

Please check the examination details below before entering your candidate information

Candidate surname		Other names	
Centre Number		Candidate Number	

Pearson Edexcel Level 3 GCE

Time 2 hours	Paper reference	9BN0/03
--------------	-----------------	----------------

Biology A (Salters Nuffield)

Advanced

PAPER 3: General and Practical Applications in Biology

You must have: Ruler, HB pencil and scientific calculator and a copy of the scientific article adapted from <i>Scientific American</i> (enclosed)	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.
- **Show all your working out** in calculations and **include units** where appropriate.
- Answer the questions in the spaces provided
– *there may be more space than you need.*

Information

- The total mark for this paper is 100.
- The marks for **each** question are shown in brackets
– *use this as a guide as to how much time to spend on each question.*
- You may use a scientific calculator.
- In questions marked 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 ►

R67092RA

©2022 Pearson Education Ltd.

Q:1/1/1/1/1/1/

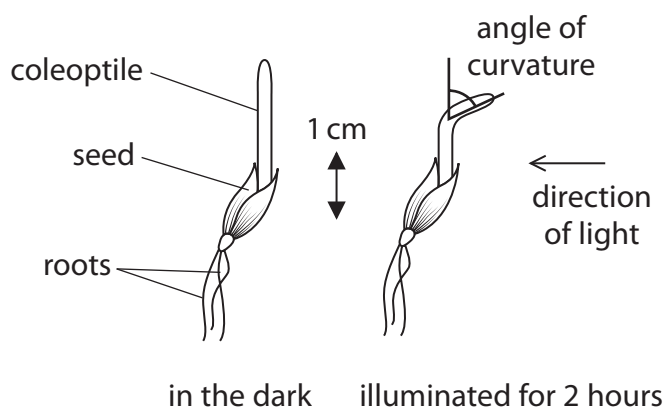
Answer ALL questions.

Write your answers in the spaces provided.

1 When oat seeds germinate, they produce roots and a coleoptile.

(a) The effect of shading the tip of the coleoptile was investigated.

The diagram shows how the coleoptile of an oat seedling can bend towards light.



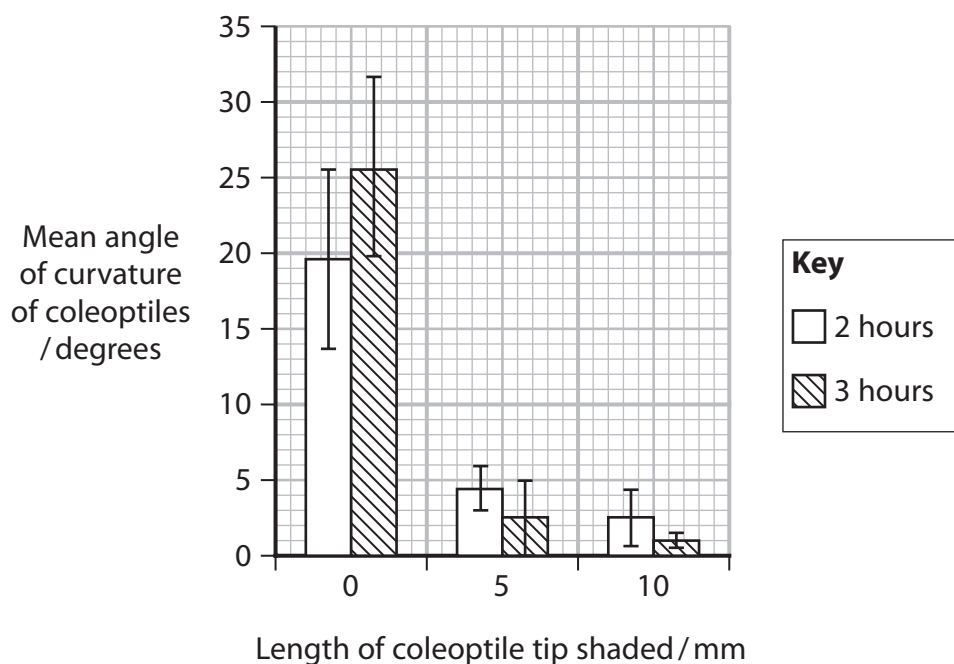
Oat seeds were germinated. When the coleoptiles were 2 cm long the seedlings were split into three groups.

- Group 1 none of the tip of the coleoptile was shaded
- Group 2 5 mm at the tip of the coleoptile was shaded
- Group 3 10 mm at the tip of the coleoptile was shaded

All the coleoptiles were then exposed to light from one side.

The curvature of the coleoptiles was measured after intervals of 2 hours and 3 hours.

The results of this investigation are shown in the graph.



- (i) Describe two conclusions that can be drawn from the results of this investigation.

(2)

- (ii) The results of this investigation were analysed using the Student's t-test.

A p value of 0.05 was used to interpret the results of the t-test.

Explain what a p value of 0.05 means.

