PLANT MICROBIOTA

Root recruiting

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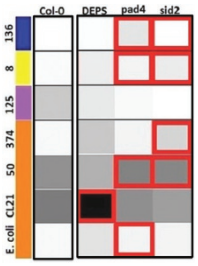
SCIENCE



The microbial communities existing among plants contain not only pathogenic species but also potentially beneficial ones. How plants signal to recruit beneficial species has not been clarified by studies that have examined the microbes present adjacent to plant roots (rhizosphere) and within the roots (endophytic compartment). Lebeis *et al.* reasoned that phytohormones that promote plant defense to pathogens could be at play, so they compared the bacterial root microbiomes present with wild-type *Arabidopsis thaliana* to those present with mutants that cannot produce salicylic acid (SA), jasmonic acid or ethylene or that lack the ability to respond to these phytohormones. Endophytic bacterial communities were less diverse, both at the level of individual species and at higher taxonomic orders, than were those from the rhizosphere, and they had altered compositions (both in number and identity) in the presence of the mutant plants. Comparing the composition patterns across the different mutants, the authors concluded that defense phytohormones, especially SA, modulate taxonomic groups of bacteria at the family level in the root. SA does so by affecting the whole population at the family level, not just a few dominant strains. Lebeis *et al.* proposed that SA is required to modulate the composition of a normal root microbiota. Similar conclusions were supported by the next set of experiments, in which the authors exposed sterile roots of wild-type and mutant plants to a synthetic community (SymCom) of 38 documented bacterial strains across four phyla, and saw increased abundance of endophytic bacterial isolates after eight weeks. Again, the mutants deficient in SA differed from the wild-type plants. Application of exogenous SA also altered the compositions of the bacterial communities. These results suggest that SA influences the structure of the microbial community on the plant root perhaps through a direct recruitment, which could prove useful for agricultural applications. *MB*

CANCER METABOLISM

Two sides of a coin

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The metabolic enzymes isocitrate dehydrogenases 1 and 2 (IDH1/2) normally catalyze the interconversion of isocitrate and α-ketoglutarate (α-keto). Somatic gain-of-function mutations in IDH1/2 result in production of the D enantiomer of 2-hydroxyglutarate (D2HG), a known onco-metabolite that prevents differentiation of cancer cells. The levels of D2HG and of the L enantiomer L-2-hydroxyglutarate (L2HG) are further regulated by D-2-hydroxyglutarate dehydrogenase (D2HGDH) and L-2-hydroxyglutarate (L2HGDH), respectively, which convert D2HG and L2HG back to α-keto. However, the potential sources and functions of L2HG in normal and malignant cellular metabolism were poorly understood. In a search to identify metabolites that are regulated under low-oxygen conditions, Oldham *et al.* and Intlekofer *et al.* performed mass spectrometry analysis of mammalian cells under hypoxic conditions and detected elevated levels of 2-hydroglutarate (2-HG). Surprisingly, both groups resolved the 2-HG enantiomers and discovered that L2HG and not D2HG levels were increased in hypoxic cells. Consistent with this, inhibition of L2HGDH increased 2-HG production whereas overexpression of L2HGDH lowered 2-HG levels under low oxygen conditions. Both studies showed a contribution of malate dehydrogenase (MDH) towards L2HG production through reduction of 2-oxoglutarate. Knockdown of either MDH isoforms decreased hypoxia-mediated L2HG production. In addition, Intlekofer *et al.* proposed that lactate dehydrogenase served as a major enzymatic source of L2HG production by utilizing glutamate-derived α-KG. Using a genetically encoded fluorescent sensor, Oldham *et al.* found that L2HG altered the cellular redox status by increasing the $NADH/NAD^+$ ratio while also functioning as a suppressor of oxygen consumption and glycolytic flux. Moreover, Intlekofer *et al.* demonstrated that hypoxia-induced L-2HG alters histone methylation by inhibiting α-keto-dependent histone demethylases. Overall, these findings suggest that manipulation of L2HG production represents a novel strategy to influence cellular responses to hypoxic stress. *GM*

CELL CYCLE REGULATION

Redox shielding

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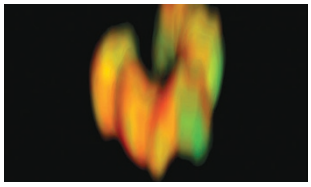
DONGMIN KANG



The regulation of the anaphase-promoting complex/cyclosome (APC/C) between kinases and phosphatases dictates the stability of cell cycle mediators. In particular, the cyclin-dependent kinases (Cdks) phosphorylate an APC/C activating-protein Cdh1, while the protein tyrosine phosphatase Cdc14B opposes this modification. These cell cycle regulators are localized at the centrosome, which is the main microtubule-organizing center of animal cells required for mitotic progression. Recent studies have introduced redox regulation into this interplay as the Cdk-cyclin B complex has been observed to phosphorylate and inhibit the hydrogen peroxide (H_2O_2)-degrading enzyme peroxiredoxin I (PrxI). In addition, changes in H_2O_2 levels are known to modulate cell cycle progression. To determine the temporal regulation between PrxI and H_2O_2, Lim *et al.* investigated how PrxI is regulated during the cell cycle. Interestingly, they found that PrxI is phosphorylated at early mitotic centrosomes but becomes dephosphorylated after anaphase onset, hinting that H_2O_2 accumulates at early mitotic centrosomes and the centrosome-associated H_2O_2 promotes mitotic entry. Consistent with this idea, reduction of centrosomal H_2O_2 levels through the expression of centrosome-targeting catalase, a H_2O_2-metabolizing enzyme, delayed mitotic entry. Biotinylation labeling studies revealed that Cdc14B is highly sensitive to H_2O_2-dependent oxidation. From these observations, the authors suspected that Cdc14B might be targeted for inhibition through oxidation. Cdc14B oxidation presumably reduced its phosphatase activity, thus resulting in the maintenance of Cdh1 serine phosphorylation that prevents interactions with the APC/C complex. Lowering H_2O_2 levels through centrosomal catalase expression also decreased Cdh1 serine phosphorylation. Hence, H_2O_2-mediated Cdc14 inactivation, and therefore Cdh1 phosphorylation during early mitosis, are likely to be critical to ensuring normal M-phase progression by preventing precocious APC/C activation. *GM*