Further work will be needed to determine how the presence of these gut bacteria can influence immune cells at distant sites.

Although faecal transplantation is effective as a treatment for a variety of human illnesses[17], for microbiota-based therapies to be more widely adopted in the clinic, the use of defined bacterial strains will probably be preferred. Generating therapeutics that contain defined strains might increase the robustness of responses and reduce the risks associated with the transplantation of faecal samples of unknown bacterial composition.

Previous studies aimed at evaluating the effects of perturbing the microbiota to augment checkpoint-blockade responses have mainly focused on trying to identify differences between the microbiota of responders and non-responders. By contrast, Tanoue and colleagues demonstrated a way to define a subset of bacterial strains that can specifically boost tumour-reactive $CD8^+$ T cells. These strains were not present in most healthy individuals whom the authors tested, and were of low abundance in the faecal sample in which they were identified. This potentially explains why previous studies have not identified these bacteria as having a role in boosting immune responses.

When checkpoint blockade is used to invigorate an immune response, it frequently causes an adverse state of inflammation and an autoimmune reaction, particularly in gut tissues[18,19]. The 11 bacterial strains had a minimal effect on reducing cancer growth in the absence of accompanying checkpoint-blockade treatment, and it remains to be determined whether the induction of $CD8^+$ IFN-γ^+ T cells might exacerbate such adverse immune reactions in people receiving checkpoint-blockade therapy. Furthermore, mouse recipients of the strains had to be pretreated with antibiotics before administration to enable the bacteria to colonize the host. This method might place individuals at risk of infection by disease-causing organisms such as *Clostridium difficile*, which typically thrive only in the absence of the normal gut bacteria.

Yet, despite this possible risk, there is reason to be cautiously hopeful. The authors found little or no evidence of colonic inflammation in mice or monkeys treated with the 11 bacterial strains. Perhaps this defined set of normal bacterial residents specifically activates only infection- and tumour-reactive T cells without triggering self-reactivity. More studies will be needed to evaluate the effects of these bacteria on inflammation and autoimmune reactions, but these promising data suggest that we are making progress in efforts to harness the microbiota to fight infection and cancer. ■

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ATMOSPHERIC CHEMISTRY

Aerosol formation assumptions reassessed

Experiments show that the amount of atmospheric particles produced from plant emissions could be lower than was thought — challenging our understanding of the processes that affect air quality and climate. SEE ARTICLE P.587

FANGQUN YU

Plants take up carbon dioxide and release volatile organic compounds (VOCs), in a similar way to how other organisms breathe in oxygen and exhale CO_2. These VOCs are oxidized in the atmosphere and then contribute substantially to the burden of tiny particles suspended in the air, which are known as aerosols. Aerosols produced from VOCs are known as secondary organic aerosols (SOAs), and affect both air quality and Earth's climate. The total rate of SOA production was thought to be the sum of the individual rates associated with the oxidation of each VOC. But on page 587, McFiggans *et al.*[1] show that a more accurate description is needed to improve the representation of SOAs in computational models of air quality and climate.

The atmosphere contains a complex mixture of VOCs, originating from biological sources and from human activities. Natural VOC emissions are globally dominated by the compound isoprene, produced by trees and shrubs[2]. However, the ability of isoprene to form SOAs is limited. Monoterpene compounds — the main components of the perfumes produced by flowers, leaves and

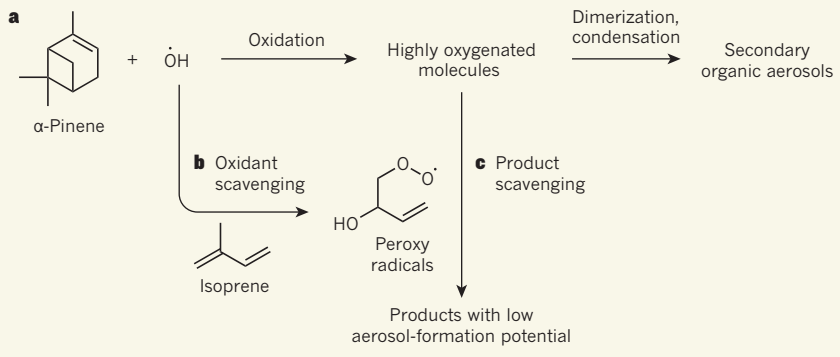

Figure 1 | Processes that affect aerosol formation from plant emissions. **a**, Many plants emit compounds known as monoterpenes (such as α-pinene) to the atmosphere, where they are oxidized by hydroxyl radicals (ȮH) to form highly oxygenated molecules (HOMs). These molecules can form dimers and contribute to the condensation of tiny particles known as secondary organic aerosols (SOAs), which affect cloud formation and air quality. **b**, Another plant emission, isoprene, can react with — and thereby scavenge — hydroxyl radicals to form peroxy radicals, suppressing SOA formation from monoterpenes[5,6]. **c**, McFiggans and colleagues' laboratory experiments[1] show that peroxy radicals formed from isoprene oxidation react with, and thus scavenge, oxidation products such as HOMs that have high SOA-formation potential, further lowering SOA production. They also find that methane and carbon monoxide suppress SOA formation through product scavenging (not shown).

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