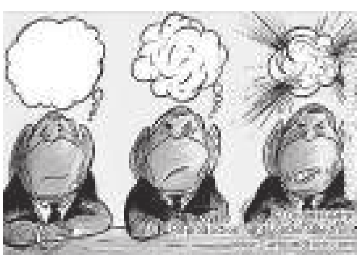
SECTION 1

brain Activating

A In 1937 the great neuroscientist Sir Charles Scott Sherrington of the University of Oxford laid out what would become a classic description of the brain at work. He imagined points of light signaling the activity of nerve cells and their connections. During deep sleep, he proposed, only a few remote parts of the brain would twinkle, giving the organ the appearance of a starry night sky. But at awakening, "it is as if the Milky Way entered upon some cosmic dance," Sherrington reflected. "Swiftly the head-mass becomes an enchanted loom where millions of flashing shuttles weave a dissolving pattern, always a meaningful pattern though never an abiding one; a shifting harmony of subpatterns."

B Although Sherrington probably did not realize it at the time, his poetic metaphor contained an important scientific idea: that of the brain revealing its inner workings optically. Understanding how neurons work together to generate thoughts and behavior remains one of the most difficult open problems in all of biology, largely because scientists generally cannot see whole neural circuits in action. The standard approach of probing one or two neurons with electrodes reveals only tiny fragments of a much bigger puzzle, with too many pieces missing to guess the full picture. But if one could watch neurons communicate, one might be able to deduce how brain circuits are laid out and how they function. This alluring notion has inspired neuroscientists to attempt to realize Sherrington's vision.



C Their efforts have given rise to a nascent field called optogenetics, which combines genetic engineering with optics to study specific cell types. Already investigators have succeeded in visualizing the functions of various groups of neurons. Furthermore, the approach has enabled them to actually control the neurons remotely—simply by toggling a light switch. These achievements raise the prospect that optogenetics might one day lay open the brain's circuitry to neuroscientists and perhaps even help physicians to treat certain medical disorders.

D Enchanting the Loom Attempts to turn Sherrington's vision into reality began in earnest in the 1970s. Like digital computers, nervous systems run on electricity; neurons encode information in electrical signals, or action potentials. These impulses, which typically involve voltages less than a tenth of those of a single AA battery, induce a nerve cell to release neurotransmitter molecules that then activate or inhibit connected cells in a circuit. In an effort to make these electrical signals visible, Lawrence B. Cohen of Yale University tested a large number of fluorescent dyes for their ability to respond to voltage changes with changes in color or intensity. He found that some dyes indeed had voltage-sensitive optical properties. By staining neurons with these dyes, Cohen could observe their activity under a microscope.

E Dyes can also reveal neural firing by reacting not to voltage changes but to the flow of specific charged atoms, or ions. When a neuron generates an action potential, membrane channels open and admit calcium ions into the cell. This calcium influx stimulates the release of neurotransmitters. In 1980 Roger Y. Tsien, now at the University of California, San Diego, began to synthesize dyes that could indicate shifts in calcium concentration by changing how brightly they fluoresced. These optical reporters have proved extraordinarily valuable, opening new windows on information processing in single neurons and small networks.

F Synthetic dyes suffer from a serious drawback, however. Neural tissue is composed of many different cell types. Estimates suggest that the brain of a mouse, for example, houses many hundreds of types of neurons plus numerous kinds of support cells. Because interactions between specific types of neurons form the basis of neural information processing, someone who wants to understand how a particular circuit works must be able to identify and monitor the individual players and pinpoint when they turn on (fire an action potential) and off. But because synthetic dyes stain all cell types indiscriminately, it is generally impossible to trace the optical signals back to specific types of cells.

G Optogenetics emerged from the realization that genetic manipulation might be the key to solving his problem of indiscriminate staining. An individual's cells all contain the same genes, but hat makes two cells different from each other is that different mixes of genes get turned on or off in them. Neurons that release the neurotransmitter dopamine when they fire, for instance, need the enzymatic machinery for making and packaging dopamine. The genes encoding the protein components of this machinery are thus switched on in dopamine producing (dopaminergic) neurons but stay off in other, non-dopaminergic neurons. In theory, if a biological switch that turned a dopamine-making gene on was linked to a gene encoding a dye and if the switch-and-dye unit were engineered into the cells of an animal, the animal would make the dye only in dopaminergic cells. If researchers could peer into the brains of these creatures (as is indeed possible), they could see dopaminergic cells functioning in virtual isolation from other cell types. Furthermore, they could observe these cells in the intact, living brain. Synthetic dyes cannot perform this type of magic, because their production is not controlled by genetic switches that flip to on exclusively in certain kinds of cells. The trick works only when a dye is encoded by a gene—that is, when the dye is a protein.

