Getting plants to hurry

Tweaking a core process that plants use during photosynthesis can raise agricultural output

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Crop yields have risen dramatically over the last few decades. Over the last century, thanks to expanding crop land and fertilisers, increased agricultural output belied Malthus' forecast of shortage and disaster. And then, with developments in the use of hybrids, pesticides and farming techniques, food has stayed on the world's table despite a huge rise in population and per capita consumption.

Nevertheless, it is uncertain whether the world's resources would measure up to food demands in the coming decades. There are limits to the land that is available and we need to curtail the pollution caused by the production and use of fertiliser and pesticides. In this context, a group of researchers in the journal, *Communication Biology*, says that accelerating the process of photosynthesis, through which plants convert the sun's energy into food, could be a promising avenue for a new phase in increase of agricultural output.

Maria Ermakova, Patricia E Lopez-Calcagno, Christine A Raines, Robert T Furbank and Susanne von Caemmerer, from the Australian National University, at Acton, Canberra, and the University of Essex, UK, write that their work on photosynthesis has led them to a way of increasing the output of the process in an important category of plants. The mechanism by which the energy of sunlight, which breaks down the water molecule, is made use of, they write, depends on and is limited by a specific protein. Stepping up the production of this protein would hence lead to faster photosynthesis, even while keeping all other functions of the plant unchanged.

They write that a way to increase the pace of photosynthesis in a larger class of plants, like rice, wheat, barley, even lawn grass, which follow a certain path of photosynthesis, has been known for some time. Doing the same with another class of plants, which include important food crops like maize, sorghum, sugarcane and millets, and sources of bio-energy, would hence be of economic value, the paper says.

Photosynthesis takes place in plants by the action of certain proteins, which are most abundant in the leaves, and the pigment, chlorophyll. The proteins, with the help of the pigment, absorb the energy in sunlight and use it to separate hydrogen and oxygen from water.

While the oxygen is released, which makes photosynthesis of great value to life itself, the energy in the system gets hydrogen to combine with carbon, usually in carbon dioxide, to form carbohydrates, the food material.

The light-absorbing proteins in plants are found in the cell membrane, or in folded structures, which have a high surface area, called thylakoids, and these are collected in organs called chloroplasts. The net result of light striking gathering centres is that water, or H_2O, is split into $2H^+$ and O^-, components that retain the charges that keep the water molecule together. Pairs of O^- get together as O_2, or molecular oxygen, but two negative charges move freely, with the energy that keeps them away from the $2H^+$.

Over a series of "passing the parcel" exchanges, the energetic negative charge and the $2H^+$ are transported to where CO_2 is available, and the combination of CO_2 with $2H^+$, to form carbohydrates, becomes possible.

We can see that in the normal course, the negative charge could just have neutralised $2H^+$ to H_2 and the only effect would be splitting water into its components and their recombination as water, with some dissipation of heat. It is the sequence followed, of transporting the negative charges and the reduction of CO_2, which allows the sun's energy to get stored in carbohydrates.

Rate-limiting step

The *Communications Biology* paper delves into the process of transporting negative charge and identifies four main protein complexes that mediate the movement of charge, called electron transfer, in the rice, wheat, et al. And the second of these four complexes, Cytochrome b_6f, the paper says, is a "rate-limiting" step in the chain.

The paper then looks into the structure of Cytochrome b_6f and says it consists of two forms of Cytochrome, a protein called Rieske FeS, which contains iron and sulphur, and some others. And, "there is an increasing amount of evidence that the amount of Rieske FeS protein.....regulates the abundance of Cytochrome b_6f ", the paper says. Model plants of this group, which had been genetically primed to produce more Rieske FeS, have shown an increase in Cytochrome b_6f as well CO_2 assimilation, the paper says.

This group of plants with smaller grain, like rice or wheat, has one way of turning CO_2 to carbohydrates, while plants like maize, sorghum, sugarcane have another. The first group uses a molecule with three carbon atoms, on the way to forming carbohydrates, and is referred to as the C3 group. The second group makes use of a four-carbon atom intermediary and the group is known as the C4 group. C4 plants produce more sugars than C3 plants in bright and warm conditions and may have evolved to tolerate conditions of low CO_2.

Although there are differences in the two routes followed, the paper notes, both pathways include the Cytochrome b_6f component. The authors of the paper hence took up a typical C4 plant, a common species of grass, to see if increasing the level of Rieske FeS led to higher content of Cytochrome b_6f and faster photosynthesis. What they find, the paper says, is just that — first, the speed of electron transport is what limits the assimilation of CO_2 in C4 plants, especially when there is plenty of light and ample CO_2, and second, the speed of transport depends on the level of Cytochrome b_6f.

