PLANT INFECTION

A decoy tactic

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AAAS

Pathogen infection of plants results in the recognition of pathogen-associated molecular patterns (PAMPs) by the host, leading to a defense response. The soybean pathogen *Phytophthora sojae*, which causes soybean root and stem rot, releases a glycoside hydrolase protein, PsXEG1, during infection that is recognized as a PAMP by the host. To further understand how soybeans might counteract PsXEG1, Ma *et al.* used a proteomic approach in *P. sojae*–infected soybeans and identified a xyloglucan-specific glucanase inhibitor protein (GmGIP1) as a direct binder of PsXEG1. GmGIP1 blocked PsXEG1-mediated enzymatic activity and infection. GmGIP1 also interacted with the related XEG-like protein 1 (PsXLP1), which promoted pathogen virulence. Interestingly, transgenic *P. sojae* lines expressing a PsXLP1 catalytic mutant resembled wild-type *P. sojae*, while lines deficient in GmGIP1 binding showed reduced virulence, suggesting that the interaction of PsXLP1 with GmGIP1 was essential for pathogen infection. ITC binding studies demonstrated that the interaction between GmGIP1 and PsXLP1 was five times stronger than the GmGIP1–PsXEG1 interaction. The authors proposed that the coexpression of PsXLP1 with PsXEG1 during infection results in PsXLP1 binding to GmGIP1, freeing PsXEG1 to promote infection. Given that homologs of PsXLP1 and PsXEG1 are found in other *Phytophthora* species, it is possible that this decoy mechanism may be a conserved strategy of counter-defense by pathogens against plants. *GM*

ENZYME MECHANISMS

Fickle about fluorine

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Supplying a multi-enzyme polyketide synthase (PKS) complex with unnatural monomer units can, under certain conditions, facilitate the production of novel polyketide natural product analogs. Using several different extender units, including one containing fluorine, and a variety of multi-domain enzymatic constructs, Ad *et al.* have explored the mechanisms controlling the incorporation of alternate moieties into the polyketide backbone of erythromycin by the 6-deoxyerythronolide B synthase (DEBS) PKS complex. While inactivation of a *cis*-acyltransferase (AT) domain in DEBS allowed the incorporation of non-native extender units by removing the enzyme's selectivity for the native substrate, it also caused a switch in the mechanism of C–C bond formation that untethered the growing polyketide chain from its carrier protein, leading to premature chain termination and product release. However, complementation of the inactive DEBS *cis*-AT with an orthogonal *trans*-AT restored the canonical PKS mechanism and enabled further chain extension following the incorporation of alternative extender units. Use of this engineered hybrid PKS system enabled the efficient production of polyketides with site-specific incorporation of fluorine, such as mono- and bisfluorinated triketide lactones. This detailed understanding of the mechanisms of elongation in an engineered PKS will enable the biosynthesis of novel polyketides, though it remains to be seen how well these insights will translate to other PKS complexes. *CD*

METABOLISM

A-way with biofilms

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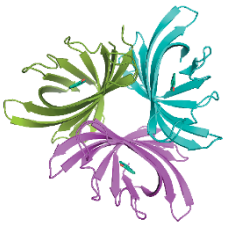

AAAS

Phenazines are redox-active metabolites produced by *Pseudomonas aeruginosa* to control virulence. While toxic to other cells, phenazines support extracellular electron transfer that promotes survival of the producer in anoxic conditions, facilitating biofilm development. In defining strategies to manipulate phenazine levels to control biofilm formation, Costa *et al.* characterized a *Mycobacterium fortuitum* enzyme, PodA, that catalyzes pyocyanin degradation via specific demethylation of N-methylated phenazines. Unlike typical demethylases, the mechanism of PodA does not proceed by electron transfer to a flavin cofactor or an iron–sulfur cluster during oxidation of the pyocyanin methyl group to formaldehyde. Instead, under anoxic conditions, PodA forms a reduced phenazine, and the substrate serves as the electron acceptor, which the authors verified by X-ray crystallography. The authors also tested their mechanistic model through mutagenesis of putative acid and base residues within PodA. Application of PodA could reduce formation of *P. aeruginosa* biofilms, presumably via metabolism of pyocyanin, which has a known role in driving release of extracellular DNA for the construction of biofilms. PodA also decreased anoxic fitness in biofilms by disrupting pyocyanin-dependent electron shuttling to oxygen. Manipulation of extracellular electron shuttles may be a viable strategy for controlling pathogenic biofilms. *MB*

mRNA LOCALIZATION

If you have to ASH

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Many eukaryotic mRNAs are transported from the nucleus, where they are produced, to distal subcellular locations where they can be locally translated. In *Saccharomyces cerevisiae*, *ASH1* mRNAs are moved to the tip of the daughter cell during mitosis, a process that requires She2p and She3p. Edelmann *et al.* have investigated the structures of *ASH1* mRNA on its own, in the presence of She2p, and in the presence of She2p and a C-terminal fragment of She3p. In the absence of protein, the mRNA forms a straight but flexible stem–loop architecture; the presence of She2p leads to the formation of a large 'kink' in the structure of the mRNA, which is critical for transport. The addition of the C-terminal fragment of She3p did not alter the overall structure of the She2p–*ASH1* mRNA complex, but further stabilized the complex via interactions with She2p and the mRNA. The authors identified several amino acids that appeared to be essential for mRNA recognition and binding, and they confirmed the importance of these protein–mRNA interactions *in vivo*. Additional work is needed to determine the structure of the entire She3p–She2p–*ASH1* mRNA complex in the presence of myosin and to probe other possible physiological roles for the flexibility of the mRNA. *JMF*

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