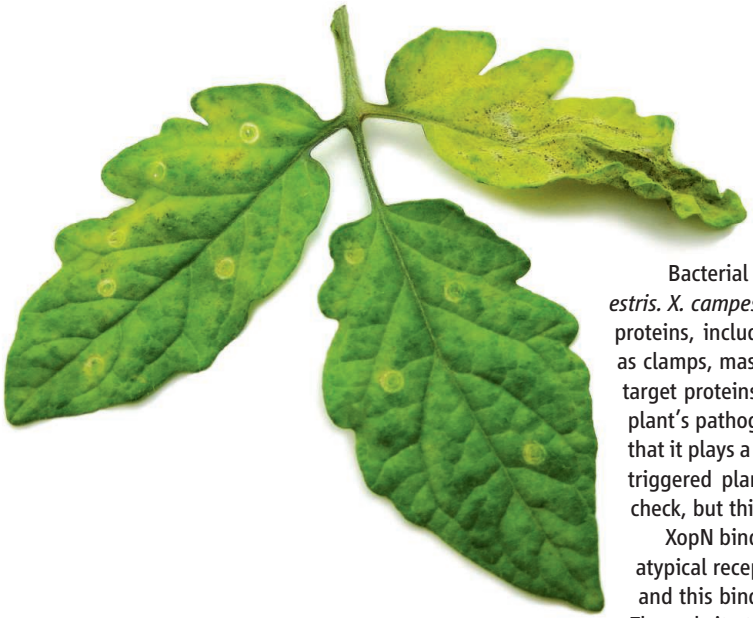


PLANT SCIENCES

Pathogens Double Down

Bacterial spot that affects tomato and pepper plants is caused by *Xanthomonas campestris*. *X. campestris* delivers a protein effector called XopN into plant cells that binds to other proteins, including the 14-3-3 protein in tomatoes, TFT1. 14-3-3 proteins serve variously as clamps, masks, and scaffolds to regulate and organize the functions of a wide variety of target proteins. Taylor *et al.* studied how the interaction of XopN with TFT1 modulated the plant's pathogen defense response. Silencing of the gene that encodes TFT1 demonstrated that it plays a protective role against bacterial infection and regulates four other pathogen-triggered plant genes. TFT1 was able to keep the growth of XopN-deficient bacteria in check, but this effect was abrogated in the absence of TFT1.

XopN binds to TFT1 through its C-terminal domain. XopN also binds to TARK1, a tomato atypical receptor kinase that plays a role in plant defense, through its N-terminal domain, and this binding interaction seems to help the XopN C-terminal domain hold on to TFT1. Through its two-domain binding system, the bacterial XopN effector sequesters the two plant-defense proteins. It is not clear yet just how TFT1 and TARK1 defend the plant normally, but what is clear is that when XopN grabs both of them together, the plant's defense collapses. — PJH

PLoS Pathog. **8**, e1002768 (2012).

IMMUNOLOGY

The Limits in Infant Immunity

Natural killer (NK) cells control viral infections swiftly, releasing lytic factors that destroy infected cells shortly after infection. But infants and neonates are susceptible to viral infections in part because they lack the mature form of these powerful immune cells. Marcoe *et al.* have discovered a factor that limits this arsenal early in life. The authors found that during mouse infancy, transforming growth factor–β (TGF-β) blocks a terminal step in NK cell maturation. TGF-β blocked the generation of mature NK cells from mouse stem cell precursors in vitro. In mice that were genetically engineered to lack a functional receptor for TGF-β in NK cells, the number of mature NK cells present at 10 days of age was equivalent to that in 56-day-old normal mice. In addition to faster maturation, infant mice lacking NK cell TGF-β receptor signaling were resistant to viral infection. Analysis of mRNA points to genes that control the cell division cycle—*p21* and *Cdc7*—as targets of TGF-β, arresting the production of NK cells as they mature. The expression of transcription factors that push NK cells through the final stage of maturation is also limited by TGF-β. The findings raise the possibility that inactivating TGF-β signaling could prevent the deficit of NK cells during infancy. — LC

Nat. Immunol. **11**, 10.1038/ni.2388 (2012).

CELL BIOLOGY

Stretch Relief

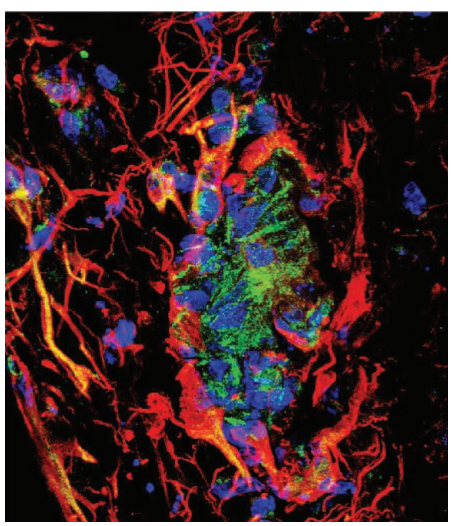
The microtubule network links mechanical stretching in heart cells to intracellular signaling events mediated by changes in concentration of reactive oxygen species (ROS) and of intracellular free calcium ions. Khairallah *et al.* show that a similar signaling system may have a role in the malfunction of muscle cells in Duchenne muscular dystrophy (DMD). In a mouse model of DMD, muscle cells generated ROS and accumulated more intracellular calcium than did normal cells in response to stretching. These responses were dependent on microtubules. Mechanical stress caused damage to dystrophic mouse muscle, but treatment of mice in vivo with an inhibitor of ROS production or a microtubule depolymerizing drug protected muscle function, perhaps offering a strategy of therapeutic benefit in this deadly disease. — LBR

Sci. Signal. **5**, ra56 (2012).