H The first demonstrations that genetically encoded a decade ago, from teams led independently by Tsien, Ehud Y. Isacoff of the University of California, Berkeley with James E. Rothman, now at Yale University. In all cases, the gene for the dye was borrowed from a luminescent marine organism, typically a jellyfish that makes the so-called green fluorescent protein .Scientists tweaked the gene so that its protein product could detect and reveal the changes in voltage or calcium that underlie signaling within a cell, as well as the release of neurotransmitters that enable signaling between cells.

Questions 1-5

Do the following statements agree with the information given in Reading Passage **1**?
*In boxes **1-5** on your answer sheet, write*

TRUE	*if the statement is true*
FALSE	*if the statement is false*
NOT GIVEN	*if the information is not given in the passage*

1 Sherrington's imaginary picture triggered scientists' enthusiasm of discovering how the whole set of neurons operates.

2 A jumped-up domain optogenetic is a pure unexpected accident.

3 Electric tension is one key component to realize the communication between neurons.

4 The variations of voltages is the sole response that the coloration of related neurons could provide when neural discharge takes place.

5 The vital defect synthetic dyes possess is the most challenging obstacle for researchers to overcome .

Questions 6-10

*The reading Passage has seven paragraphs **A-H**.*
Which paragraph contains the following information?
Write the correct letter ***A-H,*** in boxes ***6-10*** on your answer sheet.

6 a sea creature producing light triggered by certain genes
7 first attempts to make a great idea come true
8 the reason to explain the failure of synthetic dyes
9 difficulty in observing how the whole set of neurons works
10 visual indicators to show how information is handled in and between cells in the Brain

Questions 11-13

Summary

Complete the following summary of the paragraphs of Reading Passage， using ***no more than three*** words from the Reading Passage for each answer. Write your answers in boxes **11-13** on your
answer sheet.

> Synthesized by enzymatic machinery , ...11.......... plays as vehicle for the information flow between cells. Protein is the ingredient of the enzymatic machinery, so first it needs genes in charge of encoding the required protein ....12.......... before the neutrontransimitter is produced. This ....13..........can be used to differentiate the dopaminergic neurons from the nondopaminergic counterparts with a premise that the dye is a protein after a transfer process.

SECTION 2

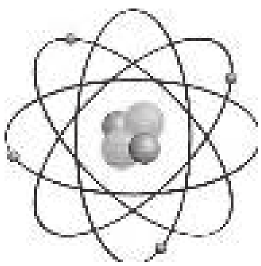
war debris could cause cancer

A Could the mystery over how depleted uranium might cause genetic damage be closer to being solved? It may be, if a controversial claim by two researchers is right. They say that minute quantities of the material lodged in the body may kick out energetic electrons that mimic the effect of beta radiation. This, they argue, could explain how residues of depleted uranium scattered across former war zones could be increasing the risk of cancers and other problems among soldiers and local people.

B Depleted uranium is highly valued by the military, who use it in the tips of armour-piercing weapons. The material's high density and self-sharpening properties help it to penetrate the armour of enemy tanks and bunkers. Its use in conflicts has risen sharply in recent years. The UN Environment Programme (UNEP) estimates that shells containing 1700 tonnes of the material were fired during the 2003 Iraq war. Some researchers and campaigners are convinced that depleted uranium left in the environment by spent munitions causes cancer, birth defects and other ill effects in people exposed to it. Governments and the military disagree, and point out that there is no conclusive epidemiological evidence for this. And while they acknowledge that the material is weakly radioactive, they say this effect is too small to explain the genetic damage at the levels seen in war veterans and civilians.

C Organisations such as the UK's Royal Society, the US Department of Veterans Affairs and UNEP have called for more comprehensive epidemiological studies to clarify the link between depleted uranium and any ill effects. Meanwhile, various test-tube and animal studies have suggested that depleted uranium may increase the risk of cancer, according to a review of the scientific literature published in May 2008 by the US National Research Council. The authors of the NRC report argue that more long-term and quantitative research is needed on the effects of uranium's chemical toxicity. They say the science seems to support the theory that genetic damage might be occurring because uranium's chemical toxicity and weak radioactivity could somehow reinforce each other, though no one knows what the mechanism for this might be.

