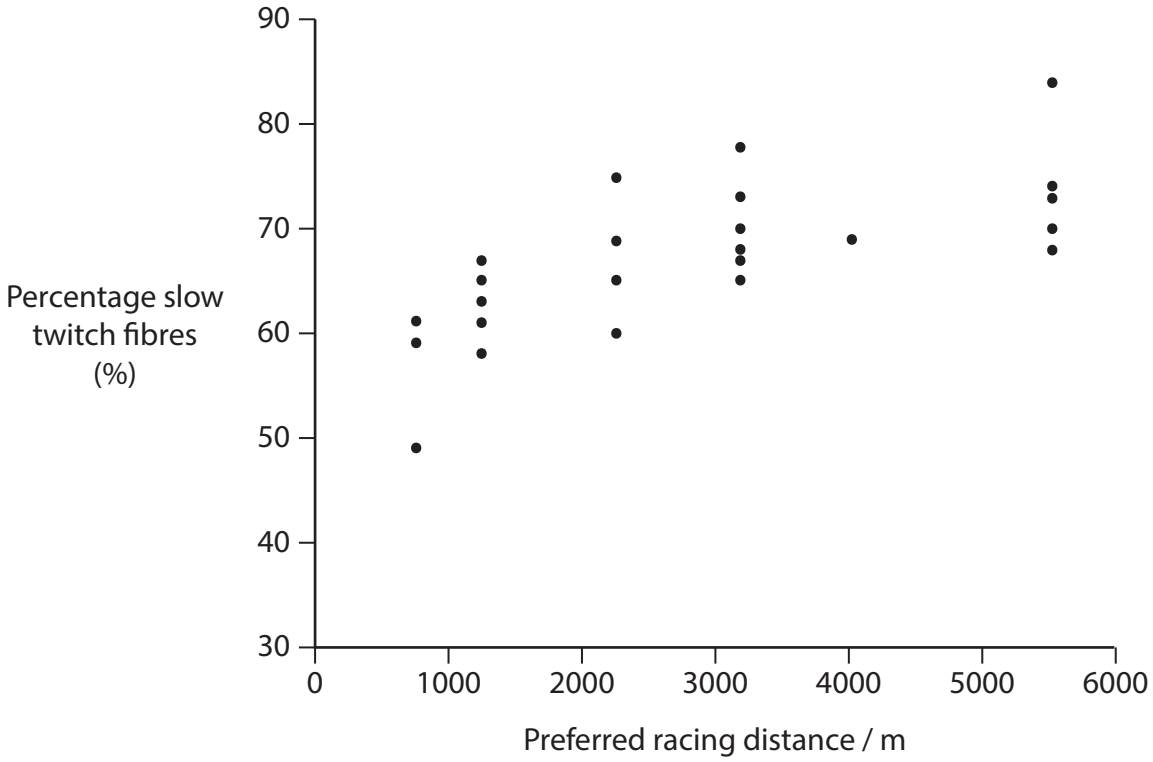
(2)

(Total for Question 3 = 9 marks)

4 The ratio of the types of fibres in muscle is connected to performance in athletes.

Speed skating includes racing distances between 500 m and 10 000 m.

4 (a) The graph shows the percentage of slow twitch fibres and the preferred racing distance of each skater.



4 (a) (i) State the relationship between the proportion of slow twitch fibres and preferred racing distance in competitive speed skaters.

(1)

4 (a) (ii) State a statistical test that could be used to analyse the data in this graph.

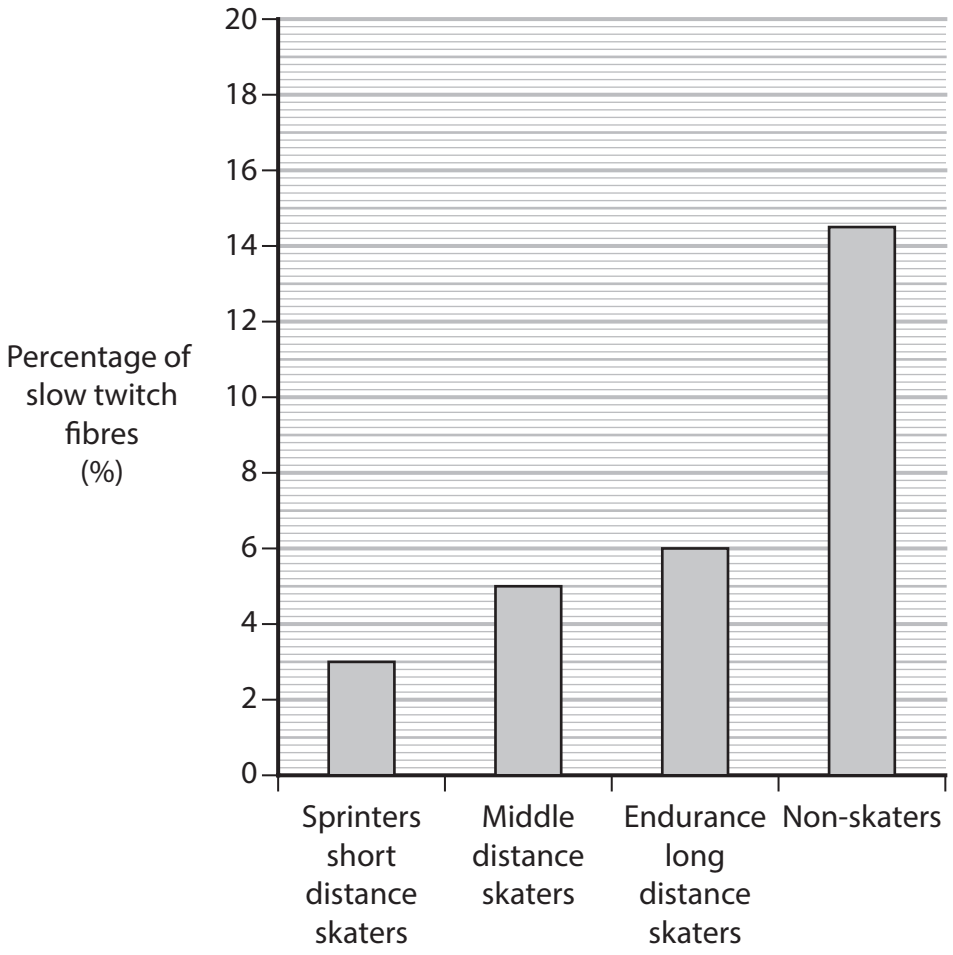
(1)

4 (b) Explain **two** structural features of slow twitch muscle fibres that would benefit the performance of long-distance speed skaters.

