spectrum of properties is only ~0.1 Å.

There are many curious features of the charge-ordered manganate systems. Thus, charge-ordering stripes are found in $La_{1-x}Ca_xMnO_3$ with $x > 0.5$ (9). In $Nd_{0.5}Ca_{0.5}MnO_3$, with its small $\langle r_A \rangle$, there is negligible change in volume at T_{co}, and the volume decreases continuously down to low temperatures. Charge localization also occurs at low temperatures, as indicated by the Mn bond valence sums (*10*). Even in the insulating phases, there would be Mn^{3+}-O-Mn^{4+} clusters and thermally activated hopping-inducing ferromagnetic interactions. Indeed, one of the problems with the charge-ordered manganates is the apparent presence of inhomogeneities in the composition of the A-site cations. How one can avoid such inhomogeneities is a question that merits further attention. Another important problem that needs further research is the effect of electric fields and radiation on the charge-ordered states. Structural studies of charge ordering and orbital ordering in manganates and the effect of magnetic fields on the charge-ordered states would also be of value.

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PLANT BIOLOGY

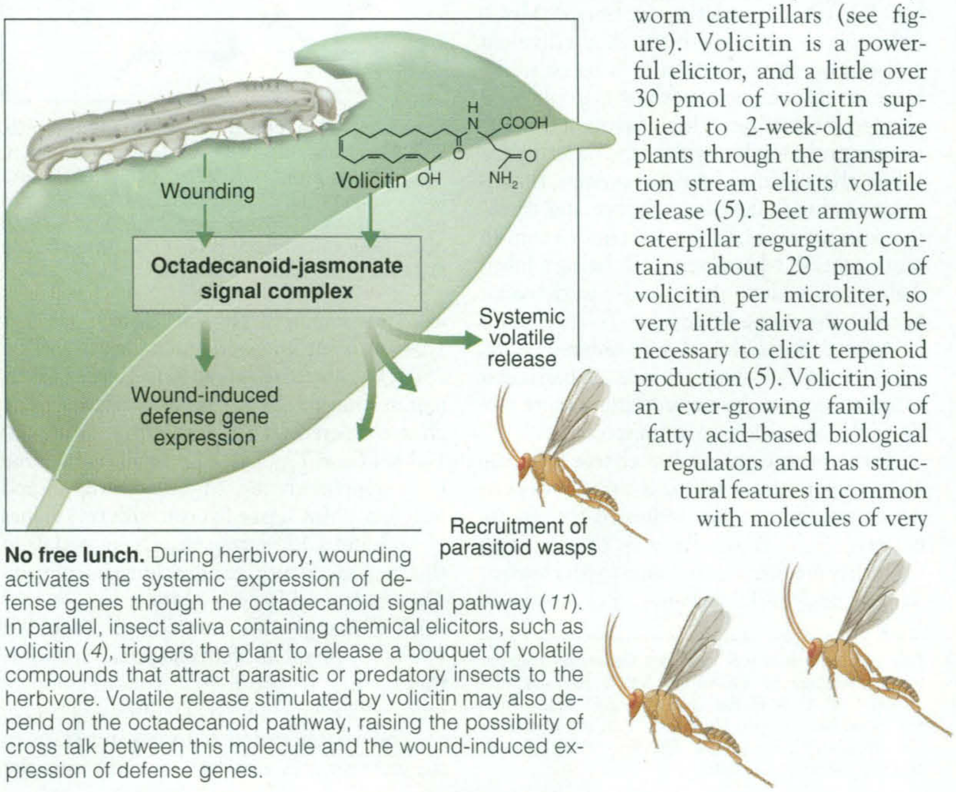
New Fatty Acid-Based Signals: A Lesson from the Plant World