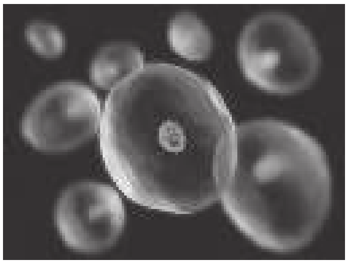
D Now two researchers, Chris Busby and Ewald Schnug, have a new theory that they say explains how depleted uranium could cause genetic damage. Their theory invokes a well-known process called the photoelectric effect. This is the main mechanism by which gamma photons with energies of about 100 kiloelectronvolts (keV) or less are blocked by matter: the photon transfers its energy to an electron in the atom's electron cloud, which is ejected into the surroundings. An atom's ability to stop photons by this mechanism depends on the fourth power of its atomic number – the number of protons in its nucleus – so heavy elements are far better at intercepting gamma radiation and X-rays than light elements. This means that uranium could be especially effective at capturing photons and kicking out damaging photoelectrons: with an atomic number of 92, uranium blocks low-energy gamma photons over 450 times as effectively as the lighter element calcium, for instance.



E Busby and Schnug say that previous risk models have ignored this well-established physical effect. They claim that depleted uranium could be kicking out photoelectrons in the body's most vulnerable spots. Various studies have shown that dissolved uranium – ingested in food or water, for example – is liable to attach to DNA strands within cells, because uranium binds strongly to DNA phosphate. "Photoelectrons from uranium are therefore likely to be emitted precisely where they will cause most damage to genetic material," says Busby.

F Busby and Schnug base their claim on calculations of the photoelectrons that would be produced by the interaction between normal background levels of gamma radiation and uranium in the body. "Our detailed calculations indicate that the phantom photoelectrons are the predominant effect by far for uranium genome toxicity, and that uranium could be 1500 times as powerful as an emitter of photoelectrons than as an alpha emitter." Their computer modelling results are described in a peer-reviewed paper to be published in this month by the IPNSS in a book called Loads and Fate of Fertiliser Derived Uranium.

G Hans-Georg Menzel, who chairs the International Commission on Radiological Protection's committee on radiation doses, acknowledges that the theory should be considered, but he doubts that it will prove significant. He suspects that under normal background radiation the effect is too weak to inflict many of the "double hits" of energy that are known to be most damaging to cells. "It is very unlikely that individual cells would be subject to two or more closely spaced photoelectron impacts under normal background gamma irradiation," he says. Despite his doubts, Menzel raised the issue last week with his committee in St Petersburg, Russia, and says that several colleagues "intended to collect relevant data and perform calculations to check whether there was any possibility of a real effect in living tissues". Organisations in the UK, including the Ministry of Defence and the Health Protection Agency, say they have no plans to investigate Busby's hypothesis.



H Radiation biophysicist Mark Hill of the University of Oxford would like to see a fuller investigation, though he suggests this might show that the photoelectric effect is not as powerful as Busby claims. "We really need more detailed calculations and dose estimates for realistic situations with and without uranium present," he says. Hill's doubts centre on an effect called Compton scattering, which he believes needs to be factored into any calculations. With Compton scattering, uranium is only 4.5 times as effective as calcium at stopping gamma photons, so Hill says that taking it into account would reduce the relative importance of uranium as an emitter of secondary electrons. If he is right, this would dilute the mechanism proposed by Busby and Schnug.

I The arguments over depleted uranium are likely to continue, whatever the outcome of these experiments. Whether Busby's theory holds up or not remains to be seen, but investigating it can only help to clear up some of the doubts about this mysterious substance.

Questions 14-18

The reading Passage has nine paragraphs A-I.

Which paragraph contains the following information?

Write the correct letter *A-I*, in boxes *14-18* on your answer sheet.

NB you may use any letter more than once

14 a famous process is given relating to the new theory.
15 a person who acknowledges but suspects the theory.
16 the explanation of damage to DNA.
17 a debatable and short explanation to the way creating the problems of soldiers.
18 Busby's hypothesis is not in the investigation plans of organisations.