(2)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

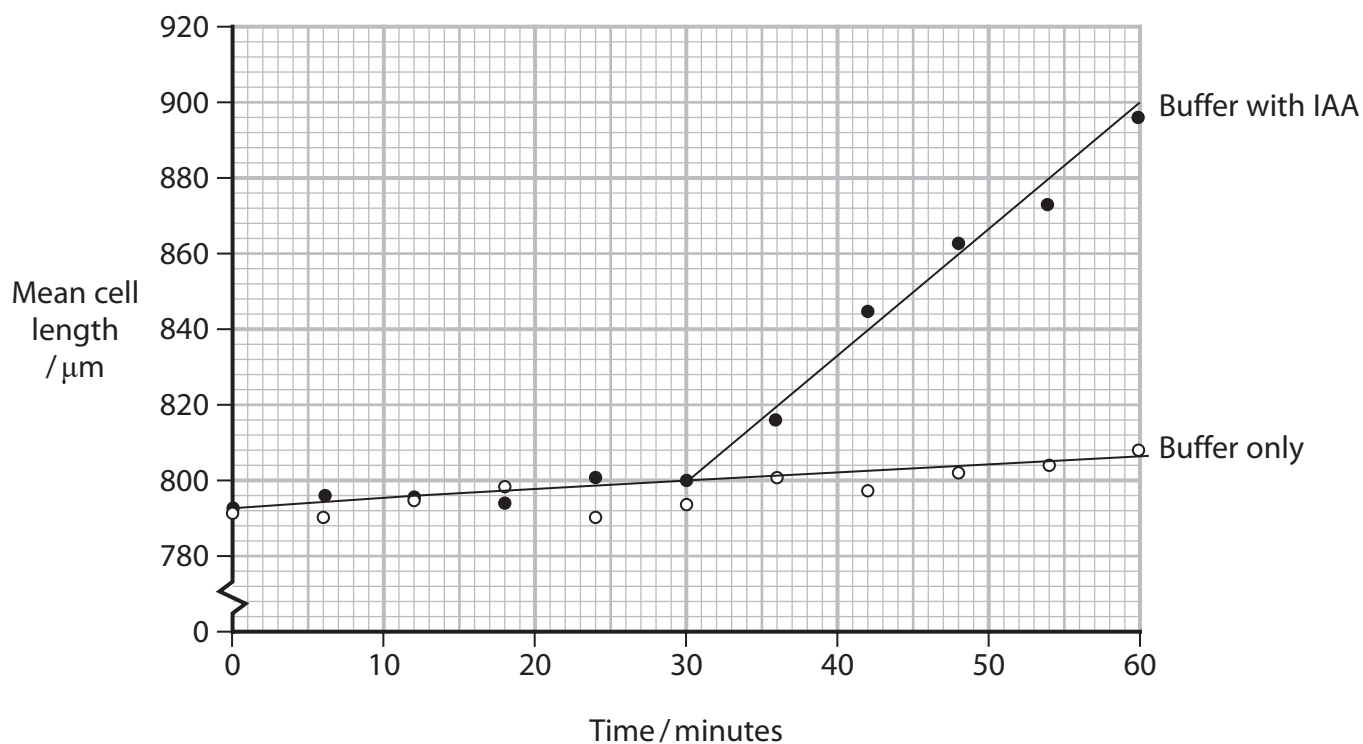
DO NOT WRITE IN THIS AREA

(b) In a second investigation, cells were taken from below the tip of the coleoptile.

These cells were soaked in buffer alone or buffer containing IAA (auxin), for a period of 60 minutes.

The lengths of the cells were measured every six minutes.

The results of this investigation are shown in the graph.



- (i) Give one abiotic factor, other than pH, that would need to be controlled in this investigation.

(1)

- (ii) Calculate the difference in the rates of change in cell length from 30 to 60 minutes, in the presence of IAA and in the absence of IAA.

(2)

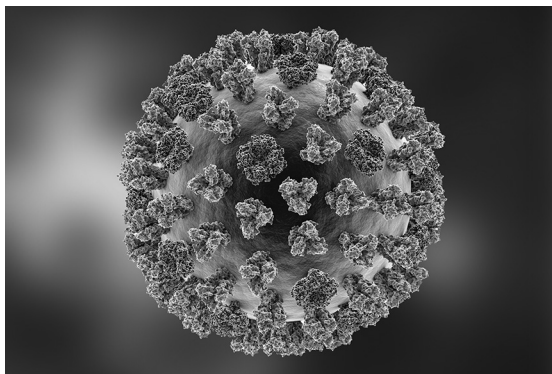
Answer

- (c) Explain how the evidence from these investigations indicates that plant hormones are involved in the phototropic response of oat coleoptiles.

(3)

(Total for Question 1 = 10 marks)

2 The diagram shows an RNA virus that causes influenza.



(Source: © Kateryna Kon/Shutterstock)

(a) Influenza cannot be treated with antibiotics.

Explain why antibiotics cannot be used to treat influenza.

(2)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

- (b) The surface of this virus capsid is covered with 'spike' proteins.

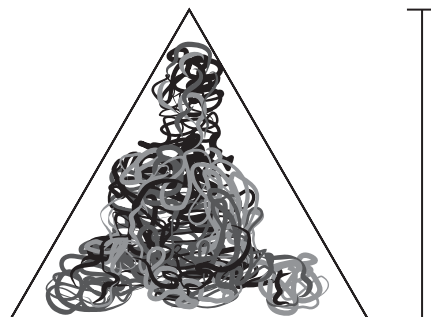
The diameter of the capsid of this virus is 240 nm.

Diagram of the surface of the virus showing each spike protein as a triangle



240 nm

The diagram shows the area occupied by one spike protein



$h = 22.4 \text{ nm}$

$b = 20.0 \text{ nm}$

Calculate the maximum number of spike proteins that can be packed on the surface of one virus particle.

The formula for calculating the surface area of a sphere is $4\pi r^2$

The formula for calculating the area of a triangle is $\frac{h \times b}{2}$

(3)

Answer

- (c) Changes in the RNA of influenza produce new strains of the virus with an altered spike protein.

Devise a procedure to determine the similarity of the strains of influenza in saliva samples collected from different people.

(4)

(Total for Question 2 = 9 marks)

3 Eukaryotic cells contain membrane-bound organelles.

(a) The photograph, obtained using an electron microscope, shows an organelle.



