from the lessons to be drawn from Gaia. The resulting conflict takes precedence over all others. The climate science controversies demonstrate that scientists are now drawn into knowledge and power struggles for which they are not well prepared. Yet, people inspired by Gaia will not necessarily be endowed with deeper foresight. In matters of politics, it is prudent to follow John Dewey's advice (*15*) that we cannot expect to know the best solution in advance, but only that we can improve the quality of the sensors—both instruments and people—that detect shortcomings and the speed with which we rectify the course. If in politics the blind lead the blind, then hope rests on finding the best way to activate the white cane to fumble in the dark.

This is where the scientific establishment will play a crucial role in multiplying the sensors, improving their qualities, speeding the dissemination of their results, improving models, and proposing alternative explanations to phenomena. Such an infrastructure cannot, however, be limited to scientists: They must collaborate with citizens, activists, and politicians to quickly realize where things are going wrong.

Creating an infrastructure of sensors that allows tracking the lag time between environmental changes and reactions of societies is the only practical way in which we can hope to add some self-awareness to Gaia's self-regulation. This framing of the problem gives a clear ethical direction: Any attempt to tamper with the sensors or slow down the reaction to errors jeopardizes the chance to learn from Gaia how to close the loops that would enable Gaia 2.0 to better sustain the human population than the present world. ■

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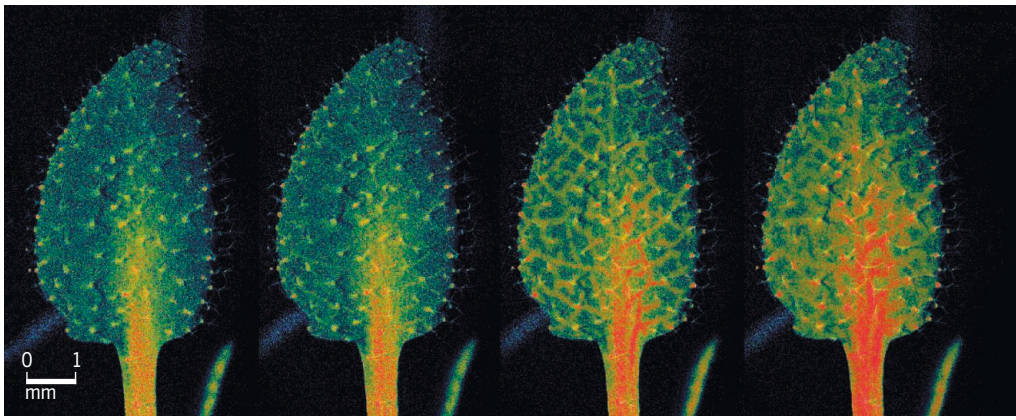

PHOTO: M. TOYOTA *ET AL.* (*1*)

PLANT BIOLOGY

Nervous system-like signaling in plant defense

Herbivory induces rapid long-distance calcium signals through glutamate-like receptors

By **Gloria K. Muday** *and* **Heather Brown-Harding**

The ability to initiate a rapid defense against biotic attacks and mechanical damage is critical for all organisms. Multicellular organisms have developed mechanisms to systemically communicate the occurrence of a wound to help them escape or defend themselves from predators. Because plants are stationary and cannot escape herbivory, they must respond with chemical defenses to deter herbivores and repair damaged tissue. On page 1112 of this issue, Toyota *et al.* (*1*) report long-distance calcium ion signaling in the model plant *Arabidopsis thaliana* in response to caterpillar herbivory or mechanical wounding (see the image). They uncover long-distance calcium signals that require glutamate-like receptor (GLR) channels for signal propagation. These channels are activated by extracellular glutamate, a well-known mammalian neurotransmitter and a more recently uncovered developmental signal in plants (*2*). In mammals, glutamate receptors are central to fast excitatory neurotransmission, which is an intriguing parallel to their role as long-distance signals in wounding and defense in plants.

This study combines genetic and imaging approaches to reveal a rapid and long-distance signaling pathway that communicates leaf damage to intact leaves that are spatially and developmentally distant from the wounded leaf. Toyota *et al.* detect increased calcium signals at the site of both herbivory and mechanical wounding within 2 s and in distant leaves within 2 min after damage. This signal moves through the plant vasculature at rates of ~1 mm/s, which is faster than can be explained by diffusion. This systemic calcium response can be induced through application of glutamate, but not with other amino acids, suggesting a role of GLRs. These GLRs are ion channels that open upon binding glutamate to allow calcium influx. Toyota *et al.* demonstrate that this long-distance signaling is lost in plants with mutations in *GLR3.3* and *GLR3.6*. These GLRs have sequence and structural similarity to mammalian ionotropic glutamate receptors (iGRs), which are critical in learning and memory in mammals, suggesting that very different physiological processes can be mediated by related proteins from the plant and animal kingdoms.