Edward E. Farmer

Herbivorous insects and higher plants have been at war for well over 100 million years. During this time, the warring parties have evolved sophisticated strategies to eat or to avoid being eaten. Plants are equipped with an arsenal of constitutive and inducible defenses ranging from chemical poisons and feeding deterrents to proteins that can block the activity of the insects' digestive enzymes. Insects, on the other hand, can sometimes detoxify, sequester, or bypass these defenses. In the face of constant assault, how can plants augment their defenses? One answer is to recruit predators that attack the herbivores. During attack by insect herbivores, certain plants emit cocktails of volatile compounds that can attract other predatory or parasitic insects (*1–3*). In this issue on page 945, Alborn *et al.* (*4*) report an impressive advance in our knowledge of the trigger for the release of volatiles in herbivore-attacked plants that may have broader implications in our understanding of recognition phenomena in plant biology

When maize is attacked by beet armyworm caterpillars, the whole plant releases a cocktail of volatile terpenoids, which attracts parasitic wasps to the herbivore (*2*). Even a stealthy caterpillar hidden out of view is at risk when the host plant releases its volatile alarm signal. The specialized parasitoid wasps home in on the scent and deposit their eggs into the body of the herbivore in the biological equivalent of a time bomb: the emerging larvae devour their host.

What are the signals that cause plants to release parasitoid-attracting odors? In maize, mechanical damage alone is not sufficient to trigger scent release. Caterpillar regurgitant, however, applied to a mechanical wound causes release of the odor bouquet (*2*). Caterpillar-induced volatile production is an active process, at least in cotton, in which de novo synthesis of terpenoids is required for volatile release (*5*). Considered together, these data indicate the presence of a signal molecule in the herbivore oral secretions; the nature of this molecule has until now remained elusive. Alborn *et al.* (*4*) report the isolation and characterization of *N*-(17-hydroxylinolenoyl)-L-glutamine, or "volicitin," from the regurgitant of beet armyworm caterpillars fed on maize. This molecule triggers the release of the terpenoid cocktail that attracts the parasitic wasp *Cotesia marginiventris* to maize plants under attack by beet armyworm caterpillars (see figure). Volicitin is a powerful elicitor, and a little over 30 pmol of volicitin supplied to 2-week-old maize plants through the transpiration stream elicits volatile release (*5*). Beet armyworm caterpillar regurgitant contains about 20 pmol of volicitin per microliter, so very little saliva would be necessary to elicit terpenoid production (*5*). Volicitin joins an ever-growing family of fatty acid–based biological regulators and has structural features in common with molecules of very



No free lunch. During herbivory, wounding activates the systemic expression of defense genes through the octadecanoid signal pathway (*11*). In parallel, insect saliva containing chemical elicitors, such as volicitin (*4*), triggers the plant to release a bouquet of volatile compounds that attract parasitic or predatory insects to the herbivore. Volatile release stimulated by volicitin may also depend on the octadecanoid pathway, raising the possibility of cross talk between this molecule and the wound-induced expression of defense genes.

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diverse function from at least three kingdoms. These include recently discovered fatty acid primary amines that induce sleep in mammals (*6*), ω-1 hydroxyl fatty acids such as 11-hydroxy-jasmonic acid involved in defense gene signaling in plants (*7*), and small quorum-sensing regulators such as hydroxybutyryl homoserine lactones, which regulate social interaction in bacteria (*8*). The overall construction of the molecule is somewhat reminiscent (with respect to the polar head and less polar fatty acid tail) of the family of Nod factors, powerful developmental regulators produced by nitrogen-fixing plant symbionts (*9*).

Some of the structural features necessary for volicitin function in maize have been identified. The L-enantiomer of glutamine is indispensable for biological activity; volicitin containing D-glutamine is completely inactive (*4*). These molecules will almost inevitably be valuable tools in the search for volicitin receptors in plants.

How then could volicitin act to control the release of volatiles from maize leaves? Some clues may come from a recent study showing that jasmonic acid, a 12-carbon cyclopentanone fatty acid regulator derived from the plant octadecanoid pathway (*7*), can stimulate the release of cocktails of volatile compounds from a wide variety of plants, including maize (*10*). Alborn *et al.* (*4*) suggest that volicitin may interact with the octadecanoid signal pathway to effect volatile release. To test this it will be interesting to see whether plants incapable of responding to jasmonate can still respond to volicitin.

