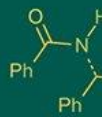


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A review on plant microbiome engineering for sustainable disease management

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Abstract

Plant disease management is undergoing a conceptual shift from chemical suppression of pathogens toward deliberate manipulation of the microbial communities that inhabit roots, leaves, and internal plant tissues. Three broad engineering routes are examined, the recruitment of native disease-suppressive consortia through root exudation and rhizosphere conditioning, the deliberate assembly of synthetic microbial communities designed around defined functional traits, and the induction of systemic resistance pathways by root-associated microbes acting at a distance from infection sites. Considerable disagreement exists over whether laboratory-derived microbiome function transfers to field soils with their own resident microbial legacy, and over whether synthetic community diversity or targeted keystone strains offer more durable protection. Field has matured in mechanistic sophistication faster than it has matured in translational reliability, and that closing this gap requires standardised metrics of colonisation persistence, greater attention to soil-type interactions, and honest reporting of negative or context-dependent results rather than continued accumulation of proof-of-concept studies.

Keywords: Rhizosphere microbiome, induced systemic resistance, synthetic microbial communities, disease-suppressive soils, biocontrol, plant-microbe interactions

Introduction

For much of the twentieth century, plant disease management operated on an essentially adversarial model, the pathogen was identified, and a chemical or resistant cultivar was deployed against it. That model has delivered enormous yield gains, yet the accumulating costs of fungicide resistance, residue regulation, and the narrowing genetic base of resistance breeding have pushed researchers toward a different premise, that a plant's health depends not only on its own genome but on the community of microorganisms occupying its rhizosphere, phyllosphere, and internal tissues (Berendsen *et al.*, 2012; Vandenkoornhuyse *et al.*, 2015) [3, 20]. Trivedi *et al.*, (2020) [19] framed this shift explicitly by describing the plant and its associated microbiota as a single ecological and evolutionary unit, the holobiont, in which microbial partners contribute functions the host genome alone does not encode, among them protection from soil-borne and foliar pathogens.

The evidence that root-associated microbiomes can suppress disease is not new. Classical work on take-all decline in wheat monocultures demonstrated that continuous cropping could select for soils in which *Gaeumannomyces graminis* var. *tritici* failed to establish despite repeated inoculation, and Mendes *et al.*, (2011) [10] later showed that this suppression in Dutch soils was attributable to a shift toward *Pseudomonas*-dominated, non-ribosomal peptide synthetase-rich communities rather than to a single antagonist. What has changed is the ambition. Rather than treating disease-suppressive soils as a phenomenon to be discovered and exploited where it already exists, researchers now ask whether such suppression can be engineered de novo, transplanted between soils, or built from defined microbial parts (Bakker *et al.*, 2018; Toju *et al.*, 2018) [2, 18].

This ambition rests on three loosely connected bodies of mechanistic work. The first concerns how plants recruit and shape their own microbiome through root exudation, a process sometimes called the cry for help response, in which pathogen attack itself alters exudate chemistry to favour protective taxa (Rudrappa *et al.*, 2008; Yuan *et al.*, 2018) [15, 23]. The second concerns systemic signalling, whereby colonisation of the root by non-pathogenic microbes primes jasmonate and ethylene-dependent defence pathways throughout

the plant, a phenomenon termed induced systemic resistance (Pieterse *et al.*, 2014) ^[13]. The third, more recent and more overtly engineering-oriented, concerns the deliberate construction of synthetic microbial communities, SynComs, assembled from culturable isolates with known functional traits and introduced as inoculants or seed treatments (Niu *et al.*, 2017; Carrión *et al.*, 2019) ^[11, 5].

Each of these bodies of work has produced compelling individual results, yet they do not obviously converge on a single engineering doctrine. Busby *et al.*, (2017) ^[4] noted, in a widely cited research-priorities paper, that the translation of microbiome science into deployable agricultural technology remained bottlenecked by an incomplete understanding of which microbial functions are context-dependent and which are portable across soils and cultivars. That bottleneck has not been resolved by the intervening years of sequencing-based descriptive work, and this review takes it as the central problem worth addressing rather than a caveat to be mentioned in passing. The sections that follow examine the mechanistic routes to microbiome-mediated disease suppression, the comparative and often conflicting evidence on synthetic versus native community approaches, and the structural gaps, methodological and regulatory, that continue to separate mechanistic insight from reliable field performance.