(Source: © Science History Images/Alamy Stock Photo)

- (i) Label a part of this organelle where carbon fixation occurs. (1)
- (ii) Give the name of the component labelled X. (1)
- (iii) Describe how the structure of a membrane in the part labelled X is related to its function. (3)

(iv) Describe how GP is produced by carbon fixation in this organelle.

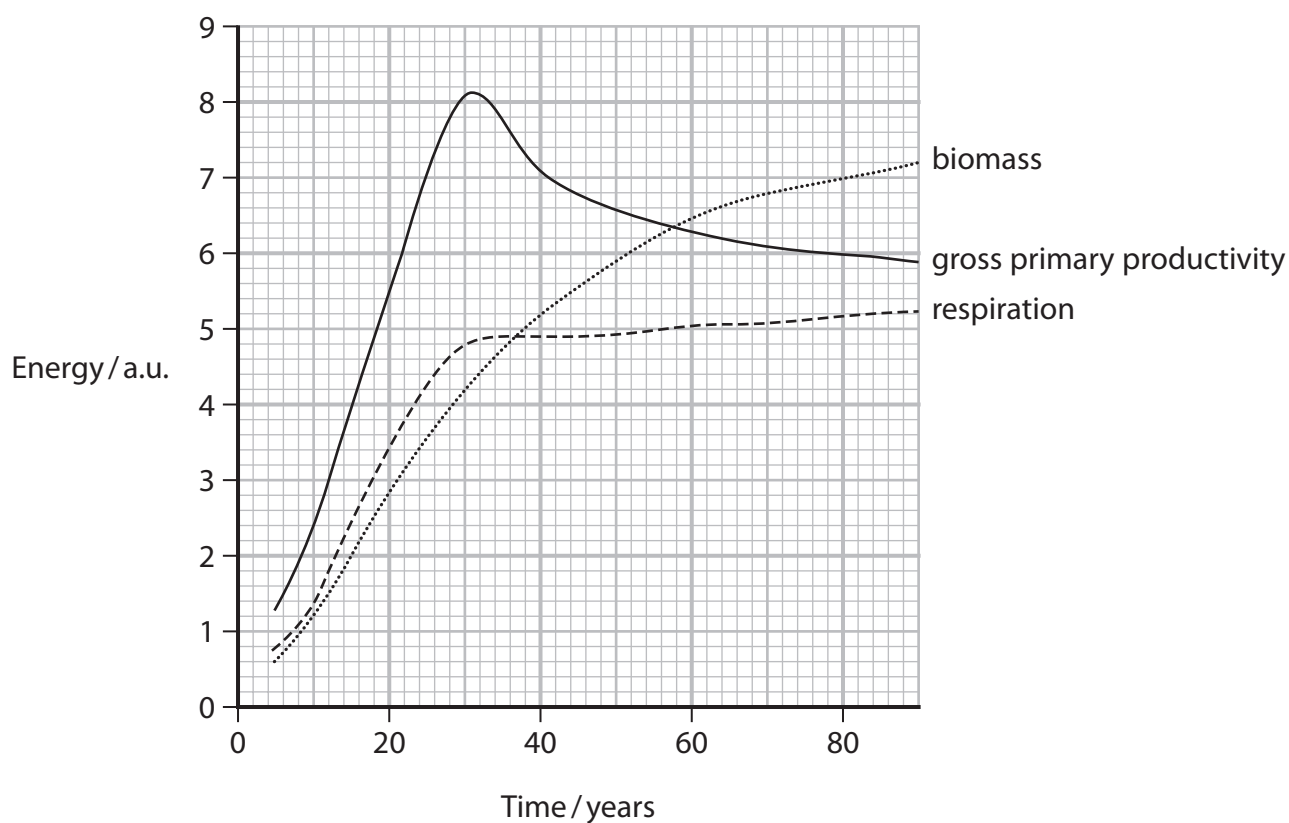
(2)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

(b) The graph shows the changes in biomass, gross primary productivity and the energy used in respiration during succession from grassland to mature forest.



(i) Add shading to the graph to show the area that represents net primary productivity.

(1)

(ii) Describe what is meant by the term succession.

(2)

(iii) Deduce the effect of succession on the level of carbon dioxide in the atmosphere.

(3)

(Total for Question 3 = 13 marks)

- 4 The photograph shows a plant called lantana (*Lantana camara*).



(Source: © Peter Vrabel/Alamy Stock Photo)

The leaves of lantana contain chemicals known to have antimicrobial properties.

The antimicrobial activity of lantana leaf extracts prepared using three different solvents, A, B and C, was compared.

Fresh lantana leaves were dried and powdered. The dried leaf material was mixed with the solvent and then the extract was purified and dried.

The mass of extract obtained from 5 g of powdered leaf, using each solvent, was measured.

Solvent used	Mean mass of extract $\pm$ SD / μg
A	501.3 ± 3.5
B	721.3 ± 1.5
C	245.6 ± 4.0

- (a) The mass of dried and powdered lantana leaves is 10.5% of the mass of fresh leaves.

Calculate the mass of fresh leaves needed to produce 1 mg of extract using solvent A.

Give your answer to three significant figures.

(2)

- (b) The antimicrobial properties of the extracts produced using these solvents are shown in the table.

Solvent used to prepare extract	Dry mass of extract / μg	Bacteria tested	
		<i>Klebsiella pneumoniae</i> (Gram negative)	<i>Micrococcus luteus</i> (Gram positive)
		Mean diameter of the zone of inhibition / mm	
A	5.0	8.3	7.1
	10.0	10.5	7.0
B	5.0	14.5	12.2
	10.0	18.1	18.0

- (i) Deduce the effect of using different solvents on the effectiveness of the extracts against these two bacteria.