(2)

4 (c) One type of slow twitch muscle fibre has been analysed in skaters who race different distances.

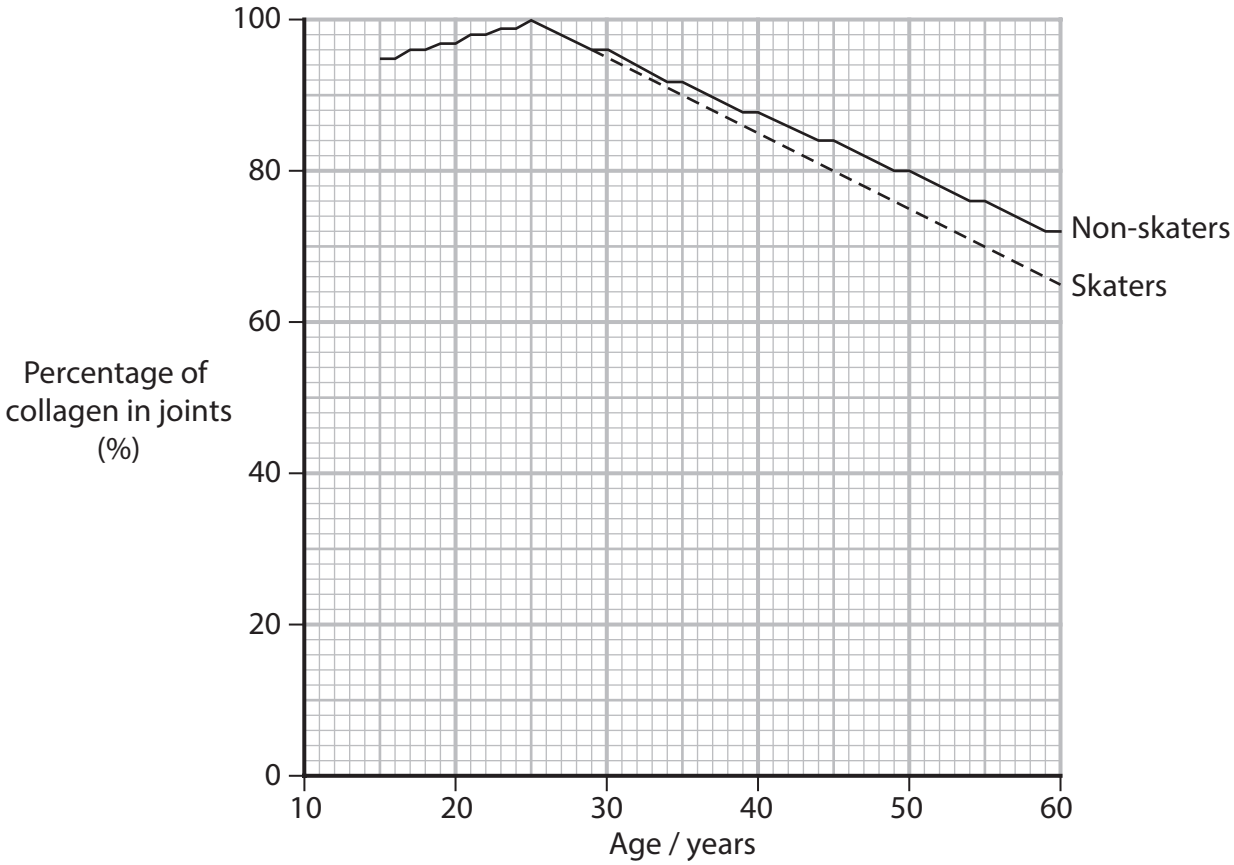
The graph shows the percentage of this type of fibre in these skaters compared with non-skaters.



Describe **two** conclusions that could be made from the data shown in this graph.

(2)

4 (d) The graph shows the percentage of collagen in the joints of skaters and non-skaters of different ages.



The recovery time from joint injury of skaters and non-skaters changes with age.

Explain why changes in the percentage of collagen in joints can affect recovery times from these injuries.

(3)

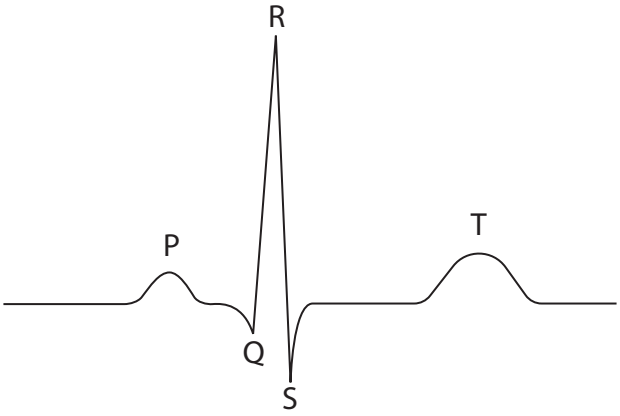
(Total for Question 4 = 9 marks)

5 The resting electrocardiograms (ECGs) of 54 male professional football players were recorded.

The ECGs were analysed to determine whether the football players had bradycardia.