Questions 19-22

Do the following statements agree with the information given in Reading Passage 2?
In boxes 19-22 on your answer sheet, write

TRUE	*if the statement is true*
FALSE	*if the statement is false*
NOT GIVEN	*if the information is not given in the passage*

19 all of people believe that depleted uranium is harmful to people's health.
20 heavier elements can perform better at preventing X-rays and gamma radiation.
21 by particular calculations, it is known that the main effect of uranium genome toxicity is phantom photoelectrons.
22 most of scientists support Mark Hill's opinion.

Questions 23-26

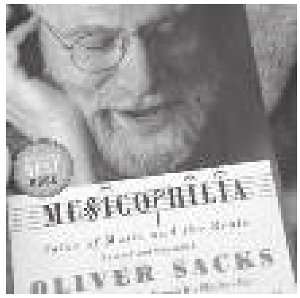
Summary

Complete the following summary of the paragraphs of Reading Passage，using ***no more than two*** words from the Reading Passage for each answer. Write your answers in boxes **23-26** on your answer sheet.

23____________ attaches importance to depleted uranium due to its 24__________ and 25__________ features, which are helpful in the war. However, it has ill effects in people, and then causes organisations' appeal to do more relative studies. According to some scientists, we should do research about the impact of uranium's 26______________ which may be enhanced with weak radioactivity.

SECTION 3

Musicophilia



Norman M. Weinberger reviews the latest work of Oliver Sacks on music.

A Music and the brain are both endlessly fascinating subjects, and as a neuroscientist specialising in auditory learning and memory, I find them especially intriguing. So I had high expectations of Musicophilia, the latest offering from neurologist and prolific author Oliver Sacks.And I confess to feeling a little guilty reporting that my reactions to the book are mixed.

B Sacks himself is the best part of Musicophilia. He richly documents his own life in the book and reveals highly personal experiences. The photograph of him on the cover of the book-which shows him wearing headphones, eyes closed, clearly enchanted as he listens to Alfred Brendel perform Beethoven's Pathetique Sonata-makes a positive impression that is borne out by the contents of the book. Sacks's voice throughout is steady and erudite but never pontifical. He is neither self-conscious nor self-promoting.

C The preface gives a good idea of what the book will deliver. In it Sacks explains that he wants to convey the insights gleaned from the "enormous and rapidly growing body of work on the neural underpinnings of musical perception and imagery, and the complex and often bizarre disorders to which these are prone." He also stresses the importance of "the simple art of observation" and "the richness of the human context." He wants to combine "observation and description with the latest in technology," he says, and to imaginatively enter into the experience of his patients and subjects. The reader can see that Sacks, who has been practicing neurology for 40 years, is torn between the "old-fashioned" path of observation and the new fangled, high-tech approach: He knows that he needs to take heed of the latter, but his heart lies with the former.

D The book consists mainly of detailed descriptions of cases, most of them involving patients whom Sacks has seen in his practice. Brief discussions of contemporary neuroscientific reports are sprinkled liberally throughout the text. Part, "Haunted by Music," begins with the strange case of Tony Cicoria, a nonmusical, middle-aged surgeon who was consumed by a love of music after being hit by lightning. He suddenly began to crave listening to piano music, which he had never cared for in the past. He started to play the piano and then to compose music, which arose spontaneously in his mind in a "torrent" of notes. How could this happen? Was the cause psychological? (He had had a near-death experience when the lightning struck him.) Or was it the direct result of a change in the auditory regions of his cerebral cortex? Electroencephalography (EEG) showed his brain waves to be normal in the mid-1990s, just after his ,trauma and subsequent "conversion" to music. There are now more sensitive tests, but Cicoria, has declined to undergo them; he does not want to delve into the causes of his musicality. What a shame!

E Part II, "A Range of Musicality," covers a wider variety of topics, but unfortunately, some of the chapters offer little or nothing that is new. For example, chapter 13, which is five pages long, merely notes that the blind often have better hearing than the sighted. The most interesting chapters are those that present the strangest cases. Chapter 8 is about "amusia," an inability to hear sounds as music, and "dysharmonia," a highly specific impairment of the ability to hear harmony, with the ability to understand melody left intact. Such specific "dissociations" are found throughout the cases Sacks recounts.