(2)

(ii) Devise a method that could be used to collect the data in the table.

(4)

(Total for Question 4 = 8 marks)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

5 Habituation is a learning response observed in many types of animal.

(a) Explain the importance of the habituation response in an animal.

(2)

(b) The 'light-off jump' assay can be used to study habituation in fruit flies.

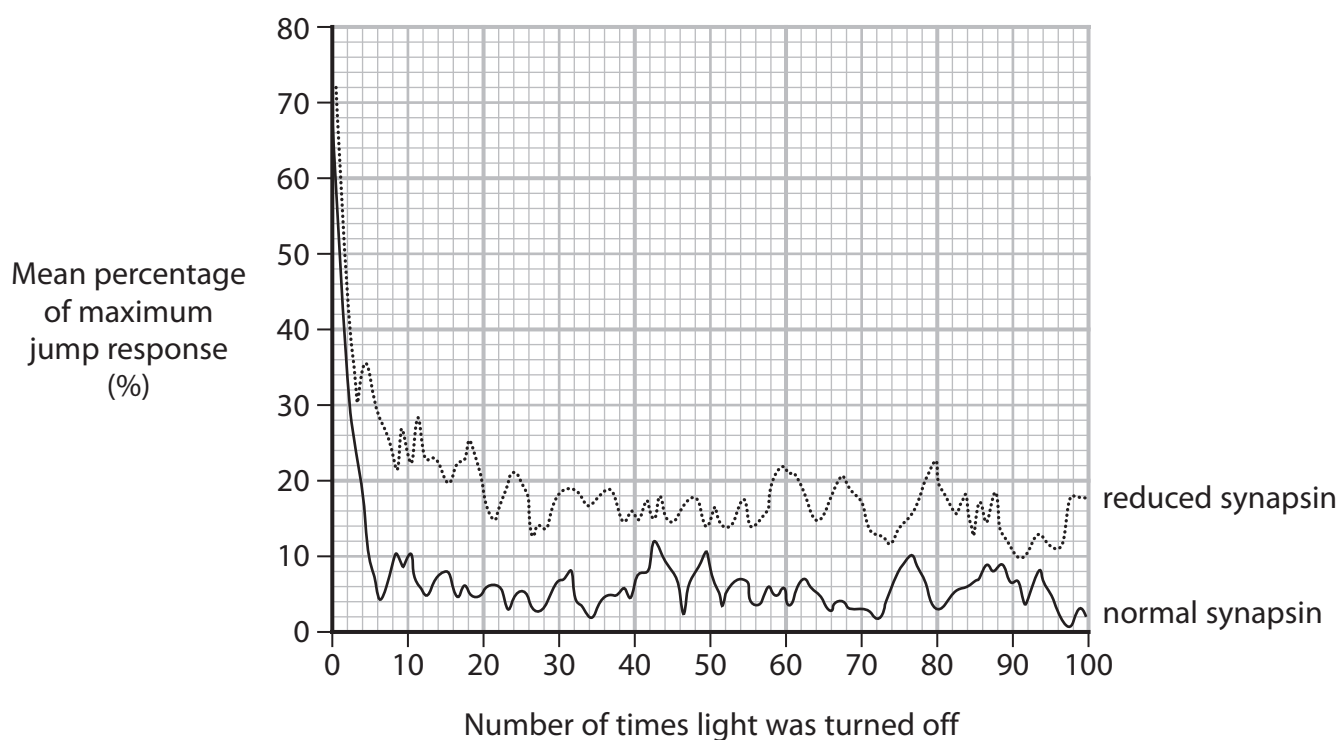
When the light is turned off the flies jump into the air and attempt to fly away.

The strength of this response can be measured by recording the noise made by the flies.

In one experiment, fruit flies with reduced expression of a protein called synapsin were compared with a control group.

Synapsin is a protein that inhibits the binding of presynaptic vesicles to the cell membrane.

The results of this experiment are shown in the graph.



- (i) State two variables associated with the light-off stimulus that need to be controlled in this experiment.

(2)

- (ii) Determine the effect of reduced synapsin on the habituation of fruit flies in this experiment.

(3)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA



(iii) Explain how reduced expression of synapsin could produce these results.

(3)

- (c) Scientists are using fruit fly habituation to investigate the role of genes associated with human autism spectrum disorders (ASD).

Before they can do this, the scientists first identify genes linked to human ASD.

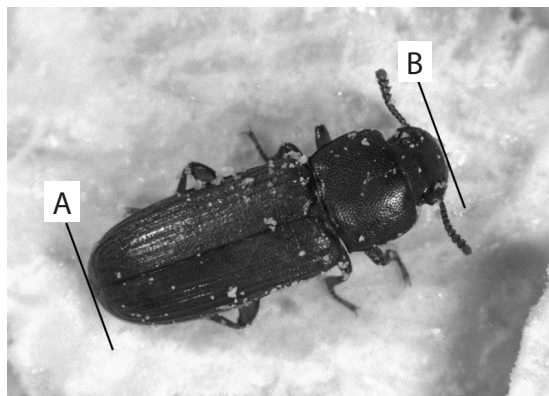
Describe how genes linked to human ASD could be identified.

(2)

(Total for Question 5 = 12 marks)

- 6 The red flour beetle (*Tribolium castaneum*) is a food pest found in flour.

The photograph shows a red flour beetle.



(Source: © Nigel Cattlin/Alamy Stock Photo)

magnification $\times 15$

- (a) Calculate the length of the red flour beetle between A and B.

(1)

mm

- (b) The main food source for adult red flour beetles is starch.

