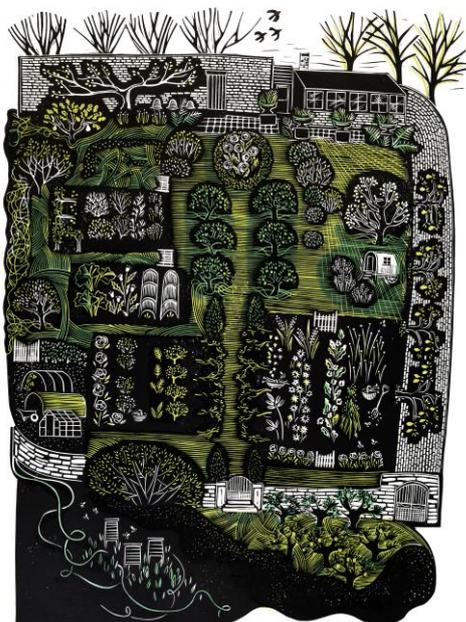


fierce

Vivienne Baillie Gerritsen

Scents communicate. Wrapped in silence perhaps, yet they have much to say. They may revolt us or delight us, go unnoticed or do nothing for us, but scents always have something to convey. So much so that many of us dab a little perfume on the nape of our neck or spray eau de toilette on our torso at the start of each day. We want to smell nice, or at the very least clean. We may even splash a little scent on before going out with an end to seduce. Much like flowers do. Who has not crossed a room or a lawn to inhale the wonderful perfume released by hyacinths, lilacs or a fragrant rose? Or leaned over to breathe in the delicate scent of jasmine wafting from a cup of tea? Long before humans, plants grasped the advantage of releasing pleasant volatile vibes to attract a pollinator or two. Some viruses have caught on with the practice too - and in a rather cunning way. The cucumber mosaic virus, for instance, is capable of infecting plants while also forcing them to become attractive to insects who will transfer the virus to other plants. Uncharacteristically, one sole protein in CMV can perform both actions. Its name is 2b, or CMV2b.



Pythouse Kitchen Garden, by Sarah Kirby

linocut, courtesy of the artist
instagram: @sarahkirbyprint

Cucumber mosaic virus has a capsid which is almost spherical in shape. It was characterised for the first time, in 1934, in cucumbers that presented mosaic patterns on their skin – hence the name. However,

besides cucumbers, CMV has since shown that it can infect an astounding variety of plants, around the globe. This is more than any other known plant virus can do, and it causes huge damage to crops of all sorts, and subsequent economic loss to farmers. One of the reasons is that the virus is particularly infectious, as it is transmitted via plant sap or simply by insects that happen to land on the plants – or are attracted to them – and unknowingly become vectors.

Over time, plants have developed different strategies to fight off viral infection. In the case of CMV, each viral capsid carries three strands of RNA which, upon infection, are released into the plant host. RNA entry triggers off the machinery which ultimately results in viral multiplication. Consequently, one of the host's choice strategies is to attack the viral RNA and degrade it – thus slowing down viral replication or, with any luck, putting an end to it. This strategy is known as post-translational gene silencing, or PTGS. In the model plant *Arabidopsis thaliana*, PTGS is triggered off by a lipid-based plant hormone known as jasmonate, or jasmonic acid. As its name infers, jasmonate was originally extracted from jasmine oil in the 1960s, itself derived from the jasmine flower which releases the sweet scent we know and that we have worn as perfume since its introduction to Europe in the 16th century.

Jasmonate is a plant hormone vital to an indispensable signalling cascade involved in key processes in plants: the jasmonate signalling pathway. This signalling pathway is governed by jasmonate-zim domain (JAZ) proteins, all of which are repressors. There are twelve known JAZ repressors (JAZ1-12) in *A.thaliana*, each independently active in processes as varied as plant growth, plant development, plant survival, plant regeneration and responses to various kinds of stress. It is this signalling pathway that CMV has – very astutely – chosen to dabble with by way of one of its proteins: cucumber virus protein 2b, or CMV2b.

CMV2b is a modest-sized protein which is thought to act as a homotetramer. When CMV infects a plant, the virus uses its host's machinery to synthesize all the proteins it needs to multiply. One of these proteins is CMV2b, which then adopts one of two roles: either it stops the plant's capacity to degrade viral RNA, or it gets the plant to send out odour signals that attract vectors, such as aphids, which will carry the virus further. How does CMV2b do this? Does it have two different functions? Or does the same action result in two different fates? The answer is: CMV2b performs both functions independently.

On the one hand, CMV2b can switch off a cell's ability to degrade viral RNA. Consequently, the cell's machinery continues to translate viral RNA which, unless checked, will give rise to progeny. It is not yet known how this occurs on the molecular level but there is reason to believe that CMV2b enters the cell nucleus to interfere directly with DNA methylation – a process involved in the

regulation of gene expression. On the other hand, CMV2b can hijack the plant's jasmonate signalling pathway and get it to attract aphids – a major insect pest of many kinds of vegetables, among which the cucumber of course. How does CMV2b manage this? By binding directly to JAZ1, one of the major repressor proteins in the jasmonate signalling pathway. What CMV2b does is take the place of a factor known as COI1 whose role is to degrade JAZ1 thus setting the signalling pathway off. However, when CMV2b takes the place of COI1, JAZ1 is not degraded and thus continues to repress the jasmonate signalling pathway – in other words, the plant's defence system. As a result, no volatiles are released to warn the surrounding plants of viral infection. Instead, aphids are literally able to sniff out the infected plants and are drawn to them. Via their stylet, they then suck the plant's sap, picking up the virus as they do so and transmitting it to any other plant they choose to feed on.

It may not seem the case but our vegetable gardens fight vicious battles. So does farmland across the world. CMV has been reported on every single continent, including Antarctica, which is hardly surprising given the range of its hosts: over 1200 plants, including crops and ornamental flowers. Moreover, besides vectors other than aphids, CMV is transmitted by over 80 different aphid species some of which are able to survive harsh winters, huddled around the roots of their hosts – it is enough to see your backyard greenfly in a different light. Certainly, CMV sounds invincible. A greater understanding of the jasmonate signalling pathway and in particular how CMV has found ways to manipulate it, could provide insight for future anti-pest strategies in agriculture.

Cross-references to UniProt

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Protein TIFY 10A/JAZ1, *Arabidopsis thaliana* (Mouse-ear cress): Q9LMA8

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