2023年度
大学院理工学研究科【環境共生工学専攻】博士後期課程
一般選抜試験(第Ⅱ期)問題

英　　語

開始時刻　午前10時00分
終了時刻　午前11時00分

【注意事項】
1．答案用紙には、氏名を必ず記入してください。
2．試験終了後、答案用紙は必ず提出してください（問題用紙は提出しなくてよい）。
<u>3．問題番号が明記された答案用紙を使用し、解答してください。</u>

1 以下の文章に基づいて続く各問に日本語で解答しなさい。

When corn farmers harvest their crop, they often leave the stalks, leaves and spent cobs to rot in the fields. Now, engineers have fashioned a new strain of yeast that can convert this inedible debris into ethanol, a biofuel. If the process can be scaled up, this largely untapped renewable energy source could help reduce reliance on fossil fuels.

Previous efforts to convert this fibrous material, called corn stover, into fuel met with limited success. Before yeasts can do their job, corn stover must be broken down, but this process often generates by-products that kill yeasts. But by tweaking a gene in common baker's yeast, researchers have engineered a strain that can defuse those deadly by-products and get on with the job of turning sugar into ethanol.

The new yeast was able to produce over 100 grams of ethanol for every liter of treated corn stover, an efficiency comparable to the standard process using corn kernels to make the biofuel, the researchers report June 25 in Science Advances.

"They've produced a more resilient yeast," says Venkatesh Balan, a chemical engineer at the University of Houston not involved in the research. The new strain may benefit biofuel producers trying to harness materials like corn stover, he says.

In the United States, most ethanol is made from corn, the country's largest crop, and is mixed into most of the gasoline sold at gas stations. Corn ethanol is a renewable energy source, but it has limitations. Diverting corn to make ethanol can detract from the food supply, and expanding cropland just to plant corn for biofuel clears natural habitats. Converting inedible corn stover into ethanol could increase the biofuel supply without having to plant more crops.

"Corn can't really displace petroleum as a raw material for fuels," says metabolic engineer Felix Lam of MIT. "But we have an alternative."

Lam and colleagues started with *Saccharomyces cerevisiae*, or common baker's yeast. Like sourdough bakers and brewers, biofuel producers already use yeast: It can convert sugars in corn kernels into ethanol.

But unlike corn kernels with easy-access sugars, corn stover contains sugars bound in lignocellulose, a plant compound that yeast can't break down. Applying harsh acids can free these sugars, but the process generates toxic by-products called aldehydes that can kill yeasts.

But Lam's team had an idea — convert the aldehydes into something tolerable to yeast. The researchers already knew that by adjusting the chemistry of the yeast's growing environment, they could improve its tolerance to alcohol, which is also harmful at high concentrations. With that in mind, Lam and colleagues homed in on a yeast gene called *GRE2*, which helps convert aldehydes into alcohol. The team randomly generated about 20,000 yeast variants, each with a different, genetically modified version of *GRE2*. Then, the researchers placed the horde of variants inside a flask that also contained toxic aldehydes to see which yeasts would survive.

Multiple variants survived the gauntlet, but one dominated. With this battle-tested version of *GRE2*, the researchers found that the modified baker's yeast could produce ethanol from treated corn stover almost as efficiently as from corn kernels. What's more, the yeast could generate ethanol from other woody materials, including wheat straw and switchgrass. "<u>We have a single strain that can accomplish all this,</u>(a)" Lam says.

modified from source: Nikk Ogasa, JULY 7, 2021, Science News
www.sciencenews.org/article/genetically-modified-yeast-ethanol-cornstalk-biofuel

(1) ここで提案されている「バイオエタノール」の製法はどのようなものか説明しなさい。
(2) 米国におけるこれまでの「バイオエタノール」の製法はどのようなもので，その欠点はなんであったのか説明しなさい。
(3) *Saccharomyces cerevisiae* とはなにか説明しなさい。
(4) *GRE2* とはなにか説明しなさい。
(5) 下線部(a)の意義を説明しなさい。

2 以下の文章に基づいて続く各問に日本語で解答しなさい。

The Black Death May Not Have Been Spread By Rats After All

The Black Death ravaged Europe between 1347 and 1353, killing millions. Plague outbreaks in Europe then continued until the 19th century.

One of the most commonly recited facts about plague in Europe was that it was spread by rats. In some parts of the world, the bacterium that causes plague, *Yersinia pestis*, maintains a long-term presence in wild rodents and their fleas. This is called an <u>animal "reservoir"</u>(a).