F To Sacks's credit, part III, "Memory, Movement and Music," brings us into the underappreciated realm of music therapy. Chapter 16 explains how "melodic intonation therapy" is being used to help expressive aphasic patients (those unable to express their thoughts verbally following a stroke or other cerebral incident) once again become capable of fluent speech. In chapter 20, Sacks demonstrates the near-miraculous power of music to animate Parkinson's patients and other people with severe movement disorders, even those who are frozen into odd postures. Scientists cannot yet explain how music achieves this effect

G To readers who are unfamiliar with neuroscience and music behavior, Musicophilia may be something of a revelation. But the book will not satisfy those seeking the causes and implications of the phenomena Sacks describes. For one thing, Sacks appears to be more at ease discussing patients than discussing experiments. And he tends to be rather uncritical in accepting scientific findings and theories.

H It's true that the causes of music-brain oddities remain poorly understood. However, Sacks could have done more to draw out some of the implications of the careful observations that he and other neurologists have made and of the treatments that have been successful. For example, he might have noted that the many specific dissociations among components of music comprehension, such as loss of the ability to perceive harmony but not melody, indicate that there is no music center in the brain. Because many people who read the book are likely to believe in the brain localisation of all mental functions, this was a missed educational opportunity.

I Another conclusion one could draw is that there seem to be no "cures" for neurological problems involving music. A drug can alleviate a symptom in one patient and aggravate it in another, or can have both positive and negative effects in the same patient. Treatments mentioned seem to be almost exclusively antiepileptic medications, which "damp down" the excitability of the brain in general; their effectiveness varies widely.

J Finally, in many of the cases described here the patient with music-brain symptoms is reported to have "normal" EEG results. Although Sacks recognises the existence of new technologies, among them far more sensitive ways to analyze brain waves than the standard neurological EEG test, he does not call for their use. In fact, although he exhibits the greatest compassion for patients, he conveys no sense of urgency about the pursuit of new avenues in the diagnosis and treatment of music-brain disorders. This absence echoes the book's preface, in which Sacks expresses fear that "the simple art of observation may be lost" if we rely too much on new technologies. He does call for both approaches, though, and we can only hope that the neurological community will respond.

Questions 27-30

Choose the correct letter，A, B, C or D.

Write the correct letter in boxes 27-30 on your answer sheet

27 Why does the writer have a mixed feeling about the book?

A The guilty feeling made him so.

B The writer expected it to be better than it was.

C Sacks failed to include his personal stories in the book.

D This is the only book written by Sacks.

28 What is the best part of the book?

A the photo of Sacks listening to music

B the tone of voice of the book

C the autobiographical description in the book

D the description of Sacks's wealth

29 In the preface, what did Sacks try to achieve?

A make a herald introduction of the research work and technique applied

B give detailed description of various musical disorders

C explain how people understand music

D explain why he needs to do away with simple observation

30 What is disappointing about Tony Cicoria's case?

A He refuses to have further tests.

B He can't determine the cause of his sudden musicality.

C He nearly died because of the lightening.

D His brain waves were too normal to show anything.

Questions 31-36

Do the following statements agree with the views of the writer in Reading Passage 3?

In boxes 31-36 on your answer sheet, write

YES	*if the statement agrees with the views of the writer*
NO	*if the statement contradicts with the views of the writer*
NOT GIVEN	*if it is impossible to say what the writer thinks about this*

31 It is difficult to give a well-reputable writer a less than totally favorable review.

32 Beethoven's Pathetique Sonata is a good treatment for musical disorders.

33 Sacks believes technological methods is of little importance compared with traditional observation when studying his patients.

34 It is difficult to understand why music therapy is undervalued

35 Sacks held little skepticism when borrowing other theories and findings in describing reasons and notion for phenomena he depicts in the book.

36 Sacks is in a rush to use new testing methods to do treatment for patients.

Complete each sentence with the correct ending, A-F, below.
Write correct letter, A-F, in boxes 37-40 on your answer sheet.

37 The content covered dissociations in understanding between harmony and melody

38 The study of treating musical disorders

39 The EEG scans of Sacks 's patients

40 Sacks believes testing based on new technologies

A **show no music-brain disorders.**

B **indicates that medication can have varied results.**

C **is key for the neurological community to unravel the mysteries.**

D **should not be used in isolation.**

E **indicate that not everyone can receive good education.**

F **show a misconception that there is function centre localized in the brain**