The conclusion, of introducing GM varieties of C4 plants with higher Cytochrome b_6f abundance would then lead to higher CO_2 assimilation and greater yield, is then presented as a possible answer to a future crisis of food insufficiency.

Will our food problems end?

That plants have a built-in limit to CO_2 assimilation has, most likely, got an evolutionary justification. Unbridled carbon fixation may have immediate benefits for the plant, but this would bleed the soil of nutrients, and may upset the balance of consumption and replenishment. There would hence be negative selection of the trait of ample Cytochrome b_6f, and the varieties that limit this protein would dominate.

To reverse the course of evolution, by genetically engineering varieties for higher food production, addresses a current problem, no doubt. But the plants would draw more nutrients, and this would call for greater use of fertilisers, which would lead to land degradation and pollution.

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Exploring newer approaches

Asthma marks the continuing battle of our immune system with the environment

LISA PARKER

India has a disproportionately high burden of chronic respiratory diseases, and research suggests that this is steadily increasing. These diseases affect the airways and other structures of the lungs, and one of the most common is asthma. A recent publication in *Lancet Global Health* by the India State-Level Disease Burden Initiative CRD Collaborators, reported that in 2016 there were 37.9 million cases of asthma in the country, which accounts for just over 10 per cent of the global estimate of 334 million people living with asthma, and chronic respiratory diseases were the second leading cause of disease burden in India that year.

Asthma is a chronic inflammatory disease that is characterised by recurrent episodes of wheezing and breathlessness, often known as acute attacks or exacerbations, which without warning make breathing extremely difficult. For many people living with chronic respiratory diseases, these exacerbations are the worst and most frightening aspect of their disease, and often require emergency treatment and hospitalisation.

During an exacerbation, the bronchial tubes lining the airways become inflamed and swell as immune cells enter the airways, excessive mucus is produced, and the muscles surrounding the bronchial tubes tighten. These responses cause airway narrowing and obstruction and reduce the flow of air into and out of the lungs.

Our lungs are different to most other organs because they are continuously exposed to the environment, with approximately 11,000 litres of air moving through our respiratory system daily. The air we breathe contains pollutants from traffic and industrial sources, indoor air pollution from the smoke of burning fuels, occupational chemicals and dusts, aerosolised toxins (like tobacco smoke) and microbes or allergens.

Breathing unhealthy air is a cause or contributor to most respiratory conditions, and the potential for environmental injury to the lung is common throughout a person's life. These micro-injuries lead to the recruitment of immune cells and development of inflammation within the lungs. Furthermore, these environmental triggers are all known risk factors for both the development of chronic respiratory diseases and the exacerbations that frequently occur.

The development of asthma in humans is complex, and relies upon a combination of genetic, social and environmental factors. Some of the factors that influence the development of asthma are reasonably well understood. For example, improving air quality is an important step in promoting respiratory health, but this is clearly an enormous task in the face of continuing industrialisation. Indeed, ambient air pollution exposure has increased substantially in most parts of India over the last few decades. In 2016, a new World Health Organization air quality model reported that 13 of the world's 20 most polluted cities are in India, with ambient air quality limits for particulate matter with a diameter of less than 2.5 micrometres ($PM_{2.5}$), which can penetrate deep into the airways, reported to have exceeded the recommended WHO levels.

As researchers, we are exploring the changes that occur in the airways in response to inhaled components — including viral and bacterial microbes, and diesel exhaust particles. We have focused particularly on the inflammation that is triggered by these components. Our laboratory research has shown that the cells lining the airways (epithelial cells) are very effective at communicating with the immune cells originating from the blood (white blood cells), and this communication allows the cells to work together to create a much larger inflammatory response to a microbe than it is possible for either cell to produce on its own. Diesel exhaust particles amplify the inflammation even further.

We discovered that a protein called interleukin-1beta (IL-1beta) is central in the communication between epithelial cells and white blood cells during the initiation of airway inflammation. IL-1beta is produced by epithelial cells in response to both bacterial and viral microbes, it can signal to white blood cells telling them to make more inflammatory proteins, which then act back on the epithelial cells, creating a feedback loop and causing even more inflammation.

Other factors involved in the development of asthma are less clear, but are also likely to have a strong immune component. For example, infections of the lower airways (caused by viral or bacterial microbes) have been among the top three causes of death and disability among both children and adults for decades and there is now a growing appreciation that these types of infections in children can also make them more susceptible to developing chronic respiratory diseases, like asthma, later in life.

Viral respiratory tract infections are one of the most common illnesses in humans. According to estimates, adults succumb to one or two colds annually whilst children can contract as many as eight. It is therefore crucial that our airways can detect and respond to microbes, allowing us to eliminate them, and in most cases this is done rapidly and with minimal damage to the airway.