While plague begins in rodents, it sometimes spills over to humans. Europe may have once hosted animal reservoirs that sparked plague pandemics. But plague could have also been repeatedly reintroduced from Asia. Which of these scenarios was present remains a topic of scientific controversy.

Our recent research, published in the Proceedings of the National Academy of Sciences (PNAS), has shown that environmental conditions in Europe would have prevented plague from surviving in persistent, long-term animal reservoirs. How, then, did plague persevere in Europe for so long?

Our study offers two possibilities. One, the plague was being reintroduced from Asian reservoirs. Second, there could have been short- or medium-term temporary reservoirs in Europe. In addition, the two scenarios might have been mutually supportive.

However, the rapid spread of the Black Death and subsequent outbreaks of the next few centuries also suggest slow-moving rats may not have played the critical role in transmitting the disease that is often portrayed.

European climate

To work out whether plague could survive in long-term animal reservoirs in Europe, we examined factors such as soil characteristics, climatic conditions, terrain types and rodent varieties. These all seem to affect whether plague can hold on in reservoirs.

For example, high concentrations of some elements in soil, including copper, iron, magnesium, as well as a high soil pH (whether it is acidic or alkaline), cooler temperatures, higher altitudes and lower rainfall appear to favor the development of persistent reservoirs, though it is not entirely clear why, at this stage.

Based on our comparative analysis, centuries-long wild rodent plague reservoirs were even less likely to have existed from the Black Death of 1348 to the early 19th century than today, when comprehensive research rules out any such reservoirs within Europe.

This contrasts sharply with regions across China and the western US, where all the above conditions for persistent *Yersinia pestis* reservoirs in wild rodents are found.

In central Asia, long-term and persistent rodent reservoirs may have existed for millennia. As ancient DNA and textual evidence hints, once plague crossed into Europe from central Asia, it appears to have seeded a short- or medium-term reservoir or reservoirs in European wild rodents. The most likely place for this to have been was in central Europe.

However, as local soil and climatic conditions did not favor long-term and persistent reservoirs, the disease had to be re-imported, at least in some instances. Importantly, the two scenarios are not mutually exclusive.

Radical difference

To go deeper into the role of rats in spreading plague in Europe, we can compare different outbreaks of the disease.

The first plague pandemic began in the early sixth century and lasted until the later eighth century. The second pandemic (which included the Black Death) began in the 1330s and lasted five centuries. A third pandemic began in 1894 and remains with us today in places such as Madagascar and California.

These pandemics overwhelmingly involved the bubonic form of plague, where the bacteria infect the human lymphatic system (which is part of the body's immune defenses). In pneumonic plague, the bacteria infect the lungs.

The plagues of the second pandemic differed radically in their character and transmission from more recent outbreaks. First, there were strikingly different levels of mortality, with some second pandemic outbreaks reaching 50 %, while those of the third pandemic rarely exceeded 1 %. In Europe, figures for the third pandemic were even lower.

Second, there were different rates and patterns of transmission between these two plague epochs. There were massive differences in the frequency and speed of transporting goods, animals, and people between the late middle ages and today (or the late 19th century). Yet the Black Death and many of its subsequent waves spread with astonishing speed. Over land, it raced almost as fast each day as the modern outbreaks do over a year.

As described by contemporary chroniclers, physicians, and others – and as reconstructed quantitatively from archival documents – the plagues of the second pandemic spread faster and more widely than any other disease during the middle ages. Indeed, they were faster than in any period until the cholera outbreaks from 1830 or the great influenza of 1918-20.

Regardless of how the various European waves of the second pandemic began, both wild and non-wild rodents – rats, first and foremost – move much slower than the pace of transmission around the continent.

Third, the seasonality of plague also shows wide discrepancies. Plagues of the third pandemic (except for the rare ones, principally of pneumonic plague) have closely followed the fertility cycles of rat fleas. These rise with relatively humid conditions (although lower rainfall is important for plague reservoirs to first become established) and within a temperature band between 10 °C and 25 °C.

By contrast, plagues of the second pandemic could cross winter months in bubonic form, as seen across the Baltic regions from 1709-13. But in Mediterranean climes, plague from 1348 through the 15th century was a summer contagion that peaked in June or July – during the hottest and driest months.

This deviates strikingly from plague seasons in these regions in the 20th century. Because of the low relative humidity and high temperatures, these months were then the least likely times for plague to break out among rats or humans.