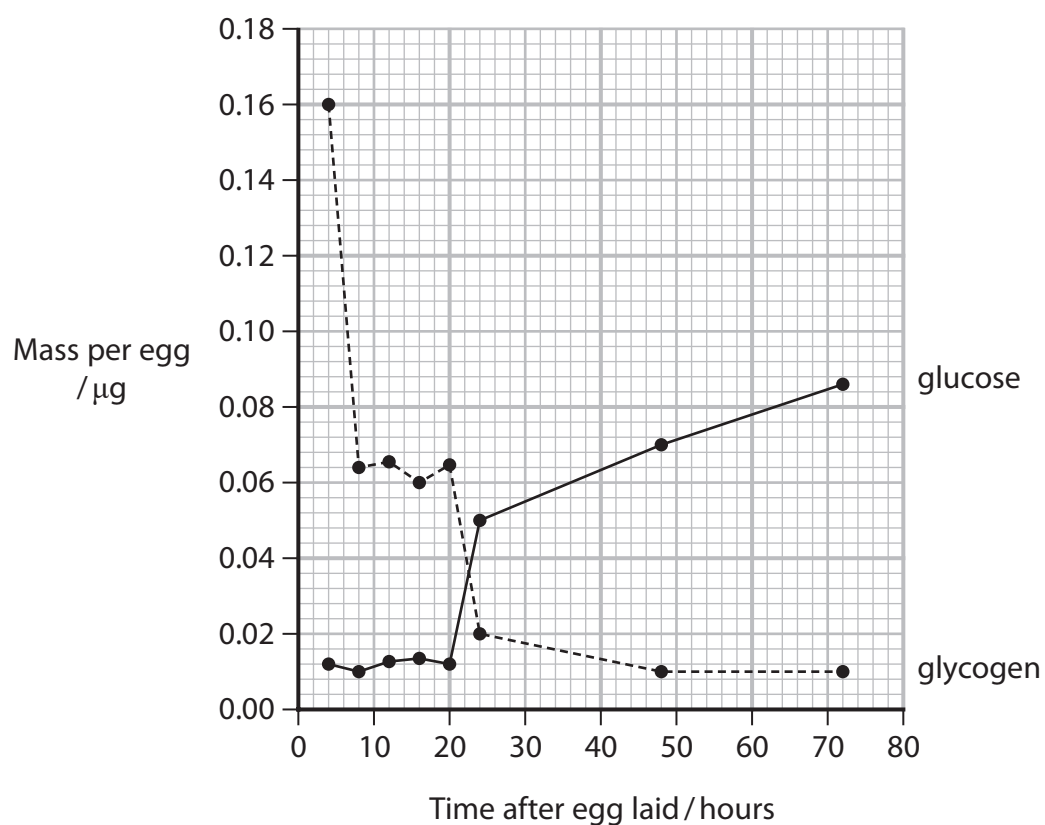
Red flour beetles lay eggs that contain all the nutrients required for the development of the embryo.

The main food source in the eggs is glycogen.

Describe how starch can be used to make glycogen.

(3)

- (c) The graph shows the changes in mass of glucose and glycogen in the eggs of the red flour beetle during embryo development.



- (i) Calculate the percentage of glycogen used from 4 to 8 hours after the eggs were laid.

(2)

%

- (ii) A student suggested that between 20 and 24 hours the cells in the developing embryo switched from anaerobic to aerobic respiration.

Describe how a respirometer could be used to test this suggestion.

(3)

(Total for Question 6 = 9 marks)

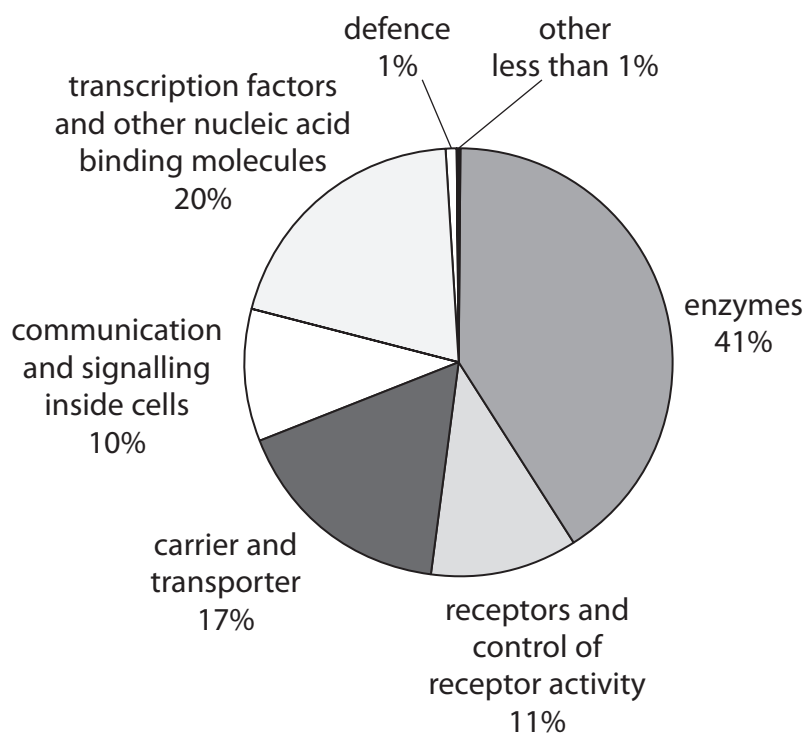
DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

*7 The human genome codes for approximately 20 000 different proteins.

The pie chart shows the proportion of proteins carrying out different functions.



The 20 amino acids used to make proteins can be classified according to the properties of their side chains (R groups).

Table 1 shows the number of amino acids with these properties.