IMMUNOLOGY

Taming Microglia

Multiple sclerosis (MS) is a severely debilitating degenerative disease of the central nervous system. Resident macrophages of the brain, called microglia, are thought to be an important driver of disease. Factors that promote the conversion of proinflammatory, or so-called "M1" microglia, which are thought to be the type of microglia that contribute to disease, into less dangerous, immunoregulatory "M2"-type microglia, are of therapeutic interest. Starossom *et al.* identified one such factor, the endogenous glycan-binding protein Galectin-1 (Gal1). In a mouse model of MS, Gal1 was expressed during the acute and chronic stages of disease by astrocytes and some populations of immune cells. Gal1 bound preferentially to M1 microglia in a glycan-dependent manner, and once bound, it inhibited the proinflammatory phenotype of M1 microglia by retaining the phosphatase CD45 on the cell surface. This resulted in the



dephosphorylation, and therefore downmodulation, of several downstream proinflammatory signaling molecules. The effects of Gal1 on M1 microglia were primarily the result of astrocyte-produced Gal1. Finally, the authors showed that mice deficient in Gal1 experienced enhanced axonal damage, whereas treatment of mice with Gal1-treated microglia or with Gal1 itself had a therapeutic effect, which suggests that Gal1 may be a potential therapeutic target in MS. — KLM

Immunity **37**, 10.1016/j.immuni.2012.05.023 (2012).

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CREDITS (TOP TO BOTTOM): K. W. TAYLOR *ET AL.*, *PLoS PATHOG.* **8**, 6 (2012); S. STAROSSEM *ET AL.*, *IMMUNITY* **37**, 10.1016/J.IMMUNI.2012.05.023 (2012)

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MICROBIOLOGY

Trading Defense for Virulence

Like larger organisms, bacteria are plagued by viruses and other mobile entities bringing in foreign DNA that may be damaging. Bikard *et al.* recognized that *Streptococcus pneumonia* (pneumococcus) lacks the CRISPR-endoribonuclease mechanism that has evolved to defend against bacteriophage infection and plasmid conjugation. Lateral gene transfer between unrelated species is a fundamental characteristic of prokaryote evolution, however, and therefore CRISPR loci could block the transfer of traits. Pneumococci rely on exogenous DNA to endow them with virulence and antibiotic resistance through direct uptake of DNA, and this may be why CRISPR is absent in this species. CRISPR sequences can be experimentally introduced into pneumococcus that prevent the pathogen from capsule-switching—a key trait that allows this bacterium to dodge host immune responses—and allow mice to survive infection with CRISPR$^+$ pneumococci. Interestingly, during the experiments, pneumococci emerged that could inactivate the introduced CRISPR, indicating that selection pressure to avoid immune responses is strong enough to leverage reacquisition of the ability to scavenge for foreign genes in this species. — CA

Cell Host Microbe **12**, 177 (2012).

PHYSICS

Perfect Transmission

In quantum mechanics, when a particle encounters a potential energy barrier that it cannot classically overcome, most of the time that particle will be reflected, but some of the time it will be able to make it through to the other side. The probability of tunneling is generally less than 1; however, in the special case where there is dissipation in the system and instead of a single barrier there is a double barrier supporting a resonant state, perfect transmission has been predicted to occur under certain circumstances. Mebrahtu *et al.* used a carbon nanotube coupled to two resistive (dissipative) leads to realize this system; two side gates controlled the coupling between the nanotube and the two leads. When the couplings were asymmetrical, there was the usual decreasing dependence of resonance conductance with lowering temperature. For exactly even couplings, the behavior was the opposite of that, and the conductance at the lowest measured temperature showed perfect transmission. The authors interpreted their results as a quantum phase transition of the unusual "boundary" type, where only a local part of the system undergoes the transition. — JS

Nature **488**, 61 (2012).

CHEMISTRY

Allergenic Terpenoids

Common ragweed is native to North America but has become widespread in most temperate regions around the world. It can cause strong allergic reactions in people with hay fever, including severe irritation of the airways; however, little is known about the secondary metabolites from this plant and their involvement in the allergic response. Taglialatela-Scafati *et al.* have analyzed the composition of the aerial parts of common ragweed collected from an urban environment in the outskirts of Novara, Italy. Common ragweed samples collected only 100 m away from each other had different phytochemical profiles, illustrating the heterogeneity of the European populations of this plant. Along with numerous known compounds, the authors discovered eight unknown sesquiterpenoids and show that these compounds are activators of TRPA1, a sensor that is a major player in the induction of inflammatory pathologies of the airways. Samples of common ragweed pollen were too small to allow the identification of specific constituents, but the chemical characteristics of the pollen extract suggest that the pollen also contains these secondary metabolites. Low-molecular terpenoids may thus play a role in the allergic response to common ragweed. — JFU

Eur. J. Org. Chem. 10.1002/ejoc.201200650 (2012).

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