

IELTS Recent Actual Test With Answers Volume 4 Reading Practice Test 4

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READING PASSAGE 1

You should spend about 20 minutes on **Questions 1-13**, which are based on Reading Passage 1 below.



T-Rex: Hunter or Scavenger?

Jack Homer is an unlikely academic: his dyslexia is so bad that he has trouble reading a book. But he can read the imprint of life in sandstone or muddy shale across a distance of I00 years, and it is this gift that has made him curator of palaeontology at Montana State University's Museum of the Rockies, the leader of a multi-million dollar scientific project to expose a complete slice of life 68 million years ago, and a consultant to Steven Spielberg and other Hollywood figures.

His father had a sand and gravel quarry in Montana, and the young Horner was a collector of stones and bones, complete with notes about when and where he found them. "My father had owned a ranch when he was younger, in Montana," he says. "He was enough of a geologist, being a sand and gravel man, to have a pretty good notion that they were dinosaur bones. So when I was eight years old he took me back to the area that had been his ranch, to where he had seen these big old bones. I picked up one. I am pretty sure it was the upper arm bone of a duckbilled dinosaur: it probably wasn't a duckbilled dinosaur but closely related to that. I catalogued it, and took good care of it, and then later when I was in high school; excavated my first dinosaur skeleton. It obviously started earlier than eight and I literally have been driven ever since. I feel like I was born this way."

Horner spent seven years at university, but never graduated. "I have a learning disability, I would call it a learning difference - dyslexia, they call it - and I just had a terrible time with English and foreign languages and things like that. For a degree in geology or biology they required two years of a foreign language. There was no way in the world I could do that. In fact, I didn't really pass English. So I couldn't get a degree, I just wasn't capable of it. But I took all of the courses required and I wrote a thesis and I did all sorts of things. So I have the education, I just don't have the piece of paper," he says.

"We definitely know we are working on a very broad coastal plain with the streams and rivers bordered by conifers and hardwood plants, and the areas in between these rivers were probably fern-covered. There were no grasses at all: just ferns and bushes -an unusual landscape, kind of taking the south-eastern United States - Georgia, Florida - and mixing it with the moors of England and flattening it out," he says. "Triceratops is very common: they are the

cows of the Cretaceous, they are everywhere. Duckbilled dinosaurs are relatively common but not as common as triceratops and T-rex, for a meat-eating dinosaur, is very common. What we would consider the predator-prey ratio seems really off the scale. What is interesting is the little dromaeosaurs, the ones we know for sure were good predators, are haven't been found."

That is why he sees T-rex not as the lion of the Cretaceous savannah but its vulture. "Look at the wildebeest that migrate in the Serengeti of Africa, a million individuals lose about 200,000 individuals in that annual migration. There is a tremendous carrion base there. And so you have hyenas, you have tremendous numbers of vultures that are scavenging, you don't have all that many animals that are good predators. If T-rex was a top predator, especially considering how big it is, you'd expect it to be extremely rare, much rarer than the little dromaeosaurs, and yet they are everywhere, they are a dime a dozen," he says. A 12-tonne T-rex is a lot of vulture, but he doesn't see the monster as clumsy. He insisted his theory and finding, dedicated to further research upon it, of course, he would like to reevaluate if there is any case that additional evidence found or explanation raised by others in the future.

He examined the leg bones of the T-rex, and compared the length of the thigh bone (upper leg), to the shin bone (lower leg). He found that the thigh bone was equal in length or slightly longer than the shin bone, and much thicker and heavier, which proves that the animal was built to be a slow walker rather than fast running. On the other hand, the fossils of fast hunting dinosaurs always showed that the shin bone was longer than the thigh bone. This same truth can be observed in many animals of today which are designed to run fast: the ostrich, cheetah, etc.

He also studied the fossil teeth of the T-rex, and compared them with the teeth of the Velociraptor, and put the nail in the coffin of the "hunter T-rex theory". The Velociraptor's teeth which like stake knives: sharp, razor-edged, and capable of tearing through flesh with ease. The T-rex's teeth were huge, sharp at their tip, but blunt, propelled by enormous jaw muscles, which enabled them to only crush bones.

With the evidence presented in his documentary, Horner was able to prove that the idea of the T-rex as being a hunting and ruthless killing machine is probably just a myth. In light of the scientific clues he was able to unearth, the T-rex was a slow, sluggish animal which had poor vision, an extraordinary sense of smell, that often reached its "prey" after the real hunters were done feeding, and sometimes it had to scare the hunters away from a corpse. In order to do that, the T-rex had to have been ugly, nasty-looking, and stinky. This is actually true of nearly all scavenger animals. They are usually vile and nasty looking.