Bradycardia is a heart rhythm below 60 bpm.

5 (a) The diagram shows an ECG trace.



Which part of the ECG trace corresponds to the delay at the atrioventricular node (AVN)?

(1)

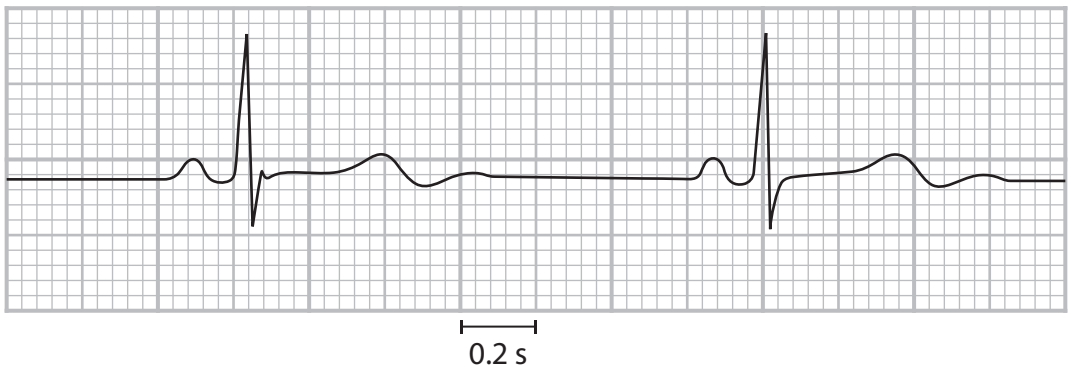
A P

B P to Q

C R to S

D T

5 (b) The ECG trace shown was taken from one of the footballers.



Determine whether or not this footballer has bradycardia.

(2)

5 (c) The table shows some of the conditions revealed by the ECGs of these 54 male footballers.

Only 19 of the footballers had a normal rhythm.

Condition	Number of footballers	Mean age in years ± SD
Normal rhythm	19	24.58 ± 4.57
Bradycardia	35	26.37 ± 5.41
Partial bundle of His block	11	25.65 ± 5.76
T wave inversion	36	25.19 ± 5.22
Total footballers	54	25.74 ± 5.16

5 (c) (i) Which of the following is the correct ratio for footballers with normal rhythm to those with bradycardia?

(1)

A 0.35 : 1

B 0.54 : 1

C 0.65 : 1

D 1.84 : 1

5 (c) (ii) A person with T wave inversions has a reduced blood flow from their left ventricle.

Explain why people with a T wave inversion have a risk of coronary heart disease.

(3)

5 (c) (iii) Discuss whether or not the information in the table about this group of footballers would be useful to other athletes.

(4)

(Total for Question 5 = 11 marks)

6 Vaccines are used to prevent diseases caused by viruses.

Crop plants can be genetically engineered to produce food containing these vaccines.

6 (a) Name the enzyme that would produce a DNA copy of a gene from an RNA molecule in a virus.

(1)

6 (b) Describe how genetic engineering could be used to produce a crop plant that contains a vaccine.

(4)

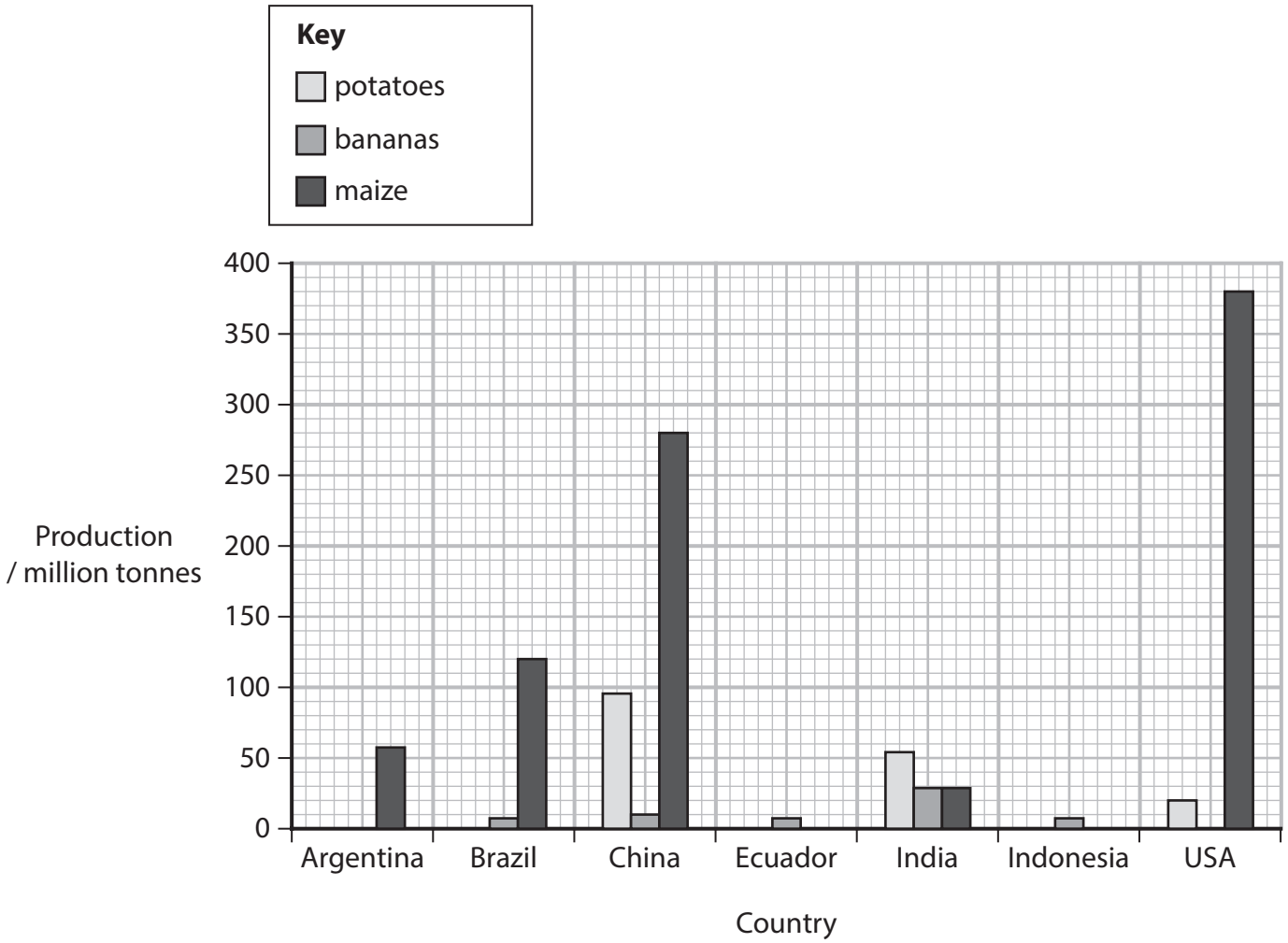
***6** (c) Vaccination programmes involve training healthcare professionals to administer vaccines, often to isolated communities.