This work builds on detailed structural and functional characterization of mammalian iGRs (*3*). Plant GLRs and animal iGRs have similarities in structure and abundance in genomes (*4*, *5*). The plant GLR genes are classified into three clades: *GLR3.3* and *GLR3.6* are in the third clade and have a "gate" domain, where glutamate is predicted to bind and open the channel, with the highest similarity to mammalian iGRs (*4*). One member of clade 3, the plasma

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The rapid wound-induced spread of a calcium signal is visualized using a genetically encoded reporter. Scale bar, 1 mm.

membrane–localized GLR3.4 protein, is the only plant GLR for which ion channel activity and gating by glutamate has been demonstrated (*6*). Clade 3 GLRs also are the only GLRs to contain a motif found in metabotropic glutamate receptors (mGluRs) (*4*). mGluRs function in a diversity of processes, including responses to pain and noxious stimuli (*7*, *8*), which are related to wounding responses. Whether these particular features of the GLR3s in plants tie them to their function in wounding or other processes awaits further investigation.

An important question raised by this study is, how does this long-distance calcium signal move through the plant? In the mammalian nervous system, glutamate is a local signal that induces a long-distance ionic response. Glutamate is released into the nerve synapse from synaptic vesicles, where it binds to iGRs on the postsynaptic neuron, opening these ion channels and allowing influx of calcium ions and other cations. The ion influx induces membrane potential changes that lead to signal propagation (*3*). The results of Toyota *et al.* suggest an endocrine action of glutamate, which is dissimilar from glutamate as a neurotransmitter in mammals and more akin to a hormonal role of glutamate. This mode of action is supported by the observations that the fluorescent calcium ion reporter GCaMP3 localizes to the vasculature and that GLR3.6 is found in the cells that line the vasculature, where other wound-signaling molecules, such as jasmonates, are synthesized (*9*). A fluorescent glutamate sensor was also used to show that glutamate was increased at the site of wounding; this signal extends along the vasculature and exits the leaf after wounding, which is consistent with a mobile glutamate signal. These results suggest a model in which herbivore and mechanical wounding releases glutamate into the vascular system, where it can travel long distances and activate GLR3 ion channels in the plasma membrane of cells that line the vasculature. This increases the calcium ion influx into these cells. Consistent with this model, GLR3.3 and GLR3.6 were previously shown to be required for systemic wound-activated surface potential changes that are linked to the predicted behavior of these proteins as gated ion channels (*10*–*12*). GLR3 activity is required for the increased synthesis of enzymes of the jasmonate biosynthesis pathway in leaves distant from the wound site (*1*, *10*), leading to the accumulation of jasmonates, which convey host resistance to various invading organisms (*9*). Additional experiments are needed to determine whether the rate of long-distance glutamate movement is sufficient to drive the rapid long-distance calcium changes.

The findings of Toyota *et al.* are only possible because of the powerful advances in microscopy and biosensors that were used for these observations. The authors used two calcium ion sensors and a glutamate sensor visualized by means of imaging approaches that allow detection of the dynamics of rapidly transmitted long-distance signals. The availability of a microscope that can capture a large field of view with a highly efficient camera that is sensitive enough to detect relatively weak fluorescence, and that is capable of time-lapse imaging to capture rapid signal transduction, provides insight that was not previously possible.

"...a rapid and long-distance signaling pathway... communicates leaf damage to intact leaves..."

This combination of approaches has the potential to provide additional evidence for the importance of glutamate and GLRs in plant signaling (*2*). Future experiments are needed to resolve whether glutamate is moving long distances rather than acting via the localized release and long-distance propagation of ionic signals. In addition to measuring long-distance glutamate biosensor changes, another approach is to use labeled glutamate to show that this amino acid is released at wounds and travels through the vasculature to distant undamaged leaves to initiate defense responses. The possibility that glutamate may participate in systemic signaling to modulate developmental and environmental responses across the plant kingdom is an exciting avenue for future studies. ■

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GENETICS

A pinch of RNA spices up DNA repair

Transient incorporation of RNA precursors helps fix broken DNA

By **Mauro Modesti**

When DNA is replicated by DNA polymerases, deoxyribonucleotides are incorporated, whereas when DNA is transcribed by RNA polymerases, ribonucleotides are used. The misincorporation of ribonucleotides into DNA occurs frequently during DNA replication (*1*). However, the presence of ribonucleotides in DNA makes it more fragile and threatens genome stability that needs to be maintained for faithful transmission of genetic information. To counteract this, cells have evolved efficient ribonucleotide removal strategies that rely on ribonuclease H2 (RNase H2) (*2*). On page 1126 of this issue, Pryor *et al.* (*3*) report the surprising discovery that ribonucleotides are frequently incorporated at broken DNA ends, which enhances repair. This important finding overturns the central dogma of molecular biology by demonstrating that transient incorporation of ribonucleotides in DNA has a biological function.

DNA double-strand breaks (DSBs) in the genome arise through the action of external agents such as exposure to ionizing radiation or clastogenic chemicals. They are also caused endogenously by reactive oxygen species produced during oxidative metabolism or when DNA replication forks collapse. In addition, DSBs initiate programmed genome rearrangements including V(D)J recombination in developing lymphocytes that shuffles antigen and T cell receptor gene segments during the adaptive immune response; or during the process of meiotic recombination in germ cells. DSBs are repaired either by homologous recombination or by nonhomologous end joining (NHEJ) path-

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