Questions 1-7

Do the following statements agree with the information given in Reading Passage 1?

In boxes 1-7 on your answer sheet, write

TRUE	if the statement agrees with the information
FALSE	if the statement contradicts the information
NOT GIVEN	If there is no information on this
1	
Jack Horner knew exac	tly the bone picked up in his father's ranch belonged to a
certain dinosaur when	he was at the age of 8.
2	
Jack Horner achieved a	distinctive degree in university when he graduated.
3 ▼ Jack Horner believes th	at the number of prey should be more than that of predators.
4 ▼ T-rex's number is equiva	lent to the number of vulture in the Serengeti.
	ne hypothesis that T-flicts with the fact of predator-prey ratio which Jack found.
6 Jack Horner refused to	accept any other viewpoints about T-rex's theory.
7 🔻 Jao	ck Horner is the first man that discovered T-

Questions 8-13

rex's bones in the world.

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 8-13 on your answer sheet.

Jack Horner found that T-rex's 8 is shorter than the thigh bone, which demonstrated that it was actually a 9 , unlike other swift animals such as ostrich or 10 that was built to 11 . Another explanation support his idea is that T-rex's teeth were rather 12 , which only allowed T-rex to 13 hard bones instead of tearing flesh

like Velociraptor.

READING PASSAGE 2

You should spend about 20 minutes on **Questions 14-27**, which are based on Reading Passage 2 below.



Leaf-Cutting Ants and Fungus

A The ants and their agriculture have been extensively studied over the years, but the recent research has uncovered intriguing new findings about the fungus they cultivate, how they domesticated it and how they cultivate it and preserve it from pathogens ([1]]). For example, the fungus farms, which the ants were thought to keep free of pathogens, turn out to be vulnerable to a devastating mold, found nowhere else but in ants'nests. To keep the mold in check, the ants long ago made a discovery that would do credit to any pharmaceutical laboratory.

B Leaf-cutting ants and their fungus farms are a marvel of nature and perhaps the best known example of symbiosis, the mutual dependence of two species. The ants'achievement is remarkable --the biologist Edward O. Wilson has called it "one of the major breakthroughs in animal evolution"--because it allows them to eat, courtesy of their mushroom's digestive powers, the otherwise poisoned harvest of tropical forests whose leaves are laden with terpenoids, alkaloids and other chemicals designed to sicken browsers.

C Fungus growing seems to have originated only once in evolution, because all gardening ants belong to a single tribe, the descendants of the first fungus farmer. There are more than 200 known species of the attine ant tribe, divided into 12 groups, or genera. The leaf-cutters use fresh vegetation; the other groups, known as the lower attines because their nests are smaller and their techniques more primitive, feed their gardens with detritus like dead leaves, insects and feces.

D The leaf-cutters'fungus was indeed descended from a single strain, propagated clonally, or just by budding, for at least 23 million years. But the lower attine ants used different varieties of the fungus, and in one case a quite separate species, the four biologists discovered. The pure strain of fungus grown by the leaf-cutters, it seemed to Mr. Currie, resembled the monocultures of various human crops, that are very productive for a while and then succumb to some disastrous pathogen, such as the Irish potato blight. Monocultures, which lack the genetic diversity to respond to changing environmental threats, are sitting ducks for parasites. Mr. Currie felt there had to be aparasite in the antfungus system. But a century of ant research offered no support for the idea. Textbooks describe how leaf-cutter ants scrupulously weed their gardens of all foreign organisms. "People kept telling me, 'You know the ants keep their gardens free of parasites, don't you?' "Mr. Currie said of his efforts to find a hidden interloper.

E But after three years of sifting through attine ant gardens, Mr. Currie discovered they are far from free of infections. In last month's issue of the Proceedings of the National Academy of Sciences, he and two colleagues, Dr. Mueller and David Mairoch, isolated several alien organisms, particularly a family of parasitic molds called Escovopsis.

F Escovopsis turns out to be a highly virulent pathogen that can devastate a fungus garden in a couple of days. It blooms like a white cloud, with the garden dimly visible underneath. In a day or two the whole garden is enveloped. "Other ants won't go near it and the ants associated with the garden just starve to death,"Dr. Rehner said. "They just seem to give up, except for those that have rescued their larvae."The deadly mold then turns greenishbrown as it enters its spore-forming stage.

