Study reveals rapid evolution and global spread of Pseudomonas aeruginosa

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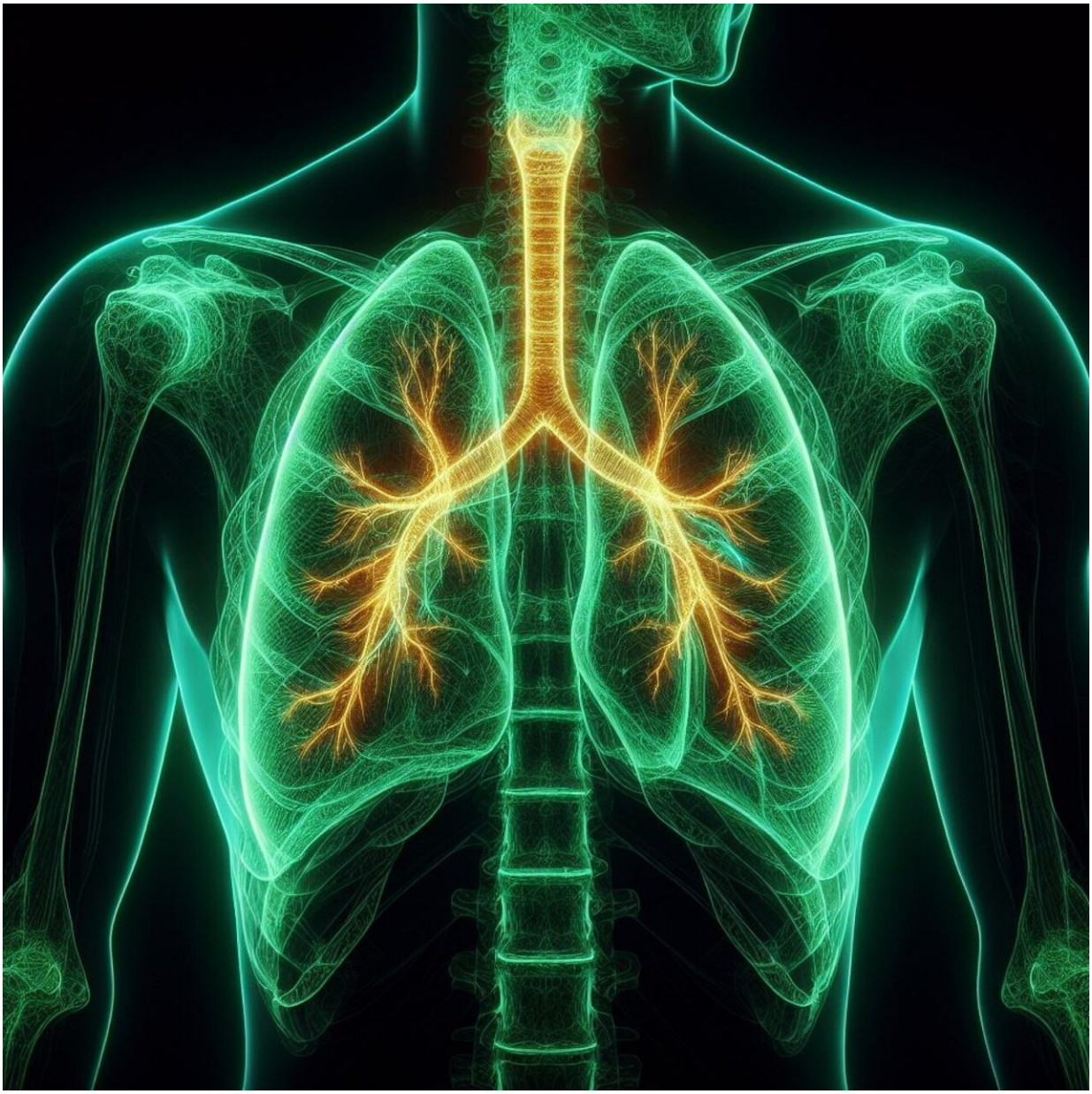

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Pseudomonas aeruginosa—an environmental bacteria that can cause devastating multidrug-resistant infections, particularly in people with underlying lung conditions—evolved rapidly and then spread globally over the last 200 years, probably driven by changes in human behavior, a new study has found.

The work appears in the journal *Science*.

P. aeruginosa is responsible for over 500,000 deaths per year around the world, of which over 300,000 are associated with antimicrobial resistance (AMR). People with conditions such as COPD (smoking-related lung damage), cystic fibrosis (CF), and non-CF bronchiectasis are particularly susceptible.

How P. aeruginosa evolved from an environmental organism into a specialized human pathogen was not previously known. To investigate this, an international team led by scientists at the University of Cambridge examined DNA data from almost 10,000 samples taken from infected individuals, animals, and environments around the world.

By mapping the data, the team was able to create phylogenetic trees—"family trees"—that show how the bacteria from the samples are related to each other. Remarkably, they found that almost seven in ten infections are caused by just 21 genetic clones, or "branches" of the family tree that have rapidly evolved (by acquiring new genes from neighboring bacteria) and then spread globally over the last 200 years.

This spread occurred most likely as a result of people beginning to live in densely-populated areas, where air pollution made our lungs more

susceptible to infection and where there were more opportunities for infections to spread.

These epidemic clones have an intrinsic preference for infecting particular types of patients, with some favoring CF patients and other non-CF individuals. It turns out that the bacteria can exploit a previously unknown immune defect in people with CF, allowing them to survive within macrophages. Macrophages are cells that "eat" invading organisms, breaking them down and preventing the infection from spreading. But a previously-unknown flaw in the immune systems of CF patients means that once the macrophage swallows P. aeruginosa, it is unable to get rid of it.

Having infected the lungs, these bacteria then evolve in different ways to become even more specialized for a particular lung environment. The result is that certain clones can be transmitted within CF patients and other clones within non-CF patients, but almost never between CF and non-CF patient groups.

Professor Andres Floto, Director of the UK Cystic Fibrosis Innovation Hub at the University of Cambridge and Royal Papworth Hospital NHS Foundation Trust, and senior author of the study said, "Our research on Pseudomonas has taught us new things about the biology of cystic fibrosis and revealed important ways we might be able to improve immunity against invading bacteria in this and potentially other conditions.

"From a clinical perspective, this study has revealed important information about Pseudomonas. The focus has always been on how easily this infection can spread between CF patients, but we've shown that it can spread with worrying ease between other patients, too. This has very important consequences for infection control in hospitals, where it's not uncommon for an infected individual to be on an open

ward with someone potentially very vulnerable.

"We are incredibly lucky at Royal Papworth Hospital where we have single rooms and have developed and evaluated a new air-handling system to reduce the amount of airborne bacteria and protect all patients."

Dr. Aaron Weimann from the Victor Phillip Dahdaleh Heart & Lung Research Institute at the University of Cambridge, and first author on the study, said, "It's remarkable to see the speed with which these bacteria evolve and can become epidemic and how they can specialize for a particular lung environment. We really need systematic, pro-active screening of all at risk patient groups to detect and hopefully prevent the emergence of more epidemic clones."

More information: Aaron Weimann et al, Evolution and host-specific adaptation of Pseudomonas aeruginosa, *Science* (2024). DOI: 10.1126/science.adi0908. www.science.org/doi/10.1126/science.adi0908

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