tissue through the taste cells or through the tight junctions, but the negatively charged acetate and gluconate ions were unable to follow at the same rate.

Sodium chloride, on the other hand, gave only a small change in the voltage, because chloride (a smaller ion than acetate or gluconate) followed sodium through the tight junctions into the epithelium, where its negative charge acted to balance some of the sodium's positive charge.

Furthermore, the team found that the voltage differences were correlated with the nerve's response to salts: the bigger voltage changes associated with gluconate and acetate ions reduced the nerve's response. That makes sense, says DeSimone, because a lot of positive charge building up around the taste cells inside the epithelium would increase the voltage across the taste cell membrane, creating a condition called hyperpolarization. Hyperpolarization would, in turn, make it more difficult for sodium to trigger the electrical excitation that stimulates the nerve. And that would neatly explain why it requires a higher concentration of sodium acetate or gluconate than of sodium chloride to get the same degree of "salty" taste.

"So far everything was consistent," says DeSimone, "but that wasn't the final proof. The final proof is to go in there and actually control that [voltage]. Don't let the salts determine it, you determine it." If the anions act by influencing the voltage across the epithelium, DeSimone and his colleagues reasoned, they should be able to hold that voltage constant—and in that way equalize the responses to all sodium salts. When they did the experiment, that's what they found. They used a technique called a voltage clamp, in which they injected current to keep the voltage steady. A constant voltage proved to be the great equalizer: the nerve's response to all the salts was the same, confirming the hypothesis that the flow of anions through tight junctions was causing the taste differences.

"It's an excellent study," says taste physiologist David Hill of the University of Virginia. "The results seem clear to everyone in the field." Questions remain about how the tight junctions restrict the flow of ions, points out Susan Schiffman, who studies the anion effect at Duke University. But that focus on the tight junctions is an important outcome of the work, says Delaware's Mierson—a view that goes beyond the solution of the anion paradox to address the bigger question of how the tongue's epithelium functions as a tissue. "The work treats the whole thing as a system," she says, and highlights the fact that important functions reside not only in cells but in the spaces between them. ■ MARCIA BARINAGA

How Plants Cope With Stress

When an insect munches on a plant the last thing it's looking for is a bad case of indigestion. But that's often what it gets, the result of a natural defense mechanism in which plants may produce proteinase inhibitors that disrupt the digestion of feeding insects, thereby encouraging them to seek their sustenance elsewhere. Despite the importance of this stress response to plant defenses—and the possibility that a better understanding of how the system operates might aid in the design of better pest control strategies—plant biologists were unable to identify the compound that triggers this stress response.

Now, in work described at last month's Third International Congress for Plant Molecular Biology in Tucson, Arizona, and also in a paper in *Science* (23 August, p. 895), biochemist Clarence Ryan and his colleagues at Washington State University in Pullman have isolated a new plant compound that seems to do the job. Even more exciting, the researchers have filled a major gap in plant biology. Previously, all the known plant hormones had been relatively simple compounds. Indeed, plants were thought to lack the capacity to use the more sophisticated polypeptide and protein hormones that so commonly serve to coordinate physiological responses in animals. But the molecule identified by the Ryan group is a polypeptide that behaves very much like animal polypeptide hormones. Says molecular biologist Mich Hein of the Scripps Research Institute in La Jolla, California, this discovery will prompt researchers to seek similar compounds in plants. "There have got to be more of these things," he notes.

CLARENCE RYAN

Dining out. *This tobacco hornworm may get more than dinner from tomato leaves.*

Twenty years ago, however, when Ryan began his search for the stress-triggering molecule, the idea that plants don't use sophisticated signaling molecules such as polypeptides was so ingrained that he never even thought about polypeptides. Instead, he focused on known plant hormones, including the auxins and cytokinins, which regulate growth and development, and also on carbohydrates, a logical choice for the stress signal molecule because they can be released from plant cell walls broken down by feeding insects. The trouble was none of these simple compounds worked. "It became clear that we had an unusual signal," Ryan says.

About 4 years ago then, he decided to take a different tack, ridding the plant extracts of all carbohydrates, as well as of the auxins and cytokinins, and looking for an entirely new class of compounds. The approach paid off. Eventually, Ryan's team, including Gregory Pearce, Daniel Strydom, and Scott Johnson, managed to isolate 1 microgram of a material that stimulates proteinase inhibitor production from about 60 pounds of tomato leaves. The material's chemical nature? It proved to be a peptide containing 18 amino acids. After watching how this newly identified peptide behaved in plants, the researchers dubbed it "systemin" because it is transported systemically throughout the plant, just as animal hormones are. Systemin also resembles animal peptide hormones in that it is very potent—as little as one part in 10 trillion can trigger a plant stress reaction—and it is synthesized as part of a larger precursor protein, from which it must be cleaved.

The next question was: How does systemin elicit plant stress reactions? And there Ryan says that his postdoc Edward Farmer may have hit on a clue when he showed that methyl jasmonate, a plant lipid, induces the production of proteinase inhibitors, just as systemin itself does. This was somewhat of a surprise, since methyl jasmonate had not previously been shown to be part of the stress response pathway. But in light of Farmer's results, Ryan speculates that systemin may work by stimulating the synthesis of methyl jasmonate, which is made from linolenic acid that can be released from membrane lipids by a lipase enzyme. More work will be needed to verify this scheme, Ryan cautions, but even if all the steps in the model are not confirmed, one thing is sure. Plants no longer have to take a back seat to animals when it comes to the sophistication of their hormonal pathways. ■ ANNE SIMON MOFFAT