Property of the side chain group	Number of amino acids with the property
Non-polar	9
Polar, uncharged	6
Negatively charged	3
Positively charged	2

Table 1

Table 2 shows three amino acids, used to synthesise proteins, that have unique properties.

Amino acid	Comment on structure
Cysteine	The side chain contains a thiol group (-S-H) that is chemically reactive.
Glycine	The side chain is a hydrogen atom which is much smaller than any other side chain. This allows tight coiling of polypeptide chains.
Proline	The side chain forms a peptide bond with the nitrogen in the amino group. This makes a polypeptide chain more rigid.

Table 2

Discuss the importance of the amino acid side chain to the structure, function and location of proteins.

(9)

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

(Total for Question 7 = 9 marks)



Scientific article for use with Question 8

Darwin's Cancer Fix

1. This year at least 31,000 men in the U.S. will be diagnosed with prostate cancer that has spread to other parts of their body, such as bones and lymph nodes. Most of them will be treated by highly skilled and experienced oncologists, who have access to 52 drugs approved to treat this condition. Yet eventually more than three quarters of these men will succumb to their illness.
2. Cancers that have spread, known as metastatic disease, are rarely curable. The reasons that patients die despite effective treatment are many, but they all trace back to an idea popularized in 1859 by Charles Darwin to explain the rise and fall of species of birds and tortoises. Today we call it evolution.
3. Think of a cancer cell like Darwin's Galapagos finches, which had slightly different beaks on various islands. Finches eat seeds, and seeds on each island had different shapes or other characteristics. The bird with a beak shape best matched to the local seed got the most food and had the most offspring, which also had that particular beak shape. Birds with less adaptive beaks did not make it. This natural selection ensured that different finch species, with various beaks, evolved on each island. The key is that when two groups of critters compete in the same small space, the one better adapted to the environment wins out.
4. Cancer cells evolve in a similar manner. In normal tissue, regular non-cancer cells thrive because they are a good fit for the biochemical growth signals, nutrients and physical cues they get from surrounding healthy tissue. If a mutation creates a cancer cell poorly adapted for those surroundings, it does not stand much chance initially; normal cells out-compete it for resources. But if the surroundings are further damaged by inflammation—sometimes a growing cancer can cause this itself—or old age, the cancer cell does better and starts to out-compete normal cells that used to crowd it out. The change in the surroundings ultimately determines a cancer cell's success.
5. This is a theory we call adaptive oncogenesis, and we have found evidence that supports it in the way cancer takes off when we change its cellular environment in experimental animals, although the internal workings of the cancer cell have not changed. Doctors have also observed this acceleration of cancer in humans with tissue-disturbing ailments such as inflammatory bowel disease. The overall implication is that we can best understand cancer by looking at its surroundings rather than solely focusing on the mutations inside a cell. By reducing tissue alterations caused by processes such as inflammation, we can restore a more normal environment and—as we have shown in animal studies—prevent cancer from gaining a competitive edge.
6. Our evolutionary perspective also has inspired a different approach to cancer therapy, one that we have successfully tested in small clinical trials. Doctors dump a lot of chemotherapy drugs on a cancer in an effort to kill every last trace of the threat, and at first this often looks like it works. The tumor shrinks or goes away. But then it comes back and is resistant to the drugs that once killed these cells, akin to crop-destroying insects that evolve resistance to pesticides. In a clinical trial with prostate cancer patients, one of us (Gatenby) tried an alternative to the scorched-earth approach, applying only enough chemo to keep the tumor tiny without killing it entirely. The goal was to maintain a small population of vulnerable chemosensitive cells. That population did well enough to prevent cells with an unwanted new trait—chemoresistance—from taking over. In a group of patients in which tumors usually start growing uncontrollably after 13 months, this regimen has kept tumors under control for 34 months on average—with less than half the standard drug dose.

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

DO NOT WRITE IN THIS AREA

7. The results of our prevention and therapeutic strategies may point to a way to ward off cancer before it becomes a danger to life and limb and to save many patients for whom a regimen of giant, toxic drug doses has failed.

WHY DO WE GET CANCER?

8. If you asked almost any doctor or cancer researcher, “Why is aging, smoking or radiation exposure associated with cancer?” you would probably get a short answer “These things cause mutations”. This assessment is partly true. Exposure to cigarette smoke or radiation does cause mutations in our DNA, and mutations do accumulate in our cells throughout life. The mutations can provide cells with new properties, such as hyperactive growth signals for cell divisions, reduced death rates or even an increased ability to invade surrounding tissue.
9. Yet this simple explanation, focused on changes within cells, overlooks the fact that a major driver of change in any single cell—or in entire collections of them such as human beings—is outside, in the cell’s environment.
10. We know that the evolution of species on the earth has been highly dependent on environmental perturbations, including dramatic changes to landmasses, the gases in the air and water, and ambient temperature. These changes led to selection for new adaptive features in organisms producing amazing diversity. As Darwin wrote in *On the Origin of Species* in 1859, “Owing to this struggle for life, any variation, however slight and from whatever cause proceeding, if it be in any degree profitable to an individual of any species, *in its infinitely complex relations to other organic beings and to external nature*, will tend to the preservation of that individual, and will generally be inherited by its offspring”. Darwin proposed that competition for limited resources would drive selection for individuals with traits that were best adapted to the environment. And when environments changed, so would these pressures, selecting for new traits that were better tuned to the new surroundings.
11. Similar Darwinian dynamics should apply to the evolution of cancers in our body. Even though we trained as a molecular biologist (DeGregori) and a physician (Gatenby), evolution and ecology have always fascinated both of us. Our extensive reading in these areas while initially driven by what we thought was curiosity unrelated to our day jobs, revealed unappreciated parallels between the driving forces of evolution and our observations of cancer development and cancer patients’ responses to therapy.
12. For instance, cancer researchers typically believed that a cancer-causing mutation would always confer an advantage to a cell that acquired it, but we recognized a classic evolutionary principle at work: a mutation does not automatically help or hinder an organism. Instead its effects are dependent on features of the local environment. In Darwin’s finches, there is no “better” beak shape per se, but certain beaks improve survival under certain conditions. Similarly, we reasoned that a mutation in a cancer-causing gene does not provide an inherent advantage to the cell and can, in fact, be disadvantageous if it makes that cell less able to use the resources of the tissue immediately around it.
13. We were also inspired by the punctuated equilibrium theory of paleontologists Niles Eldredge and Stephen Jay Gould, who noted that species often maintain stable traits through millions of years of fossil records, only to suddenly evolve rapidly in response to a dramatic environmental change. This concept stimulated our ideas about the way that some tissues could be initially unfavorable to cell mutations, but changes in those tissues, such as damage and inflammation in a smoker’s lungs, could stimulate evolutionary change—sometimes leading to cancer.