Why should a herbivorous insect make a potent molecule that betrays its presence and may ultimately lead to its demise? Presumably because the molecule has an important function in the insect. Alborn *et al.* (*4*) speculate that volicitin itself may play a hormonal role in beet armyworm, but the fact that it is present in insect oral secretions may imply that it has functions in the process of herbivory. At this stage many scenarios appear possible. For example, one could speculate that volicitin may be useful to the herbivore by disrupting part of a plant defense signal pathway, such as the jasmonate-octadecanoid pathway leading to antidigestive proteinase inhibitor production (*11*). In this way mechanical damage–induced defense gene expression would be reduced, favoring the insect. A function for volicitin in the physiology of the insect gut microflora cannot yet be ruled out.

Whatever its role in beet armyworm, volicitin must be the focus of intense selection pressure and in this respect is reminiscent of another class of elicitors that derive from plant pathogens. These are the avirulence gene products of fungi, bacteria, viruses, and nematodes (as well as a limited number of insects) that trigger the hypersensitive response of defense-related cell death in host plants of cognate genotype. Avirulence elicitors differ greatly in structure, ranging from proteins to low molecular mass fatty acid derivatives (*12*). As with volicitin, the functions of most avirulence gene products in pathogens remain unclear. Could the comparison of avirulence elicitors and volicitin be useful? The hypersensitive response leads to an easily visible phenotype (necrosis) and to powerful resistance. Are there parallels in less easily observed phenomena such as volatile release in tritrophic systems? To address this question and to better understand selection pressures acting on volicitin production, it will be interesting to know whether all maize genotypes respond in the same way to this molecule, whether the molecule is a general elicitor of volatile release in various plant species, and whether different insects produce different volicitins. This work will not only extend our knowledge of plant-insect interaction but will also provide fascinating comparisons with chemical distress signaling in other warring organisms, including vertebrates (*13*).

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NEUROSCIENCE

Adaptation by Hyperpolarization

Horace Barlow

You can get used to anything. This aphorism may not always hold, but it is true that humans (and other animals) have a powerful ability to adapt to stimuli from the outside—a noxious odor, immediately repulsive, becomes hardly noticeable, the sound of traffic no longer annoying. The physiological reflection of this ability is the property of sensory neurons to "adapt" to a sensory stimulus maintained at a high value, giving a response that declines with time. This adaptation is prominent in neurons of the cerebral cortex, and new results reported by Carandini and Ferster (page 949) reveal the surprising way in which these neurons accomplish this adjustment to the environment (*1*).

Adaptation in the neurons of the visual cortex gives rise to optical illusions such as that shown in the figure (see next page). The distortions in the grating occur only in the region of the visual field exposed to the grating and persist for a few seconds or longer if the adaptation period is prolonged. Many similar aftereffects are known, perhaps the most familiar being the "waterfall phenomenon," in which the observer first gazes for a time at the downward stream of water, then looks at the surrounding scenery and sees it apparently drifting upward. These adaptive changes are specific for the pattern. The distortions can be understood in terms of a selective and persistent desensitization of those members of an array of cortical neurons that have been most vigorously excited by the adapting stimulus. Such desensitization has been shown to occur by recording the extracellular currents that accompany the action potentials transmitted by a cortical neuron to other neurons in the brain.

In the new work, Carandini and Ferster used a technique called whole-cell patch recording, in which a fluid-filled electrode terminating in a small pore is pushed up to the membrane of the cell, the rim of the pore making a tight seal with the surface. Electrical contact with the cell's interior is then established by popping the membrane within the seal, enabling the cell's intracellular potential to be continuously recorded. The technique is difficult to perform in the cerebral cortex because tiny movements of the electrode relative to the cell break the seal with the cell surface, but it is possible to record for long enough to find out what happens during and after an adapting stimulus similar to the one in the figure.

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