1. Root Exudation and the Limits of the Cry for Help Model

The idea that plants actively recruit protective microbes in response to attack has an intuitive appeal, and the experimental evidence for it in model systems is genuinely strong. Rudrappa *et al.*, (2008) ^[15] demonstrated that *Arabidopsis thaliana* roots infected with *Pseudomonas syringae* secreted elevated concentrations of malic acid, which selectively attracted the biocontrol strain *Bacillus subtilis* FB17 to the site of infection. Yuan *et al.*, (2018) ^[23] extended this logic to a full soil-borne legacy experiment, showing that foliar infection by *Botrytis cinerea* altered root exudate composition in a way that carried over to influence the microbiome of the following crop planted in the same soil. Taken together these studies support the claim that induced changes in root chemistry are not incidental but function as a signal that shapes microbial community composition in the rhizosphere.

Where the picture becomes less settled is in the question of generality. Most of the mechanistic detail on exuded recruitment signals comes from a small number of model

pathosystems, tomato and *Arabidopsis* against a handful of well-studied bacterial and fungal pathogens, and it is not clear how far the malic acid pathway, or analogous organic acid or flavonoid signals, generalise to cereal crops with fundamentally different root architecture and rhizodeposition profiles. Xu *et al.*, (2018) ^[22] working with sorghum found that drought stress reshaped the root microbiome toward monoderm, thick-walled bacterial taxa largely independent of any pathogen cue, which suggests that abiotic stress signalling and biotic recruitment signalling may compete for the same chemical channels rather than operating as cleanly separable systems. If exudate-mediated recruitment is contingent on which stress dominates at a given moment, engineering disease suppression through exudate manipulation alone becomes a considerably less tractable proposition than the *Arabidopsis* work implies.

A further complication concerns the directionality of causation in disease-suppressive soils identified after the fact rather than engineered from the outset. Santhanam *et al.*, (2015) ^[16] working with *Nicotiana attenuata* in its native habitat found that native soil bacteria, principally *Pseudomonas* and *Bacillus* isolates, could rescue plants from a sudden wilt syndrome associated with continuous monocropping, and that this protective effect depended on the specific consortium present in the native soil rather than on any single strain applied in isolation. This finding sits awkwardly against the reductionist logic of exudate signalling, because it implies that suppression is an emergent property of community composition built up over years of soil conditioning, not a property that can be summoned in a single growing season by manipulating one or two exuded compounds. Schlatter *et al.*, (2017) ^[17], reviewing disease-suppressive soils more broadly, reached a similar conclusion, arguing that suppression typically depends on cumulative shifts in microbial community structure and function that resist rapid re-engineering.

The practical implication is that root exudate manipulation, whether through breeding for altered exudate profiles or through exogenous application of specific recruitment signals, is best understood as one lever among several rather than a stand-alone engineering strategy. Its strongest evidence base remains in controlled, short-generation model systems, and its extension to perennial or long-season field crops has not yet been demonstrated with comparable mechanistic resolution.

