fruits — also contribute greatly to global VOC emissions, and form SOAs much more readily than isoprene[1,3].

To represent SOA formation in models of air quality or climate, scientists generally focus on a few key VOCs, using the concept of SOA yield — which is normally defined as the particulate mass produced from the oxidation of a given mass of a gaseous parent VOC[4]. McFiggans *et al.* demonstrate that simply adding together the SOA masses generated from the individual components of a VOC mixture will probably substantially overestimate total SOA production. More specifically, the researchers carried out laboratory experiments that showed that the amount of SOAs formed from the oxidation of mixtures of monoterpenes and isoprene is much smaller than the sum of SOAs produced when the different VOCs are oxidized separately. They observed similar patterns when isoprene in the mixtures was replaced with other atmospheric gases (methane or carbon monoxide), showing that the amount of SOA formed from mixtures is, in general, not directly proportional to the amounts of the individual components.

Previous studies[5,6] have shown that SOA-particle formation is suppressed by isoprene, which scavenges oxidants from the atmosphere, and that highly oxygenated organic molecules (HOMs) produced from monoterpenes might have a role in forming new SOA particles[7]. McFiggans and colleagues have delved much more deeply into these issues by searching for the associated mechanisms and by quantifying the effects of isoprene, carbon monoxide and methane on SOA yield. The authors confirmed that isoprene scavenges oxidants from the atmosphere, but also found that the reactive compounds formed from this process can, in turn, scavenge monoterpene-derived HOMs that have high potential for forming SOAs (Fig. 1).

The authors went on to carry out computational simulations, which showed that the proposed scavenging mechanisms can operate effectively in the atmosphere and reduce the global mass concentration of SOAs. Taken together, the new findings suggest that laboratory studies of SOA yields must be conducted using realistic mixtures of atmospheric vapours, rather than just using single compounds, as is widely done. Moreover, model simulations need to consider the effects of mixtures on SOA formation.

McFiggans and colleagues' measurements were carried out in a chamber, in which oxidation times were less than one hour. In the atmosphere, however, oxidation takes much longer (up to a couple of days), and such longer times and multistep oxidation processes are important for SOA formation[8]. The authors also used an approximately tenfold higher concentration of oxidants (mainly the hydroxyl radical, ȮH) than is found in the atmosphere, because this allowed measurements to be made more accurately than would be possible using atmospheric concentrations. The concentrations of carbon monoxide or methane used in the chamber were 10–100-fold higher than atmospheric levels, for the same reason. The effects of VOC mixtures on SOA formation in the real atmosphere, where concentrations of oxidants and relevant chemical species are lower but reaction times are longer, should now be investigated.

The present study shows that HOMs produced from the oxidation of monoterpenes by ȮH can be scavenged effectively when a mixture of isoprene, methane and carbon monoxide is added to the reaction system. However, more than 50% of atmospheric monoterpenes seem to be oxidized by ozone, so it remains to be seen how effective this mixture is at scavenging HOMs formed by ozone. Furthermore, monoterpenes in the real atmosphere are mixed not only with isoprene, methane and carbon monoxide, but also with many other compounds at a wide range of concentrations. This will produce a wide range of oxidation products that might interact in complex ways to influence scavenging and SOA formation. It is therefore crucial to identify the main factors that control the suppression of SOA formation in realistic atmospheric conditions. McFiggans and co-workers' study is the first step in this direction.

The effect of aerosols on climate depends on the fraction of tiny particles that can seed cloud formation. In this regard, both the number of particles per unit volume and the size of particles are crucial[9]. Low-volatility HOMs produced from monoterpene oxidation have a key role in growing aerosol particles of approximately 1–2 nanometres in diameter to sizes large enough to seed clouds (60–100 nm). These HOMs might also be directly involved in the initial steps of forming nanometre-sized particles in the atmosphere[10]. It will be necessary to include the HOM scavenging observed by McFiggans *et al.* in global models that explicitly consider particle formation and growth, to understand the climatic implications.

Finally, the suppression of SOA formation by the mixtures of compounds studied will depend on the relative concentrations of those compounds and, for isoprene, on the acidity of pre-existing particles, both of which are changing in the atmosphere as a result of emissions associated with human activities. Further research is needed to understand the potentially large effects of such emissions on the magnitude of SOA suppression, and therefore on climate change. ■

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HOST–MICROBE INTERACTIONS

Plants fight fungi using kiwellin proteins

Fungal infection can affect crop yield. A plant protein found to counter fungal-induced interference with host metabolism illuminates antifungal defences and mechanisms that inhibit metabolic enzymes. SEE LETTER P.650

MARY C. WILDERMUTH

Organisms, such as fungi, that cause disease in plants often secrete proteins that aid growth and reproduction in the host. These are termed effector proteins, and some are deregulated metabolic enzymes that manipulate key metabolic pathways in plants. Han *et al.*[1] reveal on page 650 that a protein in maize (corn) blocks the enzymatic activity of a fungal effector enzyme, thereby thwarting the effector's ability to influence maize metabolism in a way that limits the plant's defence response.