G Evidently the ants usually manage to keep Escovopsis and other parasites undercontrol. But with any lapse in control, or if the ants are removed, Escovopsis will quickly burst forth. Although new leaf-cutter gardens start off free of Escovopsis, within two years some 60 percent become infected. The discovery of Escovopsis's role brings a new level of understanding to the evolution of the attine ants. "In the last decade, evolutionary biologists have been increasingly aware of the role of parasites as driving forces in evolution,"Dr. Schultz said. There is now a possible reason to explain why the lower attine species keep changing the variety of fungus in their mushroom gardens, and occasionally domesticating new ones—to stay one step ahead of the relentless Escovopsis.

H Interestingly, Mr. Currie found that the leaf-cutters had in general fewer alien molds in their gardens than the lower attines, yet they had more Escovopsis infections. It seems that the price they pay for cultivating a pure variety of fungus is a higher risk from Escovopsis. But the leaf-

cutters may have little alternative: they cultivate a special variety of fungus which, unlike those grown by the lower attines, produces nutritious swollen tips for the ants to eat.

I Discovery of a third partner in the ant-fungus symbiosis raises the question of how the attine ants, especially the leaf-cutters, keep this dangerous interloper undercontrol. Amazingly enough, Mr. Currie has again provided the answer. "People have known for a hundred years that ants have a whitish growth onthe cuticle," said Dr. Mueller, referring to the insects'body surface. "People wouldsay this is like a cuticular wax. But Cameron was the first one in a hundred years to put these things under a microscope. He saw it was not inertwax. It is alive." Mr. Currie discovered a specialized patch on the ants'cuticle that harbors a particular kind of bacterium, one well known to the pharmaceutical industry, because it is the source of half the antibiotics used in medicine. From each of 22 species of attine ant studied, Mr. Cameron and colleagues isolated a species of Streptomyces bacterium, they reported in Nature in April. The Streptomyces does not have much effect on ordinary laboratory funguses. But it is a potent poisoner of Escovopsis, inhibiting its growth and suppressing spore formation. It also stimulates growth of the ants'mushroom fungus. The bacterium is carried by virgin queens when they leave to establish new nests, but is not found on male ants, playboys who take no responsibility in nest-making or gardening.

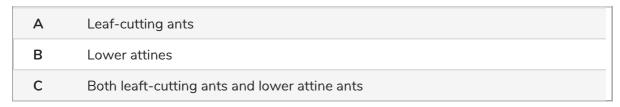
J Because both the leaf-cutters and the lower attines use Streptomyces, the bacterium may have been part of their symbiosis for almost as long as the Escovopsis mold. If so, some Alexander Fleming of an ant discovered antibiotics millions of years before people did. Even now, the ants are accomplishing two feats beyond the powers of human technology. The leafcutters are growing a monocultural crop year after year without disaster, and they are using an antibiotic apparently so wisely and prudently that, unlike people, they are not provoking antibiotic resistance in the target pathogen.

Questions 14-19

Use the information in the passage to match the options (listed A-C) with activities or features of ants below.

Write the appropriate letters A-C in boxes 14-19 on your answer sheet

NB you may use any letter more than once



can use toxic leaves to feed fungus

15	build small nests and live with different foreign fungus
16	use dead vegetation to feed fungus
17	raise a single fungus which do not live with other variety of
foreigners	
18	normally keep a highly dangerous parasite under control
19	use special strategies to fight against Escovopsis
Questions 20	-24
The reading Passag	ge has ten paragraphs A-J .
Which paragraph c	contains the following information?
Write the correct le	etter A-J, in boxes 20-24 on your answer sheet.
20 🔻	Dangerous outcome of Escovopsis.
21 🔽	Risk of growing single fungus.
22 🔻	Comparison of features of two different nests for feeding
gardens.	
23	Discovery of significant achievements made by ants earlier than
human.	
24	Advantage of growing new breed of fungus in the ant farm.

Questions 25-26

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

Write your answers in boxes 25-26 on your answer sheet.

25 How does the author think of Currie's opinion on the saying "ants keep their gardens free of parasites"?

- A C his viewpoint was verified later.
- B C his earlier study has sufficient evidence immediately.

- **C** there is no details mentioned in the article.
- **D** his opinion was proved to be wrong later on.

26 What did scientists find on the skin of ants under microscope?

- A C some white cloud mold embed in their skin
- B C that wax is all over their skin
- **C** a substance which is useful to humans
- **D** a substance which suppresses growth of fungus.