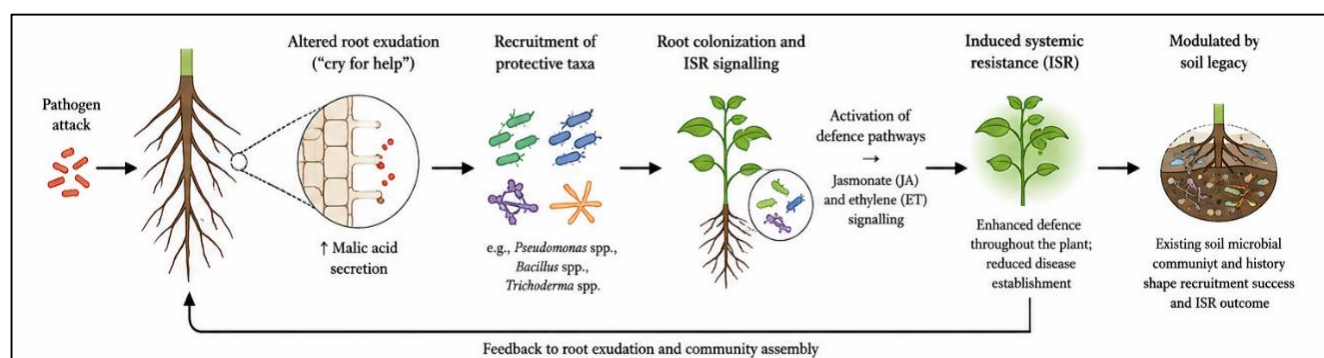


Fig 1: Proposed pathway linking pathogen-induced root exudate signalling to recruitment of protective rhizosphere taxa and downstream induced systemic resistance, integrating findings on malic acid mediated recruitment, jasmonate/ethylene priming, and soil legacy effects.

2. Induced Systemic Resistance as a Distal Mechanism

Where root exudation operates at the interface between plant and soil, induced systemic resistance, ISR, operates at a distance, priming defence responses in tissues the inducing microbe never physically contacts. Pieterse *et al.*, (2014) ^[13] synthesised several decades of work on this phenomenon and established that ISR elicited by *Pseudomonas* and *Bacillus* strains typically primes rather than directly activates jasmonate and ethylene signalling, meaning that primed plants mount a faster and stronger defence response upon actual pathogen challenge without incurring the fitness cost of constitutive defence activation. This priming logic is attractive from an engineering standpoint because it suggests that a single, well-characterised inoculant strain could, in principle, confer broad-spectrum protection against multiple unrelated pathogens without the narrow specificity problem that limits single-target biocontrol agents.

The empirical record complicates this optimism in two ways. First, the magnitude of ISR-conferred protection measured in controlled inoculation trials is frequently modest and variable across cultivars, a pattern documented across the plant growth-promoting rhizobacteria literature by Backer *et al.*, (2018) ^[1] in their review of mechanisms and commercialisation barriers. Variability of this kind is not simply noise, it appears to track host genotype, and this raises the uncomfortable possibility that ISR-based products developed and validated on one cultivar may underperform when deployed across the varietal diversity that characterises real cropping systems. Second, and more fundamentally, subsequent work by Haney *et al.*, (2015) ^[8] points to the existence of trade-offs between growth promotion and defence priming, since resources allocated to defence signalling are not simultaneously available for growth, so an inoculant optimised for disease suppression under high pathogen pressure may depress yield under low pressure conditions where the induced defence response was never needed.

Kwak *et al.*, (2018) ^[9] offer one of the more carefully documented field-relevant demonstrations of ISR-adjacent microbiome engineering, showing that resistance to bacterial wilt caused by *Ralstonia solanacearum* in tomato was associated with enrichment of *Bacillus*, *Streptomyces*, and other taxa capable of producing antimicrobial compounds in the rhizosphere of resistant lines, and that this enriched community could itself confer partial protection when transplanted to susceptible plants. The study is notable for combining amplicon sequencing with functional transplantation, a combination still rare enough in the literature that its results are sometimes cited as though they settle the broader question of whether resistance-associated microbiomes are transferable. They do not settle it. The transplantation was performed under controlled greenhouse conditions with sterilised recipient soil, conditions that remove precisely the resident microbial competition that field soils would present, and the authors themselves were explicit that field validation remained outstanding at the time of publication.

Taken together, the ISR literature supports a real and mechanistically well-characterised phenomenon, but one whose translation into a reliable engineering tool is constrained by genotype dependence, growth-defence trade-offs, and a persistent gap between sterilised transplantation experiments and the competitive, heterogeneous conditions of unmanaged field soil.

3. Synthetic Communities and the Diversity versus Targeting Debate

The most explicitly engineering-oriented strand of this literature dispenses with reliance on native soil legacy altogether and asks whether disease-suppressive function can be built from defined, culturable isolates assembled into a synthetic community. Niu *et al.*, (2017) ^[11] demonstrated proof of concept in maize, reducing a diverse native root microbiome down to a simplified community of seven bacterial strains capable of reconstituting much of the functional profile of the full community, including elements of pathogen exclusion through resource competition. The appeal of this approach is reproducibility, a defined synthetic community can in theory be manufactured, quality-controlled, and deployed with the same batch consistency expected of a chemical formulation, something native soil transplants cannot offer.