The authors studied infection of maize by the fungus *Ustilago maydis*, which can cause corn smut disease and results in substantial crop loss worldwide. The enzyme chorismate mutase (Cmu1), which catalyses the molecular conversion of chorismate to prephenate, is a known effector protein of this fungus[2]. Han and colleagues engineered a tagged version of Cmu1 and used a technique called co-immunoprecipitation to try to identify

whether any plant proteins interact with Cmu1 in maize leaves infected with *U. maydis*. This revealed a maize protein, called *Zm*KWL1 by the authors, that binds to Cmu1.

Han *et al.* determined that *Zm*KWL1 is a member of a family of proteins called kiwellins. Of the 20 kiwellin proteins found in maize, only *Zm*KWL1 is highly expressed in response to *U. maydis* infection[3]. The authors found that only *Zm*KWL1, of the four maize kiwellins they tested, interacts with Cmu1 *in vitro*. Furthermore, *Zm*KWL1 exclusively bound and inhibited purified *U. maydis* Cmu1 *in vitro*, whereas maize versions of chorismate mutase were not affected by *Zm*KWL1. The specificity of this interaction is remarkable, given the structural similarity between the fungal and maize enzymes.

Little was known previously about how kiwellins function. They are highly expressed in kiwi fruit and can trigger allergic responses in humans[4,5]. Han *et al.* found that kiwellin-encoding sequences are present both in non-seed plants, such as mosses, and in seed-producing plants, such as conifers and flowering plants. However, kiwellins are not present universally, and were not identified in the Brassicaceae family of plants, which includes the model plant *Arabidopsis thaliana*. Although the genomes of many species, including mosses, encode only one kiwellin, gene analysis by Han *et al.* suggests that some lineage-specific increases in kiwellins occurred as plants evolved, probably through gene duplication.

*Zm*KWL1 is part of small subgroup of nearly identical kiwellins found only in cereal plants. It is tempting, therefore, to speculate that this subgroup of kiwellins binds to the same target. Anecdotal evidence that requires additional experimental verification supports this hypothesis. For example, *Zm*KWL1 and its most closely related kiwellin protein Sb01g018600, from the cereal sorghum, have amino-acid sequences that are 87% identical. *U. maydis* does not infect sorghum, so *U. maydis* Cmu1 is not the target of sorghum kiwellin. However, another smut-causing fungus, *Sporisorium reilianum*, infects both sorghum and maize. This fungus encodes a secreted version of chorismate mutase (called sr16064)[6] that might be the target of Sb01g018600.

Han and colleagues also provide evidence suggesting that other subgroups of kiwellins have evolved to recognize distinct effector proteins. Apart from *Zm*KWL1, maize kiwellins did not bind to Cmu1 *in vitro*, raising the possibility that some of these other kiwellins recognize other fungal proteins. The authors also noted previous studies of potatoes[7,8] and husk tomato plants[9] in which some kiwellins were highly expressed in response to organisms that cause disease in these plants.

It is intriguing that kiwellin-encoding genes are also found in genomes of some fungi that infect plants, including *U. maydis*. Han and colleagues do not speculate about the possible origins or functions of these genes. Perhaps fungal kiwellins were acquired by gene transfer from a cereal host plant, given that they are most similar to a large group of kiwellins in cereals. Could these fungal kiwellins counter the inhibition of their effectors by the plant kiwellins? Future experiments should investigate this possibility.

To determine what features of kiwellins might allow them to form strong and specific interactions with effector proteins, Han and colleagues used X-ray crystallography to generate structural models of *Zm*KWL1. This revealed that, like kiwellins in kiwi plants[4,5], *Zm*KWL1 has a central 'β-barrel' domain that is stabilized by numerous connections called disulfide bridges. This type of arrangement is evolutionarily conserved in plant-secreted defence proteins commonly referred to as pathogenesis-related 4 family proteins[10,11], and also found in fungal secreted proteins called cerato-platanins[12] that modulate fungal interactions with hosts such as plants. However, plant kiwellins also contain a structure called an anti-parallel β-sheet, comprised of two β-strands and multiple surface-exposed loops. Han and colleagues' structural studies of *U. maydis* Cmu1 in complex with *Zm*KWL1 shows extensive interaction between the proteins. The interactions form mainly between *Zm*KWL1 amino-acid residues in the anti-parallel β-sheet and Cmu1 residues in an extensive loop region unique to the fungal enzyme.

