3 次の文を読み，設問A～Fに答えなさい。なお，星印(*)のついた単語には文末に訳注があります。

Every so often, a strain* of influenza* unfamiliar in humans suddenly begins passing from person to person. Because the virus is so unusual, few if any people have built-in immunity from past exposures. Even the vaccinated* have no defense; flu* shots shield against influenza variants that health experts have anticipated will be active in a given flu season, not against other, unforeseen kinds. Finding no deterrent*, the new strain spreads unabated*, causing illness — and death — on a global scale.

The worst worldwide epidemic*, or pandemic, on record struck in 1918 and killed more than 20 million people, sometimes within hours after the first symptoms* appeared. This disaster*, traced to the so-called Spanish influenza virus, was followed by epidemics of Asian flu in 1957, Hong Kong flu in 1968 and Russian flu in 1977. (The names reflect popular impressions of where the pandemics began, although all four episodes, and perhaps most others, are now thought to have originated in China.)

Public health experts warn that another pandemic can strike any time now and that it could well be as vicious* as the 1918 episode. In 1997, when a lethal influenza variant afflicted 18 people in Hong Kong, contributing to the death of six, officials feared the next wave had begun. Authorities in the region managed to contain the problem quickly, however, by finding the source — infected chickens, ducks and geese — and then destroying all the poultry* in Hong Kong.

Next time, humankind may not be so fortunate. If a virus as deadly as that Hong Kong strain tore* through the world's crowded communities today, 30 percent of the earth's population could

conceivably be dead (from the virus itself or from secondary bacterial infections) before a vaccine became available to protect those who initially managed to escape infection. Vaccines against any given influenza variant take about six months to produce, test for safety and distribute — too long to do much good in the face of a fast-moving pandemic.

If the feared pandemic does not materialize until next year or beyond, though, new methods for limiting sickness and death could be available. Later this year two drugs being tested in large clinical trials could be approved for sale as new missiles* in the fight against the flu. The agents — called zanamivir and GS 4104— show great promise for preventing influenza infections and for reducing the duration and severity of symptoms in people who begin treatment after they start to feel sick.

Unlike vaccines (which prime the immune system to prevent viruses from gaining a foothold* in the body) and unlike standard home remedies* (which ease symptoms but have no effect on the infection itself), these drugs have been designed to attack the influenza virus directly. They hobble* a critical viral enzyme, called neuraminidase*, and in so doing markedly reduce proliferation of the virus in the body. Additional neuraminidase inhibitors, not yet evaluated in humans, are under study as well.

As many people know, two anti-flu drugs, amantadine and rimantadine, are already on the market. But those agents, which work by a different mechanism, have serious flaws*. They can cause confusion and other neurological side effects, and they are ineffective against one of the two major influenza classes (type B) that afflict people. Moreover, influenza viruses seem to become resistant to the drugs fairly easily. Therefore, individuals treated in the first phases of an epidemic can spread a drug-resistant version of the virus to other

people, who will then prove unresponsive to the medicines. This last problem is particularly acute in "closed" communities, such as nursing homes.

The story of how the newer drugs were developed involves a wonderful combination of luck and logic. The breakthrough that led most immediately to their design was the deciphering*, in 1983, of neuraminidase's three-dimensional structure*. Yet it was a series of earlier discoveries that enabled scientists to realize that a specific part of the neuraminidase molecule was probably an Achilles' heel* for all influenza variants — a weakness that thoughtfully constructed drugs might exploit*.

One line of that early research uncovered some essential properties of influenza viruses and of their strategy for survival. Biologists have long known that viruses are basically genes wrapped in proteins that either protect the genes or help the viruses to reproduce in the body. Sometimes, as is true for influenza, these various constituents are further enveloped in a fatty (lipid) membrane. When any virus causes disease, it does so by invading selected cell types, replicating within those cells and then pouring out of the cells to infect others. Symptoms arise both because viral proliferation disrupts the colonized cells and because the immune system attempts to contain the infection, in the process causing local inflammation and systemic* aches and fever.

Scientists isolated an influenza strain from a human for the first time in 1933. Since then, they have learned that influenza viruses come in two main "flavors*" — types A and B — that differ in certain of their internal proteins. A third type (C) does not seem to cause serious disease.

Virologists* further group type A forms according to variations in two proteins that protrude* from the viral surface like spikes —

hemagglutinin* and neuraminidase (the enzyme that is the target of the new drugs). As is true of other proteins, these consist of folded strings of amino acids*. All hemagglutinin variants adopt essentially the same three-dimensional conformation, and all neuraminidase variants take on a characteristic shape. But within each group, the individual proteins can differ markedly in the sequence of their constituent amino acids. So far three hemagglutinin and two neuraminidase subtypes* have been identified on type A influenzas, which are named according to the hemagglutinin (H) and neuraminidase (N) molecules they display: H1N1, H1N2, H2N2 and so on.