Carrión *et al.*, (2019) ^[5] pushed the synthetic community approach further by identifying a specific endophytic consortium associated with suppression of *Rhizoctonia solani* in sugar beet, and by showing that a chitinase-producing subset of that consortium, rather than the full community, was sufficient to reproduce much of the suppressive effect. This finding is important because it argues against a simple diversity-equals-function assumption that has been implicit in much of the microbiome literature, suppression here tracked a specific functional guild rather than overall community richness. Gu *et al.*, (2020) ^[7] reached a related conclusion from a different angle, showing that suppression of *Ralstonia solanacearum* by natural rhizosphere microbiomes was substantially explained by competition for iron between resident *Pseudomonas* populations and the pathogen, again pointing toward a targetable functional mechanism rather than an emergent property of diversity per se.

These functionally targeted findings sit in some tension with a separate body of work emphasising diversity and redundancy as the more robust engineering principle. Wei *et al.*, (2019) ^[21] found that the initial diversity and network complexity of the soil microbiome, rather than the presence of any specific antagonist, predicted whether tomato plants would later resist bacterial wilt, arguing that redundant, highly connected communities buffer against pathogen invasion in a way that a small, targeted synthetic community cannot replicate because it lacks the interaction network needed to resist invasion by opportunists once the introduced strains decline in abundance. This is a substantive disagreement, not a difference in emphasis. If diversity and network complexity are themselves protective, then the minimal, mechanistically transparent communities favoured by Niu *et al.*, (2017) ^[11] and Carrión *et al.*, (2019) ^[5] may be engineering the wrong variable, optimising for a demonstrable mechanism at the expense of the ecological resilience that comes from redundancy.

Reconciling these positions is not straightforward on current evidence. It is possible that both are correct under different conditions, that targeted, mechanistically defined synthetic communities perform well against a specific, well-characterised pathogen threat over a single season, while diverse, redundant native communities perform better against the shifting, poly-microbial threat environment of a working field over multiple seasons. Few studies, however, have been designed explicitly to test this contingency, most synthetic community trials report performance against a

single pathogen under a single set of conditions, and few diversity-focused studies report the specific mechanistic pathway responsible for the protection they document. Until studies are designed to test targeted function and community diversity against one another directly, rather than as separate

research programmes answering separate questions, the field will continue to accumulate results that are each individually persuasive and collectively difficult to integrate into a single engineering strategy.

Table 1: Comparative overview of proposed mechanisms and evidence for plant microbiome engineering in disease suppression

Engineering Route	Pathosystem	Finding	Source
Root exudate-mediated recruitment	<i>Arabidopsis/Pseudomonas syringae</i>	Malic acid secretion attracts <i>Bacillus subtilis</i> FB17 to infection site	Rudrappa <i>et al.</i> , (2008) ^[15]
Soil-borne exudate legacy	<i>Botrytis cinerea</i> /multiple crops	Foliar infection alters root exudates, affecting the next crop's microbiome	Yuan <i>et al.</i> , (2018) ^[23]
Native disease-suppressive soil	Wheat/ <i>Gaeumannomyces graminis</i>	<i>Pseudomonas</i> -dominated, NRPS-rich community suppresses take-all	Mendes <i>et al.</i> , (2011) ^[10]
Native consortium rescue	<i>Nicotiana attenuata</i> /sudden wilt	Native <i>Pseudomonas</i> and <i>Bacillus</i> consortium rescues plants from wilt	Santhanam <i>et al.</i> , (2015) ^[16]
Induced systemic resistance	Multiple crops, broad-spectrum	Jasmonate/ethylene priming by root-associated bacteria	Pieterse <i>et al.</i> , (2014) ^[13]
ISR-associated transplant	Tomato/ <i>Ralstonia solanacearum</i>	<i>Bacillus</i> / <i>Streptomyces</i> -enriched microbiome transferable under sterile conditions	Kwak <i>et al.</i> , (2018) ^[9]
Simplified synthetic community	Maize root microbiome	Seven-strain community reconstitutes much of native functional profile	Niu <i>et al.</i> , (2017) ^[11]
Functionally targeted community	Sugar beet/ <i>Rhizoctonia solani</i>	Chitinase-producing subset sufficient for suppression	Carrión <i>et al.</i> , (2019) ^[5]
Nutrient competition mechanism	Tomato/ <i>Ralstonia solanacearum</i>	Iron competition by resident <i>Pseudomonas</i> explains suppression	Gu <i>et al.</i> , (2020) ^[7]
Diversity/network complexity	Tomato/bacterial wilt	Initial microbiome diversity, not a single antagonist, predicts resistance	Wei <i>et al.</i> , (2019) ^[21]
Abiotic stress interaction	Sorghum, drought	Drought reshapes root microbiome toward monoderm taxa independent of pathogen cues	Xu <i>et al.</i> , (2018) ^[22]
Colonisation/persistence gap	Multiple PGPR products	Greenhouse efficacy frequently fails to reproduce at field scale	Backer <i>et al.</i> , (2018) ^[1]
Sterilised substrate bias	Rhizosphere ecology, general	Gamma-irradiated soil inflates apparent colonisation and efficacy	Philippot <i>et al.</i> , (2013) ^[12]
Regulatory bottleneck	Multi-strain biopesticides, general	Per-strain approval requirements limit commercialisation of synthetic communities	Backer <i>et al.</i> , (2018) ^[1]