Han *et al.* report that *Zm*KWL1 inhibits Cmu1 by affecting the enzyme's catalysis and not by competing for binding of its substrate molecule, chorismate. When the authors prevented the expression of *Zm*KWL1 in maize, plant infections with *U. maydis* were more severe than in maize that expressed *Zm*KWL1. Presumably, this is because the absence of *Zm*KWL1 enabled *U. maydis* Cmu1 to convert chorismate to prephenate, thereby limiting the availability of chorismate for synthesis of the plant defence hormone salicylic acid (Fig. 1). Previous work[2] showed that deletion of the gene encoding Cmu1 in *U. maydis* caused an increase in salicylic acid production and associated plant-defence responses and a reduction in the success of fungal infection, compared with infection by strains that had Cmu1.

Chorismate is a key metabolite molecule in plants, and enzymes that use it compete to co-opt the molecule into their respective biosynthetic pathways, which generate molecules that include amino acids, hormones, vitamins and plant cell-wall components[13]. Enzymes that use chorismate have similar structures and reaction mechanisms[14,15]. The ability of *Zm*KWL1 to inhibit Cmu1 with such specificity raises the question of whether other kiwellins in plants have evolved to specifically inhibit related enzymes, including isochorismatases. Plant-infecting fungi can secrete isochorismatases to limit the availability of the molecule isochorismate[16]. This molecule can be converted to salicylic acid (Fig. 1). *U. maydis* encodes an isochorismatase that is predicted to be secreted. The use of multiple effectors to target the pathway that generates salicylic acid would have a major effect on the ability of plants to mount a defence response. If distinct kiwellins evolved to inhibit such fungal effectors, it might enable plants to generate sufficient salicylic acid to induce the robust defences needed to limit fungal infection.

Han and colleagues have set the stage for the identification of other kiwellin effector targets. Detailed future analyses of kiwellin evolution and function might help to reveal the full range of roles of these proteins. Perhaps naturally occurring or engineered kiwellins

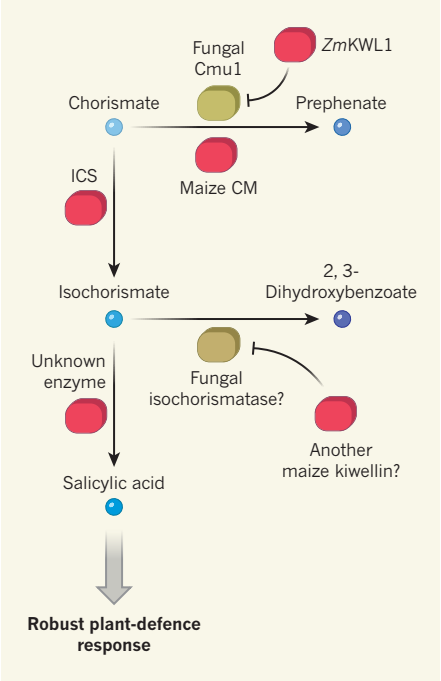

Figure 1 | A battle between plant and fungal enzymes for control of plant metabolism. Han *et al.*[1] studied the infection of maize (corn) by the disease-causing fungus *Ustilago maydis*. They report that a plant protein that they term *Zm*KWL1 can block the action of a fungal enzyme called Cmu1 in maize cells. Plant enzymes are shown in red, and fungal enzymes in yellow. Cmu1 belongs to a family of chorismate mutase (CM) enzymes that is also found in maize, and catalyses the conversion of chorismate to prephenate as part of a pathway of amino-acid synthesis. This reaction prevents chorismate from contributing to a pathway that generates the plant defence molecule salicylic acid through the action of an isochorismate synthase (ICS) enzyme[18] and another unknown plant enzyme. *Zm*KWL1, a member of a family of proteins called kiwellins, prevents Cmu1 from subverting plant metabolism to limit the production of salicylic acid. *Ustilago maydis* also encodes an isochorismatase enzyme, which is predicted to be secreted. This is a type of enzyme that can convert isochorismate to 2,3-dihydroxybenzoate, although whether the fungal enzyme functions in this way in plants is unknown. If this enzyme affects the availability of molecules in the salicylic-acid-generating pathway, it might be targeted by another kiwellin protein.

that specifically inhibit a range of enzymes that use chorismate or isochorismate could be developed to enhance agricultural productivity. Furthermore, kiwellins could be used to manipulate chorismate metabolism to enhance the production of a variety of commercial chorismate-derived products[17].