Genetically modified potatoes, bananas and maize are three crop plants that have been investigated for their suitability for use as edible vaccines.

The table gives some information about these crop plants.

Plant	Storage	Consumption	Edible vaccines to treat
Potato	stored for 2–3 months in a cool place	needs cooking before being edible forms diet of animals and humans	Norovirus, Hepatitis B
Banana	up to 6 days without a refrigerator	no cooking needed forms diet of animals and humans	Hepatitis B
Maize	a few days or up to 2 weeks in a refrigerator	no cooking needed forms diet of animals and humans	Hepatitis B, HIV, rabies

The graph shows the distribution of these crops in some parts of the world.



Discuss the advantages and disadvantages of using these crops as vaccines.

Use the information in this question and your own knowledge to support your answer.

(6)

6 (d) Give **two** reasons why some edible vaccines may not be effective at producing immunity after the food is eaten.

(2)

(Total for Question 6 = 13 marks)

7 Cisplatin is a chemotherapy drug used to treat several types of cancer.

It can damage the lining of the proximal tubule of the kidney.

Some clinical signs of kidney damage are:

- a decrease in the filtration rate of the glomerulus
- an increase in the concentration of urea in the blood.

7 (a) (i) Which of the following is the process that removes urea from the glomerulus?

(1)

A active transport

B diffusion

C ornithine cycle

D ultrafiltration

7 (a) (ii) Explain how the structure of the glomerulus relates to its function.

(2)

7 (b) (i) Name **one** substance that is reabsorbed in the proximal tubule.

(1)

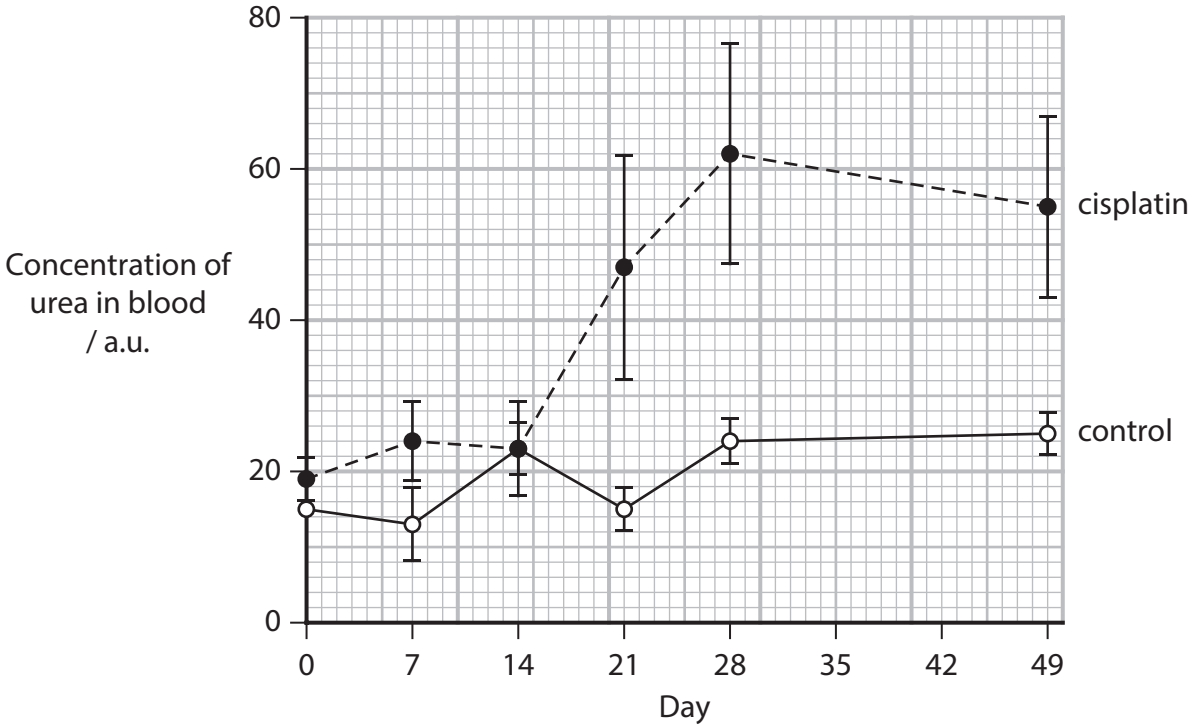
7 (b) (ii) Mice treated with cisplatin have fewer cristae in the mitochondria in the cells forming the proximal tubule.

