Suppressive Composts: Microbial Ecology Links Between Abiotic Environments and Healthy Plants

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Abstract

Suppressive compost provides an environment in which plant disease development is reduced, even in the presence of a pathogen and a susceptible host. Despite the numerous positive reports, its practical application is still limited. The main reason for this is the lack of reliable prediction and quality control tools for evaluation of the level and specificity of the suppression effect. Plant disease suppression is the direct result of the activity of consortia of antagonistic microorganisms that naturally recolonize the compost during the cooling phase of the process. Thus, it is imperative to increase the level of understanding of compost microbial ecology and population dynamics. This may lead to the development of an ecological theory for complex ecosystems as well as favor the establishment of hypothesis-driven studies.

INTRODUCTION

Sustainable and environmentally acceptable farming relies to a great extent on the ability to reduce the need for hazardous agrochemicals while maintaining plant health and productivity. Thus, agricultural practices and technologies based on ecological principles and sustainability considerations are being adopted and improved. In the case of soilborne plant pathogens, cultivation and biological practices include cover crops incorporated as green manures, organic amendments, crop rotation, minimal tillage practices, soil solarization, the application of single biocontrol agents, such as *Trichoderma* or combination of means (1, 4, 9, 37, 41, 57), and the topic discussed here: suppressive composts.

A suppressive compost or soil provides an environment in which plant disease development is reduced, even when the pathogen is favored by the presence of a susceptible host. Following the classical definition of Cook & Baker (25), suppressive soils are "soils in which the pathogen does not establish or persist, establishes but causes little or no damage, or establishes and causes disease for a while but thereafter the disease is less important, although the pathogen may persist in the soil" (25). Plant disease suppression is considered to be a direct result of the activities of microorganisms which naturally recolonize compost during the cooling phase (46). In the current review, we aim to assemble the current understanding of what makes compost suppressive.

THE PHENOMENON OF SUPPRESSION

Since the first publications describing suppressive composts (44, 45), a remarkable number of examples involving a wide array of pathosystems and composts, produced from a broad variety of raw materials and using different technologies, have been reported. Indisputably, suppression of soilborne plant pathogens by compost is a widespread and ubiquitous phenomenon. However, its practical application is still limited.

Several authors have recently surveyed the literature regarding the prevalence of disease suppression conferred by organic amendments and composts. Bonanomi et al. (14) analyzed publications on the application of organic amendments, focusing on the suppressive capacity of different organic materials and the responses of different soilborne pathogens. A total of 250 articles were analyzed, with 2,423 experimental case studies. Organic matter, such as crop residues or compost amendments, was found to be suppressive in 45% and nonsignificant in 35% of the cases. In 20% of the cases, a significant increase in disease incidence was observed. Compost was the most suppressive material, with more than 50% of cases showing effective disease control.

Based on an even larger and more recent data set, Noble (86) analyzed the risks and benefits of soil amendment with composts in relation to plant pathogens and concluded that the result of applying compost to soils is typically positive when suppression of diseases caused by soilborne pathogens is being investigated, and there is little risk of promoting or introducing diseases via compost. Noble & Coventry (87) also reviewed a wide variety of positive cases of disease suppression by composts; they concluded that the compost's effect is relatively smaller and more variable when it is applied in the field, as compared with results obtained in container media.

Other reports have focused on the characteristics that might determine, and thus predict, the suppressive capacity of composts by comparing a number of composts under identical conditions. In one study, 36 compost samples produced from diverse feedstocks and composting technologies at commercial composting facilities were analyzed for a number of physical, chemical, and biological properties, including disease suppression (98). Termorshuizen et al. (108) provided a thorough demonstration of the variability of the suppressive effect by comparing the effectiveness of 18 different composts on seven pathosystems. Significant

disease suppression was found in 54% of the cases, whereas only 3% of the cases showed significant disease enhancement. The authors emphasized that no single compost showed significant disease suppression against all pathogens and that the pathogens were not affected similarly by all composts. Noble (86) also stressed that a limiting factor for commercialization of suppressive compost is a lack of predictability and consistency against various pathogens. Ntougias et al. (88) studied nine composts derived from wastes generated during olive oil, wine, and mushroom production. They emphasized the need for individual evaluation of compost products for specific uses and the development of standardized compost production and storage protocols.

Reports providing insight into the function and structure of the microbial populations acting in compost-mediated disease suppression are on the rise. It is anticipated that discoveries focusing on microbial ecology in microenvironments generated by compost amendments will further contribute to the identification of the sources of variability and provide means for quality control of commercial products. However, a lack of ecological theory to guide research in microbial ecology in complex environments has been recently emphasized (94). This gap is still present today, limiting hypothesis-driven research and interpretation of metadata, especially regarding compost and compost-amended environments.

The remarkably widespread occurrence of compost-conveyed suppression, compared with the rather rarely and locally observed suppressive soils, leads to a key question: Is there a link between specific conditions and/or traits that are common in composts and compost-amended environments and the proliferation of microbial communities/agents that lead to plant disease suppression? And why do composts appear highly disease-specific?

To this end, we can indeed identify common traits that have been regarded as potential indicators of suppression during these three decades of worldwide research. Much progress has been made in understanding specific mechanisms of disease suppression, verifying the operation of these mechanisms in situ and also identifying and isolating specific microbial agents accountable for disease suppression. In addition, highly active suppressive composts can be produced by inoculation with well-characterized biological control agents. Studies that have attempted to decipher the key players and the molecular network operating during plant growth on compost media substantially contribute to our understanding of the occurrence and persistence of compost-derived disease suppression. We present the current knowledge regarding the different mechanisms governing compost-mediated disease suppression and recapitulate the extent to which composts' common traits are responsible for this suppression.

