ECOLOGY

Soil immune responses

Soil microbiomes may be harnessed for plant health

By **Jos M. Raaijmakers**[1,2] *and* **Mark Mazzola**[3]

Soil microorganisms are central to the provision of food, feed, fiber, and medicine. Engineering of soil microbiomes may promote plant growth and plant health, thus contributing to food security and agricultural sustainability (*1*, *2*). However, little is known about most soil microorganisms and their impact on plant health. Disease-suppressive soils offer microbiome-mediated protection of crop plants against infections by soil-borne pathogens. Understanding of the microbial consortia and mechanisms involved in disease suppression may help to better manage plants while reducing fertilizer and pesticide inputs.

There are two types of disease suppression in soils. General suppression is based on competitive activities of the overall micro- and macroflora and is universal to all soils.

"The complexity of soil microbiome-plant interactions argues for [taking] a community perspective."

Specific suppression is attributed to the enrichment of specific subsets of soil microorganisms. Specific suppression has been reported for plant pathogenic fungi, fungal-like oomycetes, bacteria, nematodes, and parasitic weeds. It is eliminated by soil pasteurization or biocides and can be transferred to conducive soils, in which only general suppression is operative, via soil transplantations. When Henry first reported transplantation of disease-suppressive soils 85 years ago, he elegantly showed that specific suppression of *Helminthosporium* foot rot of wheat was most likely

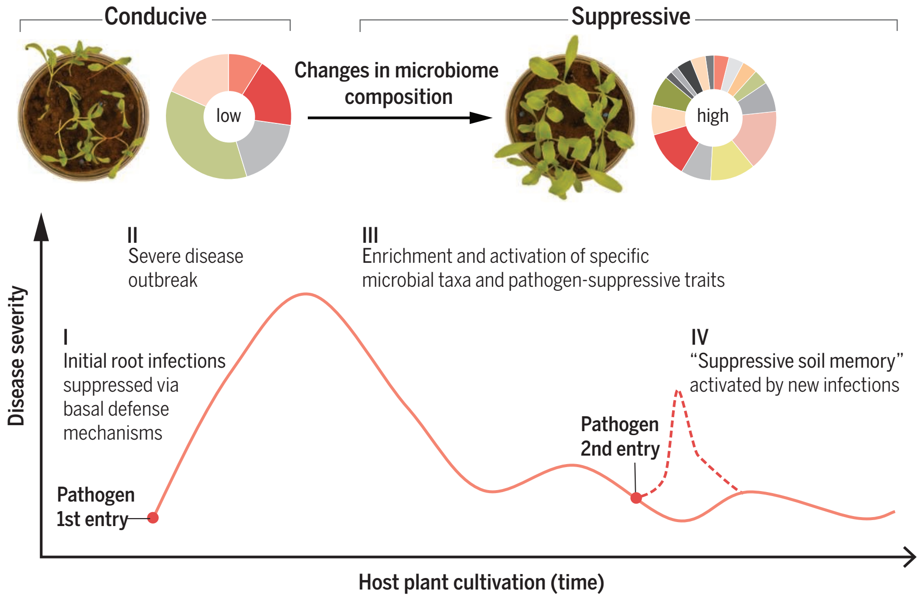
[1]Netherlands Institute of Ecology, Royal Netherlands Academy of Arts and Sciences, Wageningen, Netherlands. [2]Institute of Biology, Leiden University, Leiden, Netherlands. [3]U.S. Department of Agriculture–Agricultural Research Service, Wenatchee, WA, USA. Email: j.raaijmakers@nioo.knaw.nl

caused by the combined action of soil bacteria and fungi (*3*).

Specific suppression of various fungal root pathogens is typically induced by a disease outbreak that occurs in field soils during continuous cultivation of a susceptible host plant. Once established, specific suppression can dissipate if nonhost plants are grown or other root diseases emerge. It is rapidly regained in the presence of the original host plant and inducing pathogen (see the figure).

The characteristics of general and specific suppression of soils are comparable to those described for innate and adaptive immunity the plant, which in turn enrich and activate pathogen-suppressive microbes (*4*).

Specific suppression of several fungal root pathogens has been attributed, in part, to the production of antifungal metabolites by different bacterial genera (*5*, *6*) and to carbon competition and induced systemic resistance by nonpathogenic fungi (*7*, *8*). Kinkel *et al*. have implicated *Streptomyces* species in suppression of scab, a bacterial disease of potato (*9*). Olatinwo *et al*. have proposed parasitism by the fungus *Dactylella oviparasitica* as a key mechanism in suppression of a plant pathogenic nematode (*10*). Although



Lines of defense. If a pathogen can circumvent the basal defenses in both soil and plant, a severe disease outbreak may occur. This disease outbreak can last for years but will ultimately enrich for specific microbial consortia and pathogen-suppressive traits in the soil and plant microbiome. This specific suppression can dissipate but is rapidly regained in the presence of the original host plant and inducing pathogen. The images show plants exposed to a fungal pathogen in disease-conducive and -suppressive soils. In the conducive soil with a low abundance of antagonistic microbial consortia, the fungal pathogen causes disease (left), whereas in the suppressive soil with a high abundance of antagonistic microbial consortia, most seedlings remain healthy (right).

in animals. Both general suppression of soils and innate immunity in animals provide a fast, nonspecific line of defense against an invading pathogen. Both specific suppression of soils and the adaptive immune response in animals require time to react to the invading pathogen, are specific to the pathogen, and have a memory of the previously encountered pathogen (see the figure).