14. We first saw this dynamic at work with aging-associated changes in bone marrow that led to the development of leukemias. Working with groups of young and old mice in DeGregori's Colorado lab, Curtis Henry, now at Emory University, and Andriy Marusyk, now at the Moffitt Cancer Center, created the same cancer-causing mutations in a few of the mice's bone marrow stem cells. The results showed that the same cancer-causing mutations can have very different effects on the fate of these cells, depending on age: the changes promoted the proliferation of mutated cells in the old mice but not in the young ones. And the determining factor did not appear to be in the mutated cells but in the metabolism and gene activity of the normal cells around them. For example, the activity of genes important for stem cell division and growth was reduced in non-mutated stem cells in the bone marrow of old mice, but it was restored in these cells when we introduced the cancer-causing mutation. Yet the mutation that helped these cells had bad effects on the mice. These stem cells normally generate key players in the body's immune system, but the population explosion of the cancer-mutated version of the cells instead led to the development of leukemias.
15. On the other hand, the fit young stem cells in the tissues of a young mouse already had levels of growth and energy use that nicely matched what their surroundings could provide. Therefore, such cells did not benefit from the cancer-causing mutations when we introduced them. The mutated cell populations did not grow. By favoring the status quo, youth is tumor-suppressive.
16. Why does any of this matter? Although we can avoid some mutations by not smoking and keeping clear of other mutagenic exposures, many, if not most, of the mutations that we accumulate in our cells during life cannot be dodged. But this new focus on tissue environments introduces a way to limit cancer: reversing tissue alterations caused by aging, smoking and other insults will reduce the success of cancer-causing mutations. The mutations will still occur, but they will be much less likely to give cells an advantage and thus will not grow in number.
17. Of course, there is no Fountain of Youth to reverse or prevent aging. Doing the things we know we should, such as exercising, eating a balanced diet and not smoking, can improve the maintenance of our tissues, which may be the best strategy we can use for the moment. But, if we can figure out what key tissue environmental factors favor cancer development, we should be able to change these factors to limit malignancies. Indeed, in our mouse experiments, we showed that when we reduced the activity of inflammation-causing and tissue-damaging proteins in old mice, cells with the cancer-causing mutations did not proliferate; normal cells maintained their dominance. But we must proceed cautiously. Blocking inflammation in mice living in sterile cages may reduce cancer, but a similar strategy in people in the real world could limit defenses against infections because inflammation is part of our immune response.

FROM PREVENTION TO THERAPY

18. In addition to primary prevention, an evolutionary understanding can help make therapies for existing cancers more effective by reducing the nasty tendency of such cells to develop drug resistance. The evolution of resistance happens in other realms. Perhaps the most familiar example is the centuries-old contest between farmers and crop-destroying insects. For more than a century pesticide manufacturers produced a steady stream of new products, but the pests always evolved resistance. Eventually, manufacturers recognized that trying to eradicate the pests by spraying high doses of pesticides on fields was making the problem worse because of an evolutionary process termed competitive release.
19. To understand competitive release, remember that all the insects within a large population occupying a field are continuously competing with one another for food and space, and they are not identical (as is also the case for cancer cells). In fact, for nearly every trait, including sensitivity to a pesticide, there is inevitable variability within a population. By spraying a large amount of insecticide (or administering a large dose of chemotherapy), the farmer (or oncologist) may kill the vast majority of insects (or cancer cells). Yet a few insects (or cells) have traits that make them less vulnerable and with the highly vulnerable organisms removed, the resistant ones begin to spread. A farming strategy called integrated pest management tries to deal with this situation by using pesticides sparingly. Rather than trying to eradicate the pests, farmers spray only enough to control them and lower crop damage without resulting in competitive release. In this way, sensitivity of the pest to the pesticide is maintained.
20. The medical community has learned a similar lesson with antibiotics: excessive use must be stopped to curtail the constant evolutionary cycle that produces the development of drug-resistant pathogens. But this lesson has not yet taken hold in the cancer field.
21. Like farmers who used to blast fields with huge amounts of insecticides, doctors today typically give chemotherapy to patients at “maximum tolerated dose (MTD) until progression”. Nearly all cancer drugs also damage normal tissues in the body, and these side effects can be very unpleasant and even fatal. MTD means the drugs are given in amounts that fall just short of killing the patient or causing intolerable side effects. Giving the same treatment “until progression” emerges from a traditional metric of treatment success, based on the tumor’s change in size. Drugs are deemed successful when the tumor shrinks, and the treatment is abandoned if it gets bigger.
22. To most patients and doctors, treatment designed to kill the maximum number of cancer cells with relentless administration of the greatest possible amount of lethal drugs feels like the best approach. But as in the control of insects and infectious diseases, this strategy, in the setting of an incurable cancer, is often evolutionarily unwise because it sets in motion a series of events that actually accelerate the growth of drug-resistant cancer cells.
23. The other evolutionary lesson learned from pest control is that a “resistance management plan” can keep unwanted populations in check, often indefinitely. Can this strategy also lead to better outcomes for patients with incurable cancers? The answer is not yet clear, but there are hints from experimental studies and early clinical trials that it could do just that.

