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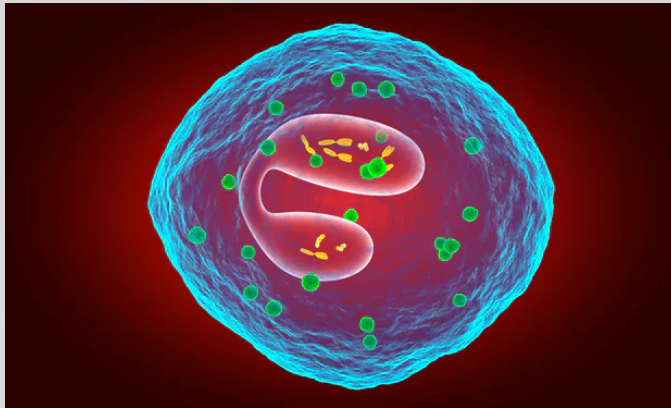
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Scientists are now looking at new ways to understand asthma – a complex respiratory disease that afflicts over 5 million people in the UK



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▲ Eosinophils – a type of white blood cell – are a biomarker for asthma: too many of them in the lungs can cause inflammation. Photograph: Getty Images/Science Photo Library RF

Almost everyone reading this will know someone with asthma. Worldwide, there are about 334 million people with asthma, and 5.4 million people in the UK are being treated for it. But people with asthma vary greatly in their experiences of it.

Lehanne Sergison, 46, was diagnosed with asthma as a young child. In her case, the condition got progressively worse, and is now classified as severe. She had to give up work as a chartered surveyor six years ago.

"A lot of people think a blue inhaler is all it takes to manage asthma. They don't understand the complexities or the burden," she says. "I use home nebulisers [mouthpieces enabling medicine to be taken in as a mist] six times a day. I have physiotherapy to keep my chest clear – exercises to avoid muscle weakness and stay as flexible as possible. It is my responsibility to take all my medicines too, so I can stay as well as possible."



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"I was in the intensive care unit three times last winter," she says. "It's so difficult to plan your life. To try to avoid the trauma of emergency admissions, I have elective hospital admissions every six weeks for 10 days of intravenous treatment."

It is now increasingly recognised that asthma is a complex disease with a number of underlying causes. Steve Yancey, medicines development lead, GSK, explains why: "I go back to the biology. Each of us is different. Typically, our differences don't have an impact on our health status, but once in a while they do."

Asthma is what is known as a "gene-environment interaction" condition, where two different genotypes may respond to an environment in different

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ways. Some people may be at risk of developing asthma when they are exposed to particular allergens that would not lead to asthma in someone else.

"One person may be fine with tree pollen, with dust mites, with mould, and not go on to develop asthma. But those who have genetic predisposition, they will go on to develop it," Yancey says. So a person with no genetic predisposition to develop asthma will most likely not get it, whether or not they are exposed to an allergen – both allergen and predisposition have to be present.

Asthma can also present itself in different ways. Some patients cough, others are short of breath, some wheeze when exercising. Understanding the way that biology works differently in different people is at the heart of personalised treatment: it means that treatment can be targeted very specifically.



▲ GSK scientists David Gordon, Steve Yancey, Narendra Bam and Tim Hart. Photograph: Alan Brian Nilssen



This is done using biomarkers. A biomarker is a molecule, gene or characteristic by which a particular disease can be recognised. By understanding more about the role of biomarkers, scientists can identify ways of developing personalised treatment for respiratory conditions.

One example of a biomarker is eosinophil. Eosinophils are a type of white blood cell that everyone has in their body; these cells are thought to play a role in protecting the body, by increasing in number to defend the body against parasites or other infections.

But, in some people, eosinophils can contribute to respiratory diseases, such as asthma. If there are too many eosinophils and they are very active, they may travel to the lungs where they can cause inflammation. That is what may lead someone to have severe asthma.

"By knowing more about the underlying causes of asthma, you are able to target treatment more effectively, and provide the right medicine to the right patient," says Yancey.

However, while eosinophil-related asthma accounts for many of the people with severe asthma, there is still a large group of people with asthma who need to be understood better.

"There is still more that needs to be understood based on their biology, so they can have targeted treatment developed just for them," says Yancey.

At present, much of this personalised research and treatment is being done with those patients with the most severe forms of asthma, who are on the highest doses of their current medicine.

"Things are changing in asthma treatment, but there is still a massive need for research," says Sergison, who wants to raise awareness about severe asthma and gives a patient's perspective to researchers and charities working on asthma.

"We hope that one day we will be able to look ahead and treat patients earlier in the disease state," says Yancey. "It will be more preventative, not just treating the person in need. This is the cutting edge of science."

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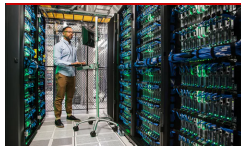
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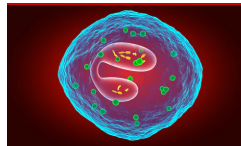
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Artificial intelligence: the key to developing effective drugs quickly

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By recognising patterns in large amounts of data, AI has made huge advances in industries as varied as finance, aviation and customer service. Now GSK are seeing the possibilities of using it to speed up drug discovery and combat intractable diseases



For many people, artificial intelligence, or AI, means intelligent robots taking over the world; Facebook or Google algorithms predicting our wishes before we know them ourselves; or, at the very least, automation replacing all of our jobs.

But behind the doomsday scenarios, AI is increasingly being used to ensure that drugs are being brought to patients more quickly, and more effectively.

GSK, like other pharmaceuticals companies, generates far more data than it can use – but this is now changing. “Data for a pharmaceuticals company today is its currency – it’s like pounds in the bank,” says John Baldoni, who leads GSK’s drugs discovery unit, Medicines Discovered Using Artificial Intelligence (MEDDAI).

“The drug development process is very complex and not always successful. What success the industry has is predicated by a lot of failure,” he continues. But as companies develop drugs, they create huge amounts of data, much of which has never