Explain why this reduces the ability of these cells to function properly.

(3)

7 (c) The concentration of urea in the blood of mice is used to determine the level of kidney damage.

7 (c) (i) The graph shows the concentration of urea in the blood of male mice when given repeated doses of cisplatin compared with a control group of mice.



Explain how this graph shows that repeated doses of cisplatin cause kidney damage.

(3)

7 (c) (ii) Comment on the extent to which these results are useful for predicting the effects of cisplatin on the kidney in humans.

(3)

(Total for Question 7 = 13 marks)

8 The scientific document you have studied is from Scientific American entitled *New Painkiller Could Bring Relief to Millions without Addiction Risk* published 20 August 2024.

Use the information from the scientific document and your own knowledge to answer the following questions.

8 (a) There are many different types of sodium ion channels in the body.

The drug VX-548 'gums up sodium channels in peripheral nerve cells' (paragraph 1).

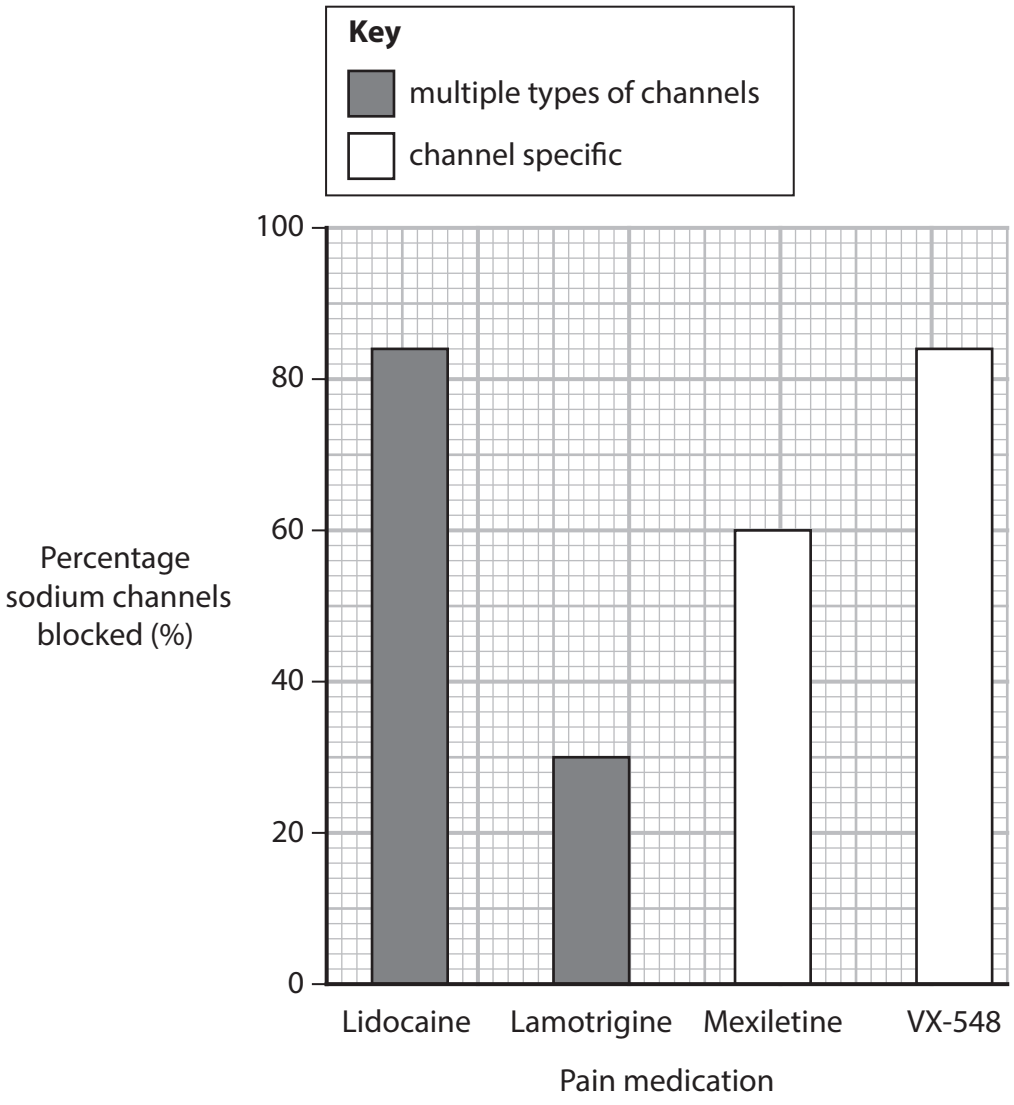
State the effect this would have on nerve cells.

(1)

8 (b) Some drugs work on more than one type of channel in nerve cells.

Other drugs work on one specific type of channel in nerve cells.

The graph shows the new drug VX-548 compared with several other existing pain medications.



Explain why VX-548 would be a more effective painkiller than the other drugs (paragraph 1).

(4)

8 (c) Depression is a common condition that may be caused by an imbalance of serotonin in the brain.

Explain how levels of serotonin can be regulated by drugs (paragraph 3).

(2)

8 (d) Suggest **two** advantages of using aspirin produced in laboratories rather than using willow bark as a pain medication (paragraph 5).

(2)

8 (e) Lidocaine is a drug used as a numbing agent but can cause serious side effects if administered systemically (paragraph 9).

Lidocaine works in a similar way to VX-548.

It can be used to reduce the number of action potentials during the cardiac cycle.

Lidocaine can be used to treat people with abnormal heartbeats.

Deduce how this drug works.

Use the information in the scientific article and your own knowledge of action potentials.

(3)

8 (f) AstraZeneca and Genentech developed painkillers that were abandoned after phase 1 trials (paragraph 11).