These differences raise a crucial question about whether the bubonic form of the plague depended on slow-moving rodents for its transmission when instead it could spread much more efficiently directly, from person to person. Scientists have speculated that this could have occurred because of ectoparasites (fleas and possibly lice), or through people's respiratory systems and through touch.

Questions such as the precise roles played by humans and rats in past plague pandemics need further work to resolve. But as shown by this study, and others, major steps forward can be made when scientists and historians work together.

modified from source: S.Cohn and P.Slavin, Jan 20, 2023, IFLScience
www.iflscience.com/the-black-death-may-not-have-been-spread-by-rats-after-all-67193

(1) 下線部(a)の「animal reservoir」に適切な訳語を提案しなさい。

(2) 1330年代に始まるヨーロッパの「black death」を以下では「中世ヨーロッパの black death」と呼ぶ。これは何処からもたらされたと考えられているか。

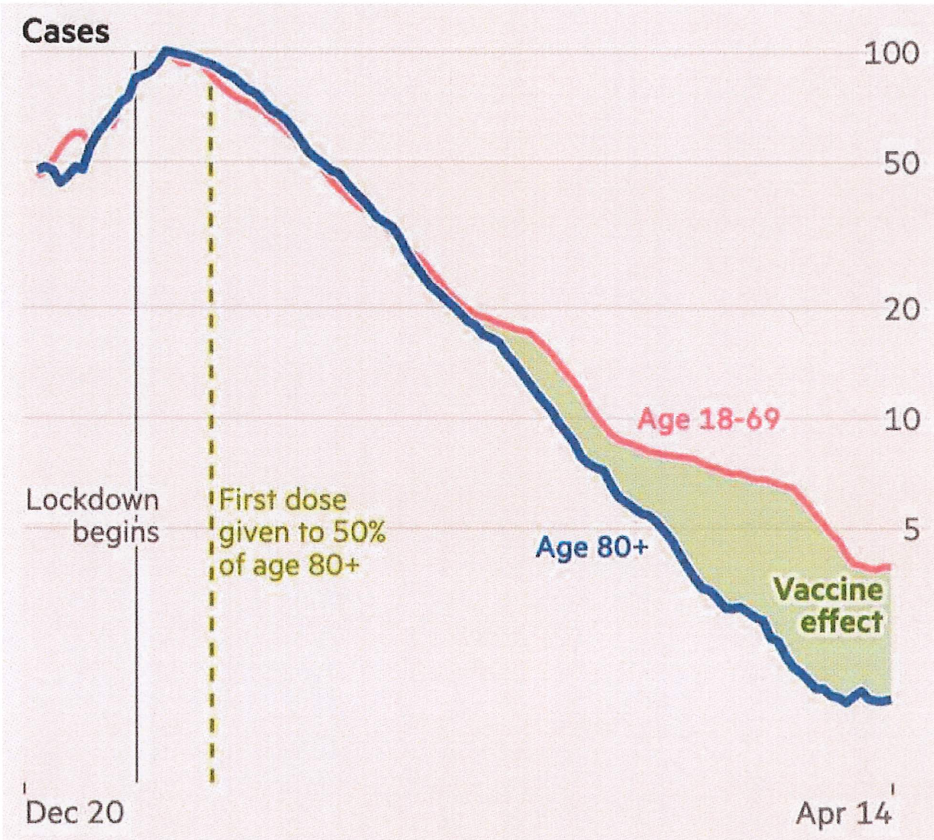
(3) この論考において中世ヨーロッパの black death の感染源が rat ではないとする根拠はなにか説明しなさい。

(4) この論考において中世ヨーロッパの black death の主要感染経路としてなにを提案しているか説明しなさい。

3 Answer the questions below in English.

1. Describe what facts are displayed in the figure, in details.
2. Give free discussion based on the information in the figure.

【注釈】図は，英国における COVID-19 感染状況についての 2020 年 12 月〜2021 年 4 月までのデータを示している。2020 年は各国における COVID-19 pandemic の始まりの年であり，英国では 2020 年 12 月 21 日から何度目かのロックダウンが宣言されている。他方，英国は世界に先駆けてワクチン接種を認可し実施した国であり，2020 年 12 月 8 日に高齢者及び医療関係者へのワクチン接種が開始された。
（この注釈部分を英訳する必要はありません）



Cases as a percentage of winter peak, by age group (log scale)

source: https://www.ft.com/content/d71729a3-72e8-490c-bd7e-757027f9b226