4. Comparative Evidence and Context Dependency across Soil Types and Scales

A recurring and largely unresolved theme across the studies discussed so far is that soil type functions less as a background variable and more as an active determinant of whether a given microbiome engineering strategy succeeds. Raaijmakers and Mazzola (2016) ^[14], commenting on the broader concept of soil immune responses, argued that soils vary enormously in their baseline capacity to suppress pathogen establishment, independent of any deliberate intervention, and that this baseline capacity is itself shaped by long-term factors such as organic matter content, mineral composition, and cropping history that are not easily replicated in a pot trial. This matters directly for engineering studies, because a synthetic community or exudate manipulation validated in a single potting mix, as most greenhouse studies are, has been tested against only one point on a very wide axis of soil suppressive potential, and there is no strong basis for assuming the result will hold at other points on that axis.

Philippot *et al.*, (2013) ^[12], in an influential synthesis of rhizosphere microbial ecology, made a related methodological point, that much of the mechanistic literature on rhizosphere function derives from studies using sterilised or gamma-irradiated soil as a growth substrate, a practice that removes the very microbial competition an introduced strain or community would need to overcome under field conditions. Sterilisation is a reasonable experimental control for isolating the effect of an introduced

organism, but it systematically inflates apparent colonisation success and functional persistence relative to what would be observed in live, competitive field soil. Several of the studies discussed above, including elements of the Kwak *et al.*, (2018) ^[9] transplantation work and portions of the Niu *et al.*, (2017) ^[11] reconstitution experiments, rely at least partly on this kind of simplified substrate, which is worth bearing in mind when comparing their reported efficacy against studies conducted in unamended field soil.

Where direct field comparisons do exist, the results are frankly mixed. Backer *et al.*, (2018) ^[1] noted, in surveying the commercialisation pipeline for such products, that a substantial fraction of inoculants showing strong greenhouse efficacy fail to reproduce that efficacy at field scale, and attributed this gap primarily to poor rhizosphere colonisation and persistence once the inoculant is competing against an established resident community rather than to any failure of the underlying suppressive mechanism itself. This is a distinction worth taking seriously. It reframes microbiome engineering not primarily as a problem of discovering the right suppressive mechanism, since several credible mechanisms already exist, but as a problem of ecological invasion, getting an introduced organism or consortium to establish, persist, and remain functionally active in a resident community that has its own competitive dynamics and is not a blank space waiting to be filled.

Wei *et al.*, (2019) ^[21] and Gu *et al.*, (2020) ^[7] both offer partial support for this invasion-ecology framing, since both studies locate the mechanism of native suppression in the

resident community's own competitive behaviour toward the pathogen, rather than in any trait unique to an introduced biocontrol agent. If resident community competition is doing much of the protective work in naturally suppressive soils, then engineering strategies premised on introducing an

outside organism may be working against the same competitive forces that make the target soil suppressive in the first place, a structural tension that has received comparatively little explicit attention.