Suggest why drugs may fail to progress beyond this stage of the drug testing protocol.

(2)

8 (g) Suggest **two** ways that natural toxins could "disable the transmission of pain signals" (paragraph 23).

(2)

8 (h) Explain why reducing the activity of genes that encode "pain channels" has been linked to a reduction in pain in rodents (paragraph 25).

(4)

(Total for Question 8 = 20 marks)

TOTAL FOR PAPER = 90 MARKS

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Thursday 4 June 2026

Morning (Time: 1 hour 45 minutes)

Paper reference **WBI15/01A**

Biology

International Advanced Level

UNIT 5: Respiration, Internal Environment, Coordination and Gene Technology

Scientific article for use with Question 8

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Pearson

Scientific article for use with Question 8

1. There is a new drug, a pill called VX-548, that blocks pain signals before they can reach the brain. It gums up sodium channels in peripheral nerve cells, and obstructed channels make it hard for those cells to transmit pain sensations. Because the drug acts only on the peripheral nerves, it does not carry the potential for addiction associated with opioids—oxycodone (OxyContin) and similar drugs exert their effects on the brain and spinal cord and thus can trigger the brain's reward centers and an addiction cycle.

2. In January 2024 Vertex announced promising results of clinical trials of VX-548, which it is calling suzetrigine, showing that it dampened acute pain levels by about one half on a 0-to-10 pain scale. The company applied for U.S. Food and Drug Administration approval for the drug that same year. Other pain drugs that target sodium channels are now being developed, some by firms motivated by Vertex's success.

3. Therapeutics, led by biomedical engineer Ana Moreno, is even using molecular-editing tools such as CRISPR to suppress genes involved in chronic pain. "We are definitely hopeful that we can replace opioids, and that's the goal here," she says. One in five U.S. adults—51.6 million people as of 2021—is living with chronic pain. New cases arise more often than other common conditions, such as diabetes, depression and high blood pressure. Yet pain treatments have not kept pace with the need. There are over-the-counter pills such as aspirin, acetaminophen (Tylenol) and nonsteroidal anti-inflammatories (NSAIDs) such as Advil. And there are opioids.

4. VX-548 does have limits. It left some patients in significant discomfort, and so far it has been tested mostly in those with acute pain, not the much larger problem of chronic pain. But the compound has shown that a new mechanism of pain relief is possible, says Stephen Waxman, a neurologist at Yale University who studies pain signals—and who is not involved in the Vertex clinical trials. Future drugs using that mechanism are likely to be even more effective, he notes.

5. Waxman used to tell patients that a new means of managing their pain was on the way but that it may not happen for many years. "Now I can relax the caveat and say I think things are going to happen fairly quickly," he says. The pain medications that exist today are, in large part, derivatives of natural products that have been around for thousands of years. Aspirin originally came from willow bark. Morphine and codeine were derived from the opium poppy plant. Prescriptions for what evolved into the two major classes of pain drugs—NSAIDs and opioids—were etched on clay tablets by ancient Sumerians 4,000 years ago.

6. Modern research on the molecular mechanisms underlying pain, conducted during the past two decades, makes a different approach possible. Scientists know that our body is home to large numbers of pain-signaling nerve cells that innervate our skin, muscle and visceral tissues. These cells act like an alarm system, detecting threatening stimuli such as extreme temperatures, sharp objects or noxious chemicals. In response to these cues, they create impulses that carry pain signals along nerve fibers to clusters of cells known as dorsal root ganglia, which are tucked beside the spinal cord. From there, the signals continue their journey upward to the brain, where pain becomes reality. "This is the axis of pain," says Rajesh Khanna, a pharmacologist and pain researcher at the University of Florida.

7. Central to this pathway are sodium channels, cellular gates scattered throughout the membranes of nerve cells. Whenever there is a shift in membrane potential, these gates open to allow the influx of sodium ions that generate the electric currents responsible for nerve impulses. Normally those pain signals serve a protective purpose— alerting someone to pull their hand away from a hot stove or noting inflammation or injury that needs to be addressed. But in chronic pain, those protective mechanisms can go awry.

8. A voltage-gated sodium channel (or NaV, Na standing for sodium and V for voltage) seems like the ideal target for treating pain; after all, if you can stop it, you can stop pain signals from being transmitted. Yet because these channels control electrical impulses that power the heart and brain, blocking them willy-nilly would impair vital functions.

9. That's why novocaine and lidocaine—sodium channel blockers—are used as local numbing agents but can cause serious side effects if administered systemically. So scientists trying to block these pain pathways searched for channels that act more often in the peripheral sensory nerves, eventually identifying three: NaV1.7, NaV1.8 and NaV1.9. NaV1.7 and NaV1.8 are the pivotal players in pain signaling. "They work in tandem, like dominoes," Waxman says. "NaV1.7 initiates the electrical signal, and NaV1.8 takes off, producing 80 percent of the current underlying the action potential."

10. Beginning about 20 years ago, a series of reports linked these channels to pain disorders in humans. A mutation in the SCN9A gene, which encodes NaV1.7, was discovered in a family in China who suffered from a rare condition called erythromelalgia, or "man on fire" syndrome. In people with this condition, mild warmth can trigger attacks of searing pain that feels like a blowtorch. Waxman found that mutations in patients with erythromelalgia made the NaV1.7 channel overactive, causing pain-signaling neurons "to scream when they should be whispering." Elsewhere, researchers found a mutation with the opposite effect in a young Pakistani firewalker. That mutation extinguished the flow of pain-signaling ions through the NaV1.7 channel. As a result, the boy could walk on burning coals without feeling pain. The discovery of the genetic basis of his condition—known as congenital insensitivity to pain—set off a race in the pharmaceutical industry to identify molecules that could block NaV1.7.