24. An evolution-based strategy for a patient who, after a month on an anti-cancer drug, had a 50 percent reduction in tumor size would be to stop treatment. This approach would be used only when we knew from past experience that available treatments—chemo, hormone therapy, surgery, immune system boosters—would not cure the cancer in this patient. Because a cure would not be possible, the goal would instead be to keep the tumor from growing and metastasizing for as long as possible. By stopping therapy, we would leave behind a large number of treatment-sensitive cancer cells. The tumor would then begin to grow back and eventually reach its previous size. Yet during this regrowth period, because no chemotherapy would be administered, the majority of tumor cells would still be sensitive to the anti-cancer drug, not resistant to it. In effect, we would use the sensitive cells that we could control to suppress the growth of the resistant cells that we could not control. As a result, the treatment would be able to maintain tumor control much longer than the conventional approach of continuous administration of maximum dose and, because the drug dose would be significantly reduced, with much less toxicity and better quality of life.
25. Gatenby's lab began by investigating the approach in 2006 using mathematical models and computer simulations. Although such models had rarely been employed in cancer treatment planning, the large number of possible treatment options required us to adopt an approach common in physics, in which mathematical results help define experimental methods that are likely to be successful. Our models defined the levels of drugs we wanted to test. The next step was to try those doses in mouse experiments, and doing so confirmed that tumor control could be greatly improved by evolution-based strategies.
26. The results were good enough to prompt a move into the clinic and a test on human cancer patients. We were joined in this effort by Jingsong Zhang, an oncologist at the Moffitt Cancer Center who treats men with prostate cancer. With the help of Zhang, along with mathematicians and evolutionary biologists, we developed a model of the evolutionary dynamics of prostate cancer cells during treatment. We used this model to simulate the responses of prostate cancer to a variety of drug doses administered by an oncologist. Then we ran these encounters over and over again until we arrived at a series of doses that kept the cancer in check for the longest time without increasing the population of drug-resistant cells.
27. Next, we asked patients with aggressive prostate cancer that had already metastasized to other locations—the kind that doctors cannot completely eliminate from the body—to volunteer for a clinical trial. So far the patients have had excellent outcomes. Of the 18 people enrolled, 11 are still in treatment. Standard therapy typically maintains control of metastatic prostate cancer for an average of about 13 months. In our trial, average tumor control is at least 34 months, and because more than half of our patients are still being treated actively, we cannot yet place an upper limit on how well they do. Furthermore, this control is being achieved using only 40 percent of the drug dose that patients would have received in standard treatment. But it is still early days for this treatment approach. Just because it works in prostate cancer does not mean it works in stomach cancer, for instance. And it may be tough to convince patients, even those with an incurable disease, that the best approach is not to kill as many cancer cells as possible but as few as necessary.

THE RULES OF CANCER

28. In many ways, the evolutionary model of cancer development and treatment serves to dispel the 'mystery' of cancer. The proclivity of the disease to strike without any clear cause, along with its ability to overcome and return even after highly effective and often highly toxic therapy, can be viewed by patients and caregivers as both hopelessly complicated and magically powerful. In contrast, understanding that cancer obeys the rules of evolution like all other living systems can give us confidence that we have a chance to control it. Even without a cure, by using our understanding of evolutionary dynamics, we can strategically alter therapy to get the best possible outcome. And prevention strategies can be geared toward helping to create tissue landscapes in the body that favor normal cells over cancer cells.
29. For more than a century the cancer research community has sought "silver bullets"—drugs that can eliminate all cancer cells while sparing all normal cells. Cancer has been taking advantage of evolution to sidestep these drugs. But we can use evolution, too. We have the opportunity to expand the work of Darwin and his successors to develop more realistic approaches to both prevent and tame this deadly disease.

Source adapted from:

James DeGregori and Robert Gatenby, 'Principles of Evolution and Natural Selection Drive a Radical New Approach to Drugs and Prevention Strategies' in *Scientific American*, August 2019

8 The scientific article you have studied is adapted from *Scientific American*.

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

- (a) Explain why natural selection took place in Darwin's Galapagos finches (paragraph 3).

(3)

- (b) Explain how inflammation and the immune response can cause damage to tissues (paragraph 4).

(3)

(c) Describe how tumour shrinkage could be observed (paragraph 6).

(2)

(d) "Why is aging, smoking or radiation exposure associated with cancer?" (paragraph 8).

Explain why "These things cause mutations".

(3)

(e) Explain why a cancer cell needs to 'use the resources of the tissue immediately around it' (paragraph 12).

(2)

- (f) Explain how 'a cancer-causing mutation' could increase the rate of division in stem cells (paragraph 14).

(3)

- (g) Describe how a cell that is 'a key player in the body's immune system' differs from a stem cell (paragraph 14).

(4)

- (h) Describe how the effects of 'competitive release' could be demonstrated (paragraphs 18 and 19).

(4)

- (i) Describe how evasion mechanisms can enable pathogens to become drug-resistant (paragraph 20).

(2)

- (j) Discuss the ethical issues relating to the use of mice in experiments such as those described in the article (paragraph 25).

(4)

(Total for Question 8 = 30 marks)

TOTAL FOR PAPER = 100 MARKS