READING PASSAGE 3

You should spend about 20 minutes on **Questions 27-40**, which are based on Reading Passage 3 below.



Stealth Forces in weight Loss

The field of weight loss is like the ancient fable about the blind men and the elephant. Each man investigates a different part of the animal and reports back, only to discover their findings are bafflingly incompatible.

A. The various findings by public-health experts, physicians, psychologists, geneticists, molecular biologists, and nutritionists are about as similar as an elephant's tusk is to its tail. Some say obesity is largely predetermined by our genes and biology; others attribute it to an overabundance of fries, soda, and screensucking; still others think we're fat because of viral infection, insulin, or the metabolic conditions we encountered in the womb. "Everyone subscribes to their own little theory," says Robert Berkowitz, medical director of the Center for Weight and Eating Disorders at the University of Pennsylvania School of Medicine. We're programmed to hang onto the fat we have, and some people are predisposed to create and carry more fat than others. Diet and exercise help, but in the end the solution will inevitably be more complicated than pushing away the plate and going for a walk. "It's not as simple as 'You're fat because you're lazy,'" says Nikhil Dhurandhar, an associate professor at Pennington Biomedical Research Center in Baton Rouge. "Willpower is not a prerogative of thin people. It's distributed equally."

B. Science may still be years away from giving us a miracle formula for fat-loss. Hormone leptin is a crucial player in the brain's weight-management circuitry. Some people produce too little leptin; others become desensitized to it. And when obese people lose weight, their leptin levels plummet along with their metabolism. The body becomes more efficient at using fuel and conserving fat, which makes it tough to keep the weight off. Obese dieters' bodies go into a state of chronic hunger, a feeling Rudolph Leibel, an obesity researcher at Columbia University, compares to thirst. "Some people might be able to tolerate chronic thirst, but the majority couldn't stand it," says Leibel. "Is that a behavioral problem - a lack of

willpower? I don't think so."

C. The government has long espoused moderate daily exercise - of the evening-walk or takethe-stairs variety - but that may not do much to budge the needle on the scale. A 150-pound person burns only 150 calories on a half-hour walk, the equivalent of two apples. It's good for the heart, less so for the gut. "Radical changes are necessary," says Deirdre Barrett, a psychologist at Harvard Medical School and author of Waistland. "People don't lose weight by choosing the small fries or taking a little walk every other day." Barrett suggests taking a cue from the members of the National Weight Control Registry (NWCR), a self-selected group of more than 5,000 successful weight-losers who have shed an average of 66 pounds and kept it off 5.5 years. Some registry members lost weight using low-carb diets; some went low-fat; others eliminated refined foods. Some did it on their own; others relied on counseling. That said, not everyone can lose 66 pounds and not everyone needs to. The goal shouldn't be getting thin, but getting healthy. It's enough to whittle your weight down to the low end of your set range, says Jeffrey Friedman, a geneticist at New York's Rockefeller University. Losing even 10 pounds vastly decreases your risk of diabetes, heart disease, and high blood pressure. The point is to not give up just because you don't look like a swimsuit model.

D. The negotiation between your genes and the environment begins on day one. Your optimal weight, writ by genes, appears to get edited early on by conditions even before birth, inside the womb. If a woman has high blood-sugar levels while she's pregnant, her children are more likely to be overweight or obese, according to a study of almost 10,000 mother-child pairs. Maternal diabetes may influence a child's obesity risk through a process called metabolic imprinting, says Teresa Hillier, an endocrinologist with Kaiser Permanente's Center for Health Research and the study's lead author. The implication is clear: Weight may be established very early on, and obesity largely passed from mother to child. Numerous studies in both animals and humans have shown that a mother's obesity directly increases her child's risk for weight gain. The best advice for moms-to-be: Get fit before you get pregnant. You'll reduce your risk of complications during pregnancy and increase your chances of having a normal-weight child.

E. It's the \$64,000 question: Which diets work? It got people wondering: Isn't there a better way to diet? A study seemed to offer an answer. The paper compared two groups of adults: those who, after eating, secreted high levels of insulin, a hormone that sweeps blood sugar out of the bloodstream and promotes its storage as fat, and those who secreted less. Within each group, half were put on a low-fat diet and half on a low-glycemic-load diet. On average, the low-insulin- secreting group fared the same on both diets, losing nearly 10 pounds in the first six months - but they gained about half of it back by the end of the 18-month study. The high-insulin group didn't do as well on the low-fat plan, losing about 4.5 pounds, and gaining back more than half by the end. But the most successful were the high- insulin-secretors on the low-glycemic-load diet. They lost nearly 13 pounds and kept it off.