Specific suppression is mechanistically complex, requiring enrichment and activation of select microbial consortia and antagonistic traits that interfere with the infection cycle. Eliciting specific suppression requires multilateral interactions between pathogen, host plant, and soil microbiome. The initial interaction between pathogen and plant, leading to a disease outbreak, may cause the release of metabolites from the pathogen and the interactions in soils suppressive to a specific pathogen are biologically complex, the mechanisms appear to be the same in different soils from geographically distinct regions (*11*). This functional similarity across many agroecosystems suggests that it may be possible to develop a universal approach to engineer disease-suppressive soil microbiomes.

Molecular and chemical technologies now allow identification of differences in microbiome composition between suppressive and conducive soils beyond the description of select microbial genera. They further enable comprehensive analyses of the temporal changes in microbiome activities as the soil shifts from the conducive to the disease-suppressive state. This knowledge also allows elucidation of the mechanisms that lead to the onset of specific disease suppression.

GRAPHIC: N. CARY/*SCIENCE*

Studies of disease-suppressive soils have not yet yielded far-reaching solutions to soil-borne disease management and enhancing crop productivity. Rather, the main outcome has been the isolation of single microbial species subsequently applied to soil or plant seeds as biological agents for pathogen control. Many of these microbial strains fail to establish or survive in soil or on plant roots because of competition with the indigenous soil microbiome. As a result, this approach has met with limited success in large-scale agriculture. The complexity of soil microbiome–plant interactions argues for new strategies that go beyond "one-microbe-at-a-time" approaches and take a community perspective. This includes the design and application of mixtures of different microbial species, referred to as synthetic communities or syncoms (*12*). A second strategy involves augmenting indigenous disease-suppressive consortia native to the soil ecosystem. Engineering such indigenous microbial consortia could yield a more stable soil memory that limits pathogen infestations.

Practical means to attain this outcome in sustainable disease management include selection of or breeding for plant genotypes with specific root traits that recruit or activate pathogen-suppressive microbial populations (*12*, *13*). Agricultural production system inputs, including soil amendments such as compost and seed meal, can also be used, like prebiotics in humans, to selectively drive the microbiome to a composition and active state that limits proliferation of soil-borne pathogens (*14*). To this end, fundamental knowledge of coevolutionary trajectories in plant-pathogen-microbiome interactions is needed (*10*). Mechanistic understanding of specific plant metabolites and pathogen effectors that trigger, like vaccines in animals, the adaptive immune response of soils may provide practical means to engineer the indigenous soil microbiome for enhancing plant health and securing future crop yields. ■

REFERENCES

1. U. G. Mueller, J. L. Sachs, *Trends Microbiol.* **23**, 606 (2015).
2. S. F. Bender, C. Wagg, M. G. A. van der Heijden, *Trends Ecol. Evol.* **31**, 440 (2016).
3. A. W. Henry, *Can. J. Res.* **4**, 69 (1931).
4. E. Chapelle, R. Mendes, P. A. H. M. Bakker, J. M. Raaijmakers, *ISME J.* **10**, 265 (2015).
5. R. Mendes *et al.*, *Science* **332**, 1097 (2011).
6. J.-Y. Cha *et al.*, *ISME J.* **10**, 119 (2016).
7. P. Lemanceau, C. Alabouvette, *Biocontrol Sci. Technol.* **3**, 219 (1993).
8. R. P. Larkin, D. L. Hopkins, F. N. Martin, *Phytopathology* **86**, 812 (1996).
9. L. L. Kinkel *et al.*, *Res. Microbiol.* **163**, 490 (2012).
10. R. Olatinwo, B. Yin, J. O. Becker, J. Borneman, *Phytopathology* **96**, 111 (2006).
11. D. M. Weller *et al.*, *Annu. Rev. Phytopathol.* **40**, 309 (2002).
12. S. L. Lebeis *et al.*, *Science* **349**, 860 (2015).
13. M. Mazzola, D. L. Funnell, J. M. Raaijmakers, *Microbial Ecol.* **48**, 338 (2004).
14. M. Mazzola *et al.*, *Phytopathology* **105**, 460 (2015).

10.1126/science.aaf3252