Kiwellins might also have the potential to be developed as antimicrobial agents for the treatment of human disease. Certain bacteria, including the bacterium that causes tuberculosis, use chorismate to make molecules that they require for infection[14]. The human genome encodes neither chorismate-using enzymes nor kiwellins; therefore, kiwellin-based inhibition of these microbial targets should be investigated. The range of metabolic proteins bound by kiwellins probably extends beyond enzymes that use chorismate, and it will be exciting to uncover the full versatility of those proteins. ■

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PLASMA PHYSICS

Statistical models boost fusion performance

Research into a technique called inertial confinement fusion aims to enhance nuclear–fusion performance in laboratory experiments. Improvements in the technique have been made using a clever statistical approach. SEE ARTICLE P.581

MARK C. HERRMANN

The pursuit of thermonuclear fusion, the power source of stars, in the laboratory is an ambitious endeavour. For a useful number of fusions to occur, fusion fuel must be heated to tens of millions of degrees so that it produces an ionized gas called a plasma. If such a plasma could be confined for long enough, the energy released by fusions, known as the yield, would greatly exceed the energy invested in the plasma — a long-elusive goal of fusion researchers. In inertial confinement fusion (ICF) experiments, the fusion plasma is generated when high-power drivers, such as lasers, are used to implode fusion fuel. On page 581, Gopalaswamy *et al.*[1] report the use of experimentally trained statistical models to triple the fusion yield and substantially improve the plasma confinement in ICF experiments.

Gopalaswamy and colleagues studied ICF implosions at the OMEGA Laser Facility at the University of Rochester in New York[2]. In these experiments, 60 high-power laser beams are directed onto a millimetre-sized capsule that contains fusion fuel (Fig. 1). The intense light produces large pressures, imploding the fuel at high velocities. When the imploding fuel stagnates, kinetic energy is rapidly converted to temperature and pressure, generating a fusion plasma that is confined by its own inertia.

The number of design choices for optimizing this fusion plasma is enormous, because all aspects of the capsule's dimensions and structure, as well as the details of the laser and the time dependence of the laser's power, can be varied. Implosion performance can also be considerably affected by 'hydrodynamic' instabilities that are seeded by inevitable imperfections in the manufactured capsule and imbalances or instabilities in the applied laser light. Unsurprisingly, the complexity of this implosion system leads to fusion performance that is extremely sensitive to design details and instabilities.

With so many design choices, and with limited experimental data, the standard approach to optimizing fusion performance has been to use theoretical insights along with sophisticated radiation–hydrodynamic simulations that follow, as well as we know how, the physics of the implosions and their degradations. This technique produced the previous record fusion yield at OMEGA[3]. However, for these implosions, the simulations significantly overestimate the experimentally observed fusion performance. The causes of this discrepancy are not fully understood. A lack of accurate physics models in the simulations or

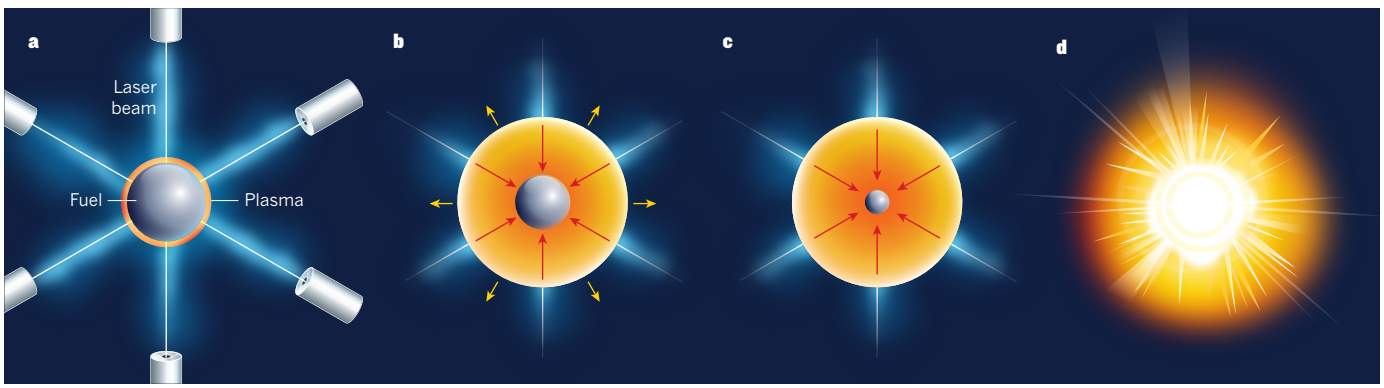

Figure 1 | Inertial confinement fusion. Gopalaswamy *et al.*[1] present a method for increasing the energy output from experiments at the OMEGA Laser Facility[2] that involve a process called inertial confinement fusion. **a**, In these experiments, laser beams rapidly heat the surface of a millimetre-sized fuel capsule, producing an envelope of highly ionized gas known as a plasma. **b**, The plasma blasts outwards (yellow arrows), generating large forces (red arrows) that compress the fuel. **c**, The compression continues until the fuel reaches extreme pressures. **d**, Finally, the fuel heats up and undergoes nuclear fusion.