F. What if your fat is caused not by diet or genes, but by germs - say, a virus? It sounds like

a sci-fi horror movie, but new research suggests some dimension of the obesity epidemic may be attributable to infection by common viruses, says Dhurandhar. The idea of "infectobesity" came to him 20 years ago when he was a young doctor treating obesity in Bombay. He discovered that a local avian virus, SMAM-1, caused chickens to die, sickened with organ damage but also, strangely, with lots of abdominal fat. In experiments, Dhurandhar found that SMAM-1-infected chickens became obese on the same diet as uninfected ones, which stayed svelte.

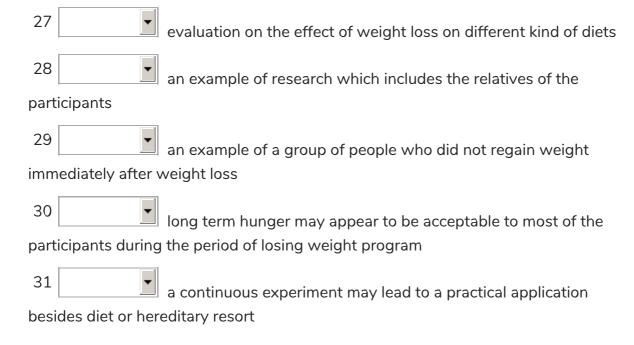
G. He later moved to the U.S. and onto a bona fide human virus, adenovirus 36 (AD-36). In the lab, every species of animal Dhurandhar infected with the virus became obese - chickens got fat, mice got fat, even rhesus monkeys at the zoo that picked up the virus from the environment suddenly gained 15 percent of their body weight upon exposure. In his latest studies, Dhurandhar has isolated a gene that, when blocked from expressing itself, seems to turn off the virus's fattening power. Stem cells extracted from fat cells and then exposed to AD-36 reliably blossom into fat cells - but when stem cells are exposed to an AD-36 virus with the key gene inhibited, the stems cells don't differentiate. The gene appears to be necessary and sufficient to trigger AD-36-related obesity, and the goal is to use the research to create a sort of obesity vaccine.

Questions 27-31

Reading Passage has seven sections, A-G. Which section contains the following information?

Write the correct letter, A-G, in boxes 17-31 on your answer sheet.

NB You may use any letter more than once.



Questions 32-36

Look at the following researchers and the list of findings below.

Match each researcher with the correct finding.

Write the correct letter in boxes 32-36 on your answer sheet.

NB You may use any letter more than once

Α	Robert Berkowitz
В	Rudolph Leibel
С	Nikhil Dhurandhar
D	Deirdre Barrett
E	Jeffrey Friedman
F	Teresa Hillier

A person's weight is predetermined by the interaction of his/her DNA and the environment

Pregnant mothers who are overweight may risk their fetus in gaining weight.

The aim of losing weight should be keeping healthy rather than being attractive.

Small changes in lifestyle will not help in reducing much weight.

Researchers should be divided into different groups with their own point of view about weight loss.

Questions 37-40

Complete the summery below. Choose **NO MORE THAN ONE WORD** from the passage for each answer.

Write your answers in boxes 37-40 on your answer sheet.

In Bombay Clinic, a young doctor who came up with the concept 'infectobesity' believed that				
the obesity is caused by a kind of virus. For years, he conducted experiments on				
37	. Finally, later as he moved to America, he identified a new virus named			
38	which proved to be a significant breakthrough inducing more weight.			
Although there seems no way to eliminate the virus still now, a kind of 39 can be				
separated as to block the effectiveness of the virus. In the future, the doctor future is aiming				
at developing a new 40 which might effectively combat against the virus				

Solution:

- 1 TRUE
- 3 TRUE
- 5 TRUE
- 7 NOT GIVEN
- 9 slow walker
- 11 run fast
- 13 (only) crush
- **15** B
- 17 A
- 19

- 2 FALSE
- 4 NOT GIVEN
- 6 FALSE
- 8 shin bone
- 10 cheetah
- 12 blunt
- 14 A
- **16** B
- 18 A
- **20** F

D

C

.

G

A

C

E

D

C

B

G

F

F

34 E

D

A

37 chickens

38 adenovirus/AD-36

Gene

40 Vaccine