11. The goal was to provide a similar pain-free existence to the rest of the population. "This was the holy grail. You have a protein, you mutate it, you have no pain—it's got to be the target," Khanna says. "A lot of pharma companies put a lot of money into this effort, but none of those compounds have been successful." Many compounds targeting NaV1.7 looked promising in the laboratory, only to fail in clinical trials. Pharma companies AstraZeneca and Genentech both developed candidates that stalled after phase 1 trials.

12. Pfizer's PF-05089771 failed to perform in a battery of tests evoking pain in healthy volunteers. Biogen scrapped development of its NaV1.7 inhibitor, vixotrigine, after lackluster results from a string of phase 2 trials in several types of neuropathic pain. After more than a decade of false starts, investment dwindled, and drug candidates disappeared from development pipelines.

13. One big reason for the difficulty had to do with the nature of the targets themselves. The NaV channel family contains nine closely related members that share more than 50 percent of their genetic sequence. Because of this similarity, the sodium channel inhibitors developed in the 2000s were often unable to target one subtype without hitting others. "The selectivity was terrible, frankly," says John Mulcahy, a chemist and CEO of the San Francisco based biotech firm SiteOne Therapeutics. "It's taken a long time to overcome that."

14. At Vertex, researchers believed that the compounds that had been tested before were simply not selective enough or didn't attach to a channel for enough time and that to find molecules that worked they just needed to keep searching. To speed up their hunt, they had been working on a technology that could measure the effect of massive numbers of molecules, at various concentrations, on the opening and closing of several types of sodium channels.

15. Traditionally, researchers have studied sodium channels using a laborious method called patch-clamp electrophysiology. The technique involves isolating part of a cell's membrane, applying voltage to trigger its channels to open, adding one single potential drug, and then recording the oscillating waves of electrical activity. In the early 2000s Vertex scientists Jesús González and Michael Maher designed a system called E-VIPR (for electrical stimulation voltage ion probe reader) to test many compounds against one channel very quickly. The system uses a high-density array of cells, each of which expresses one type of sodium channel. Tiny electrodes generate an electrical field that can stimulate the channels as many as 100 times per second. As these channels open, a voltage-sensitive dye shifts from orange to blue, and the color change is captured by a sophisticated optical detection tool. "It's a very quick process, faster than the human eye can detect, but incredibly rich in information," says Paul Negulescu, Vertex's senior vice president of research and head of its pain program.

16. With this method Vertex could extensively test how a potential drug interacts with a particular channel. Sodium channels undergo big shape changes as they open and close, with pieces of the protein moving up and down with dizzying speed. "It's kind of like a bucking bronco," Negulescu says. "The drug has to get on the bucking bronco and stay on while it's going through its paces and eventually settle that bucking bronco down so it stops moving." A drug candidate might land on the channel for a time, only to get kicked off once its gyrations prove too much. Or it might hop onto another channel, generating unwanted off-target effects.

17. As the industry continued to focus on NaV1.7, Vertex started to see success with NaV1.8 and pushed forward a program on the neglected channel. "I think we zigged when others zagged," Negulescu says. Vertex launched its first clinical trials of a NaV1.8 inhibitor in 2015. It wasn't effective enough, and neither were the two immediately following it. But finally one was tolerated well by a small group of patients and relieved some of their pain. That was VX-548, and it prompted the company to move ahead with bigger studies in 2022.

18. Two years later, in January 2024, Vertex announced positive results of two large, pivotal clinical trials. The researchers enrolled about 1,100 people, each of whom was undergoing bunion removal or tummy tuck surgery, operations commonly used to model acute pain. Study participants got a placebo, VX-548, or the drug combo of hydrocodone (an opioid) and acetaminophen, known as Vicodin. When measuring pain relief on the 0-to-10 pain scale, the new drug performed just as well as Vicodin without the addiction risk.

19. Both treatments reduced pain by about three points, from about a 7 to a 4. And in the people recovering from abdominal surgery, relief kicked in more quickly with VX-548 than it did for those who got Vicodin. The drug provided less relief than Vicodin for bunionectomy patients when using a different pain scale. Still, those taking VX-548 reported fewer side effects—such as nausea, constipation, headache and dizziness—than those on the placebo, indicating the treatment was generally safe. (Untreated pain in the placebo group could increase side effects because it can elevate stress levels, upsetting digestion or triggering headaches.) Studies suggest that even a 3-point drop in pain can have a meaningful impact on quality of life.

20. If approved, VX-548 could help people by offering relief that lands somewhere in between that provided by drugs such as acetaminophen, which are safe but limited in their power, and stronger opioids, which come with serious risks. It could provide relief to patients who are allergic to or who simply cannot tolerate the other drugs.

21. Vertex scientists think the drug will work for that type of agony because the mechanisms underpinning chronic and acute pain are similar. It reported positive results from a smaller efficacy and safety trial of VX-548 in diabetic peripheral neuropathy, a common type of chronic pain caused by nerve damage from high blood glucose, and plans to move forward with a phase 3 trial. In addition, the company launched a separate study testing the drug in a form of chronic lower back pain known as lumbosacral radiculopathy.

22. And Vertex researchers continue to use their drug discovery platform to evolve compounds that are more potent and more selective. "We are all about serial innovation," Negulescu says. The company already has a next-generation NaV1.8 inhibitor, VX993, moving toward phase 2 clinical trials. Others in the pain field have been watching Vertex closely and are excited by its results. "I think the great contribution that Vertex has made here generating the clinical data that they have with their program is to make people understand that, hey, this is not a hopeless thing," Harper says. He, with other investors, recently launched a company called Latigo Biotherapeutics to develop sodium channel inhibitors. Waxman says the Vertex findings were modest yet important—so important that he called VX-548 "a game changer," not because it will change clinical practice on its own but because it will transform the research pipeline. "This is going to be like the development of the statin drugs," he says. "The first statin drugs were, in retrospect, not very good. But they set the stage and really were the impetus, and the ones we have now are life-changing."