Aside from their chemistry, type A and type B influenzas differ in their range of activity. Type B viruses infect only humans, and they cause regional epidemics rather than pandemics. Type A influenzas, in contrast, affect pigs, horses, seals, whales and birds as well as humans, although not all strains infect all species. They are also responsible for all of this century's pandemics.

In spite of their differences, both influenza types have the same basic life cycle. For a single copy of an influenza virus, or particle, to enter a human cell, hemagglutinin on the virus must link to a sugary* molecule, sialic acid*, on the surface of the cell. This binding induces the cell to engulf* the virus, which is initially sequestered* within a kind of bubble. Soon, though, the viral genes (consisting of strands of RNA) and internal proteins are freed, and they work their way into the cell nucleus.

There some of the viral proteins set about replicating the viral RNA strands and also constructing a form (called messenger RNA) that can be read out and translated into proteins by the cell's protein-making machinery. Eventually the newly made genes and proteins come together and bud from the cell as new viral particles.

Inconveniently for the virus, the emerging particles are coated with sialic acid, the very substance that binds influenza viruses to the cells they attempt to invade. If the sialic acid were allowed to remain on the virus and on the surface of a virus-making cell, hemagglutinin on the newly minted* particles would bind to the sialic acid, causing the particles to clump* together on the cell, like insects trapped on flypaper. So trapped, they would be unable to spread to other cells.

But the virus has an ace in the hole*. The neuraminidase molecules on the freshly made particles can cleave* sialic acid. In other words, the neuraminidase spikes essentially dissolve the unwanted sialic acid "glue," thereby enabling the viral particles to travel. The enzyme also helps the virus to plow* through the mucus* between cells in the airways.

(W.G. Laver ら著：Scientific American, 280 巻 1 号, 56–65頁, 1999 年から引用。その一部を改変)

訳 注

strain: 系統, 種類, 株

influenza: インフルエンザ

vaccinate: ワクチンを接種する

flu: インフルエンザの略, 風邪

deterrent: 妨げる

unabated: おさまらない

epidemic: 伝染病

symptom: 症状

disaster: 災害

vicious: はげしい

poultry: 家禽

tore: (tear の過去形)暴れまわる

missile：ミサイル

foothold：足がかり

remedy：治療(法)

hobble：妨げる

neuraminidase：ノイラミニダーゼ(酵素のひとつ)

flaw：弱点

decipher：解読する

three-dimensional structure：3次元構造

Achilles' heel：アキレスのかかと，唯一の弱点

exploit：利用する

systemic：全身の

flavor：特徴，特質

virologist：ウイルス学者

protrude：突き出す

hemagglutinin：(赤)血球凝集素

amino acid：アミノ酸

subtype：亜型

sugary：糖の

sialic acid：シアル酸(ノイラミン酸)(糖のひとつ)

engulf：取り込む，巻き込む

sequester：隔離する

mint：造りだす

clump：凝集させる

an ace in the hole：最後の切り札

cleave：切り裂く

plow (through)：かきわけて進む

mucus：粘液

設　問

A：次の表の空欄［　　］にあてはまる日本語または数字を答案用紙［3—1］のA欄(ア)～(ク)に記入しなさい。

年(西暦)		風邪の名称など	備　考
［ア］	年	スペイン風邪	死者数2000万人
［イ］	年		インフルエンザウイルスをヒトから分離
1957	年	［ウ］	
［エ］	年	香港風邪	
1977	年	［オ］	
［カ］	年		ノイラミニダーゼの3次元構造解明
［キ］	年	香港で発生	死者数［ク］人

B：zanamivir やＧＳ4104がワクチンや一般の風邪薬と違う点をひとつ，答案用紙［3—1］のB欄に日本語25字以内で記入しなさい。

C：zanamivir やＧＳ4104のインフルエンザウイルス攻撃方法を，答案用紙［3—1］のC欄に日本語50字以内で説明しなさい。

D：amantadine や rimantidine について，抗インフルエンザ薬としての欠点3つを，答案用紙［3—2］のD欄(ア)，(イ)および(ウ)にそれぞれ日本語25字以内で記入しなさい。

E：インフルエンザは大流行することがあります。その理由について，あなたの考えを答案用紙［3—2］のE欄に日本語50字以内で記入しなさい。

F：本文中の命名法にしたがって，A型インフルエンザウイルスの分類名6つを，答案用紙［3—2］のF欄(ア)～(カ)に記入しなさい。