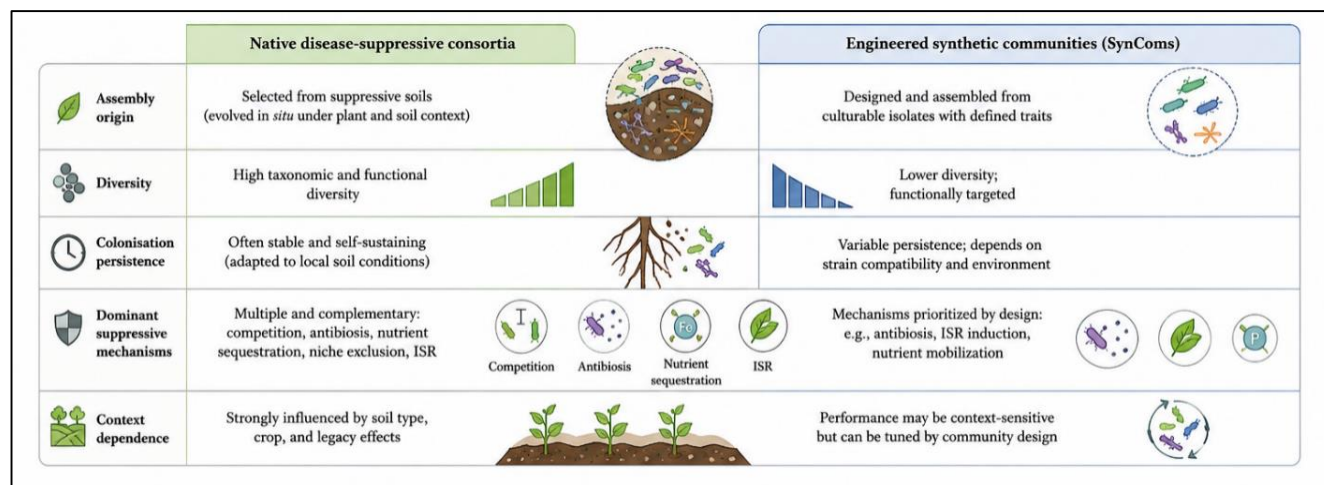


Fig 2: Comparative schematic of native disease-suppressive consortia versus engineered synthetic communities.

5. Current Gaps and Limitations

Three gaps stand out as limiting factors on the reliability of plant microbiome engineering as a disease management strategy, and none of them will be closed by further descriptive sequencing of microbial communities. The first is the absence of standardised metrics for colonisation persistence. Studies report inoculant success using inconsistent proxies, colony-forming unit counts at a single time point, relative abundance from amplicon sequencing, or simply visible disease reduction, and these proxies are not interchangeable. A strain can show strong initial colonisation and then decline to functional irrelevance within weeks, a trajectory that a single end-point measurement will not detect. Compant *et al.*, (2019) [6], reviewing plant microbiome applications broadly, called for longitudinal monitoring standards as a precondition for meaningful cross-study comparison, a call that subsequent synthetic community and rhizobacteria literature has only inconsistently followed.

The second gap concerns regulatory and production pathways that were built around single-strain biopesticides and are poorly matched to multi-strain synthetic communities. Backer *et al.*, (2018) [1] documented the practical difficulty of securing regulatory approval and consistent manufacturing quality control for products containing more than one active microbial component, since safety and efficacy data are typically required per strain, which multiplies the cost and time of bringing a genuinely synthetic, multi-organism community to market relative to a single well-characterised biocontrol strain. This creates a perverse incentive structure in which the scientifically most

defensible engineering strategy, diverse or functionally redundant consortia, is also the commercially least tractable one, while single-strain products, whose ecological fragility is well documented, remain easier to register and sell.