However, if the "normal" response to a virus is enhanced, for example due to ongoing inflammation (and therefore the presence of white blood cells) within the airways of people with asthma, or by pollutants in the environment, then an excessive inflammatory response will occur and potentially trigger an acute exacerbation, which in some cases can be severe enough to require hospitalisation.

Our research in the University of Sheffield has established that one of the most frequent causes of the common cold, rhinovirus, also triggers the IL-1beta feedback loop and subsequent inflammation. We are therefore taking a multi-faceted approach to identify potential therapeutic targets to reduce the ability of rhinovirus infections to cause detrimental inflammation within the airways. We have identified several proteins within the epithelial cell that can regulate the inflammatory pathways that are triggered by rhinovirus infection.

We found that one protein, called Dusp10, is present within airway epithelial cells and can turn off the signalling pathways activated by IL-1beta and rhinovirus, reducing the amount of inflammation produced. A second protein we are studying is tenascin-C, this is located in the extracellular matrix (which surrounds the cells and helps maintain their environment) but is present in only small quantities in the normal airway due to its inflammatory properties.

We have recently confirmed that tenascin-C levels are higher in the airways of people with asthma, and shown for the first time that rhinovirus is capable of increasing tenascin-C levels airway epithelial cells and surrounding environment. Our work has therefore identified DUSP10 and tenascin-C as important regulators of airway inflammation in response to viral infection, suggesting that they may be exploitable as drug targets in the future.

Finally, our work has also utilised liposomes, microscopic spherical vesicles consisting of one or more phospholipid bilayers. We found that one liposome, Saps, markedly reduced inflammatory responses to rhinovirus without impairing the antiviral cellular responses (those aimed at controlling and removing the virus). This is particularly exciting as Saps liposomes are small enough to be administered directly to the lungs using an inhaler, and biologically similar liposomes are currently being used to deliver drugs to treat bacterial infections in cystic fibrosis patients.

Although in the early stages, these approaches offer exciting insights into exactly how rhinovirus is causing airway inflammation, which can be so detrimental in people with asthma, and potential new approaches for reducing this inflammation therapeutically in the future.

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PLUS POINTS

Much like home

Scientists have worked out just how many planets like our own might be waiting out in the universe. The new study gives the best estimate yet of how many Earth-like planets are orbiting around Sun-like stars. The discovery will help guide astronomers as they search those planets for signs of alien life, by trying to understand more about the planets.

Nasa's Kepler Space Telescope helped find that there are thousands of planets waiting outside of our solar system, orbiting around their own suns. Over the nearly 10 years it scanner the sky, it was watching out for transit events — the slight dips in light that happen when a planet moves in front of a star, which can be used to understand those planets' size and characteristics. But scientists want to know how many of those alien worlds are like ours, sitting close enough to their star that they get enough light to provide energy for life.

"Kepler discovered planets with a wide variety of sizes, compositions and orbits," said Eric B Ford, professor of astronomy and astrophysics at Penn State and one of the leaders of the research team. "We want to use those discoveries to improve our understanding of planet formation and to plan future missions to search for planets that might be habitable. However, simply counting exoplanets of a given size or orbital distance is misleading, since it's much harder to find small planets far from their star than to find large planets close to their star."

To try and understand how many of those planets might be out there waiting, scientists came up with a new method to help estimate how many planets might have formed. They developed a model that allowed them to create new, imaginary universes of stars and planets — and then watch to see how many of those would have been picked up by the Kepler Space Telescope, and how many would have been missed.

"We used the final catalogue of planets identified by Kepler and improved star properties from the European Space Agency's Gaia spacecraft to build our simulations," said Danley Hsu, a graduate student at Penn State and the first author of the paper.

"By comparing the results to the planets catalogued by Kepler, we characterised the rate of planets per star and how that depends on planet size and orbital distance. Our novel approach allowed the team to account for several effects that have not been included in previous studies."

Scientists can now use the findings to comb through the rest of the universe looking for planets that look like our own, with the hope of launching a major mission to study them. How ambitious that mission needs to be will depend partly on just how many planets there are waiting to be found, and so the discovery will help decide the scale of that search.

The researchers found that there are probably planets like our own — between three-quarters and one-and-a-half times as big as our planet, and similar length years — waiting to be found around roughly one in four stars.

But they also worked out how accurate that estimate could be. The potential uncertainty means that missions should plan to find such worlds as often as every two stars, they suggest.

The independent

Robotic tail

Millions of years after the ancestors of humans evolved to lose their tails, a research team at Japan's Keio University have built a robotic one they say could help unsteady elderly people keep their balance. Dubbed Arque, the grey one m-long device mimics tails such as those cheetahs and other animals use to keep their balance while running and climbing, according to the Keio team.

"The tail keeps balance like a pendulum," said Junichi Nabeshima, a graduate student and researcher at the university's Embodied Media Project, displaying the robotic tail attached to his waist with a harness.

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