23. Only a handful of companies are openly developing pain therapeutics going after NaV1.8 or NaV1.7, which remains a viable target. More may be working in "stealth" (Merck's patent activity indicates it is dabbling in the field), and others most likely will join the effort, emboldened by Vertex's progress. Some are already designing small molecules to block the sodium channels or nearby proteins; some are modifying natural toxins to disable the transmission of pain signals; still others are using gene therapy to turn down the signal at its source.

24. Michael Oshinsky, director of the Office of Preclinical Pain Research at the National Institute of University of Arizona spin-off Regulonix is sticking with NaV1.7 as a target but is going after it differently than its predecessors. Rather than blocking the sodium channel, the company is trying to remove NaV1.7 from the cell membrane. Without the channel there will be fewer sodium ions that can cross into the cell. University of Florida's Khanna, who co-founded Regulonix and is chief scientific officer of the company, says an early version of its compound successfully affected

NaV1.7 signaling in rat, mouse and pig models of acute and chronic pain. But, he admits, "we're nowhere close to being in humans." A different approach is to take naturally occurring sodium channel blockers—such as the tetrodotoxin that makes puffer fish so lethal— and modify them to block channels predominantly found in pain-sensing neurons.

25. Finally, instead of manipulating existing pain channels, some researchers are trying to keep so many of them from forming by reducing the activity of genes that encode them. That type of gene therapy is being pursued by Moreno and her company Navega. They are working with a technology that Moreno developed during her doctoral research at the University of California, San Diego. There she used CRISPR and its older gene-editing counterpart, zinc finger proteins, to target genes that help to build NaV1.7; the result was suppression or even prevention of pain in rodents. Since launching Navega, she and her team have shown that the approach works for various kinds of pain—including neuropathic, chemotherapy induced, inflammatory, visceral and arthritic—and they are quickly advancing toward first-in-human trials.

Source:

New Painkiller Could Bring Relief to Millions—Without Addiction Risk By Marla Broadfoot edited by Josh Fischman © Scientific American, August 2024

Please check the examination details below before entering your candidate information

Candidate surname	Other names

Centre Number | Candidate Number

Pearson Edexcel International Advanced Level

Thursday 4 June 2026

Morning (Time: 1 hour 45 minutes)

Paper reference **WBI15/01A**

Biology

International Advanced Level

UNIT 5: Respiration, Internal Environment, Coordination and Gene Technology

Answer Book

You must have:
Question Paper (sent separately)
Scientific article (enclosed), scientific calculator, ruler, HB pencil

Total Marks

Instructions

- Use **black** ink or ball-point pen.
- **Fill in the boxes** at the top of this page with your name, centre number and candidate number.
- Answer **all** questions.
- Answer the questions in the spaces provided
  – *there may be more space than you need.*

Information

- The total mark for this paper is 90.
- The marks for **each** question are shown in brackets
  – *use this as a guide as to how much time to spend on each question.*
- In the question labelled with an **asterisk (*)**, marks will be awarded for your ability to structure your answer logically, showing how the points that you make are related or follow on from each other where appropriate.

Advice

- Read each question carefully before you start to answer it.
- Try to answer every question.
- Check your answers if you have time at the end.

Turn over ▶

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P 8 7 9 2 3 A 0 1 2 4

Pearson

Answer ALL questions.

Write your answers in the spaces provided.

Some questions must be answered with a cross in a box ☒. If you change your mind about an answer, put a line through the box ☒ and then mark your new answer with a cross ☒.

Question 1

1 (a) (i)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

1 (a) (ii)

(1)

.....

1 (b) (i)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

1 (b) (ii)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

1 (c)

(2)

.....

.....

.....

.....

.....

.....

(Total for Question 1 = 6 marks)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

Question 2

2 (a) (i)

(1)

- ☒ **A**
- ☒ **B**
- ☒ **C**
- ☒ **D**

2 (a) (ii)

(1)

- ☒ **A**
- ☒ **B**
- ☒ **C**
- ☒ **D**

2 (a) (iii)

(2)

Number of individuals in the population with type III .....

Number of males .....

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

2 (b)

(2)

2 (c)

(3)

(Total for Question 2 = 9 marks)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

Question 3

3 (a)

(1)

- ☒ **A**
- ☒ **B**
- ☒ **C**
- ☒ **D**

3 (b)

(2)

Answer .....

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

3 (c)

(4)

3 (d)

(2)

..... %

(Total for Question 3 = 9 marks)

Question 4

4 (a) (i)

(1)

4 (a) (ii)

(1)

4 (b)

(2)

4 (c)

(2)

4 (d)

(3)

(Total for Question 4 = 9 marks)

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DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

Question 5

5 (a)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

5 (b)

(2)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

5 (c) (i)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

5 (c) (ii)

(3)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

5 (c) (iii)

(4)

(Total for Question 5 = 11 marks)

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DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

Question 6

6 (a)

(1)

6 (b)

(4)

***6** (c)

(6)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

6 (d)

(2)

(Total for Question 6 = 13 marks)

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Question 7

7 (a) (i)

(1)

☒ **A**

☒ **B**

☒ **C**

☒ **D**

7 (a) (ii)

(2)

7 (b) (i)

(1)

7 (b) (ii)

(3)

7 (c) (i)

(3)

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DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

7 (c) (ii)

(3)

(Total for Question 7 = 13 marks)

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DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

Question 8

8 (a)

(1)

8 (b)

(4)

8 (c)

(2)

8 (d)

(2)

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DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

8 (e)

(3)

8 (f)

(2)

8 (g)

(2)

8 (h)

(4)

(Total for Question 8 = 20 marks)

TOTAL FOR PAPER = 90 MARKS

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