The third gap is conceptual rather than logistical, and concerns the difficulty of attributing observed protection to a specific, generalisable mechanism rather than to an idiosyncratic feature of the particular pathosystem studied. Trivedi *et al.*, (2020) [19], surveying the field at the level of community assembly theory, observed that most mechanistic claims in the literature are drawn from a narrow set of model pathogens and model crops, and cautioned against extrapolating mechanisms established in *Arabidopsis-Pseudomonas* systems to the polygenic, multi-pathogen pressure typical of field cereals or perennial tree crops. This review's own reading of the comparative evidence in the preceding sections supports that caution, the recruitment, induced resistance, and synthetic community literatures each rest on a handful of intensively studied systems whose generality remains an assumption rather than a demonstrated fact.

Addressing these gaps does not require abandoning the mechanistic ambitions of the field. It requires accepting that the next phase of useful work is less about identifying additional suppressive taxa or defence-priming strains, of which a considerable inventory already exists, and more about the harder, less publishable work of establishing under what soil, cultivar, and pathogen-pressure conditions each known mechanism actually holds, and being willing to report the conditions under which it does not.

Table 2: Structural and functional attributes distinguishing native disease-suppressive consortia from engineered synthetic communities

Attribute	Native	Engineered Synthetic Communities	References
Origin	Selected over multiple cropping cycles	Assembled from culturable isolates	Toju <i>et al.</i> , (2018) ^[18]
Reproducibility	Low; soil-and site-specific	High; defined composition	Niu <i>et al.</i> , (2017) ^[11]
Mechanistic transparency	Often unresolved, multifactorial	Generally well characterised per strain	Carrión <i>et al.</i> , (2019) ^[5]
Community diversity	High, functionally redundant	Deliberately minimised	Wei <i>et al.</i> , (2019) ^[21]
Colonisation persistence	Established, self-sustaining	Frequently declines post-introduction	Backer <i>et al.</i> , (2018) ^[1]
Regulatory pathway	Not applicable; a soil management practice	Complex; per-strain approval typically required	Backer <i>et al.</i> , (2018) ^[1]
Field validation	Extensive, historical	Largely greenhouse or sterilised substrate	Philippot <i>et al.</i> , (2013) ^[12]
Resilience to opportunist invasion	High, attributed to network buffering	Uncertain; declines as introduced strains fade	Wei <i>et al.</i> , (2019) ^[21]
Primary suppressive mechanism	Resource competition, induced suppression	Targeted trait, e.g. chitinase or siderophore production	Gu <i>et al.</i> , (2020) ^[7] ; Carrión <i>et al.</i> , (2019) ^[5]
Response to abiotic stress	Buffered by community diversity	Poorly characterised	Xu <i>et al.</i> , (2018) ^[22]
Approximate cost of establishment	Low; achieved via crop rotation and residue management	Higher; requires fermentation, formulation, and quality control	Backer <i>et al.</i> , (2018) ^[1]
Time to establish suppression	Years of cumulative conditioning	Claimed within a single season	Santhanam <i>et al.</i> , (2015) ^[16] ; Niu <i>et al.</i> , (2017) ^[11]

Conclusion

Plant microbiome engineering has moved a considerable distance from the observational disease-suppressive soil studies of previous decades toward a genuinely mechanistic and, in places, deliberately constructive science. Root exudate signalling, induced systemic resistance, and synthetic community assembly each rest on defensible experimental foundations, and each has produced results that would have seemed implausible to plant pathologists working purely with chemical and genetic resistance tools a generation ago. What the field has not yet produced is a reliable account of when these mechanisms transfer beyond the soils, cultivars, and pathosystems in which they were first characterised, and the comparative evidence reviewed here suggests that this failure of transfer is not incidental but structural, rooted in the mismatch between controlled experimental substrates and the competitive, historically conditioned communities of working agricultural soils. The most productive path forward is unlikely to come from further proof-of-concept demonstrations in sterilised potting mix. It will come from field-scale, multi-season trials that report colonisation persistence alongside disease outcomes, from head-to-head comparisons of targeted functional consortia against diverse redundant communities rather than each being validated in isolation, and from a research culture willing to publish and interpret context-dependent failure with the same rigour currently reserved for success. Sustainable disease management through microbiome engineering remains a credible long-term proposition, but its credibility will ultimately rest on evidence generated under the conditions farmers actually work with, not on the elegance of the mechanisms discovered in the greenhouse.

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