COMMON TRAITS OF SUPPRESSIVE COMPOSTS

The Life Cycle of Composts

Composts are the product of oxidative aerobic microbial decomposition of organic matter under controlled conditions. A compost pile has a life cycle in the sense that it goes through several rather distinct decomposition phases: (*a*) an initial microbial adaptation phase (usually short but occasionally longer, depending mainly on labile-carbon (C) availability and carbon:nitrogen (C:N) ratios of the raw materials); (*b*) a thermophilic phase (which may be recurring if highly available C substrates are released following compost pile turnings); (*c*) a stabilization stage (during which further turning does not cause any rise in temperature); (*d*) a cooling/maturation phase (during which metabolism of complex C compounds and humification processes occur and phytotoxic effects are reduced); (*e*) a maturity phase (the useful life stage of composts during which metabolism of complex C compounds and humification processes are still occurring but at diminishing rates); and (*f*) an overmaturity phase (when metabolic processes have practically ceased and the established microbial community is no longer active). Approximately

50% of the C contained in the raw materials is generally mineralized to CO_2 up to the stabilization phase. The characteristic succession of microbial communities occurring during the compost's life cycle is directly related to establishment of the suppression phenomenon. Although the microbial community surviving the thermophilic phase of composting is not sufficient to support disease suppression, an active microflora adapted to the available substrates following compost maturation or stabilization is essential (28, 73, 82, 98, 119). At this stage, readily available organic substrates have already been oxidized by a zymogenous, copiotrophic, and thermotolerant microbial community. The remaining slowly biodegradable, but still available, semihumified materials, lignins, recalcitrant microbial metabolites, and low amounts of partly decomposed celluloses and hemicelluloses appear to favor the rise of a competitive microbial community that is well established and antagonistic in terms of specialized substrate utilization. Mesophilic gram-negative bacteria, actinomycetes, and fungi often dominate at these stages (21, 63, 109). The capacity for disease suppression in overmatured or long-stored composts and potting media often deteriorates (13, 88), indicating an important role in compost suppressiveness for the microbial community adapted to the C substrates available during the maturity phase. Because suppression is related to the stage of the composting process, it is important to define critical phases using relatively easy measurement methodologies. Indeed, a variety of methods have been examined to estimate compost stability and maturity. A low concentration of dissolved (water-extractable) organic carbon in compost extract is a useful indicator of the final stage of the composting process (124, 125). However, the literature shows some disagreement as to the best combination of protocols to evaluate compost stability and maturity (116).

The conditions that favor natural recolonization in mature, stabilized composts also favor the increase in populations of specific microbial agents, which suppress plant diseases and have repeatedly been isolated from compost and compost-treated environments. Some of these organisms have been extensively studied and a diverse range of modes of action have been described, as illustrated below. A number of such biological control agents, originally isolated from composts, have been used to inoculate composts in order to enhance their suppressive capability, including *Trichoderma hamatum* and *Flavobacterium balustinum* (65), *Verticillium biguttatum*, a mycoparasite of *Rhizoctonia solani* (93), cyanobacterial/bacterial cultures (33), *Bacillus subtilis* (80, 101), and others. In many cases, the addition of a specific biological control agent to compost can lead to a substrate with a broader-range suppressive effect (65, 95, 101). It has been suggested that the level of reproducibility and consistency of disease control can also be improved using fortified composts (47, 50). Again, the time of inoculation, i.e., after peak heating but before substantial recolonization with mesophilic microorganisms (47), and establishment of the introduced biocontrol agent at high densities in the compost (2, 65, 80, 101) are essential for the successful production of a fortified compost.

Raw Materials, C Substrates, and Suppression

The links between specific C substrates that become available during composting and disease suppression remain elusive. A notable exception is the clear causative link between the abundance of chitins and chitin-derived C compounds in certain composts and the potential proliferation of chitinolytic microbial agents (19, 59). In this case, there is enough evidence to suggest that a link between the proliferation of chitin-degrading microorganisms and degradation of fungal pathogen cell walls is operational, leading to compost suppression of fungal soilborne disease pathogens. Attempts to pin down links to other specific C substrates or to degradative enzymes such as β-glucosidase (15, 16) do not appear to show general applicability. However, a correlation of actinobacteria and fungal populations with β-glucosidase and esterase activities in composts has been

observed (109). In contrast, labile C substrates, such as sugars (21), and high availability of celluloses (23, 24) arrest the suppressive activity of composts or the disease control capability of certain microbial agents (29, 81, 106, 111).

Microbial Activity

Microbial activity in composts plays a major role in the suppression of soilborne plant pathogens (45, 99). Two main types of microbially mediated disease suppression have been shown at the population level (51, 88): (*a*) disease suppression induced by a large metabolically active microbial community, identified as a general suppression effect and (*b*) suppression attributed to specific microbial agents that proliferate upon, or are favored by, compost application. These two general modes of disease suppression may coexist.

The microbial activity estimator commonly used to address whole microbial communities is fluorescein diacetate (FDA) hydrolysis. A correlation between FDA hydrolysis and disease suppression has been repeatedly reported with oomycete plant pathogens, such as *Phytophthora* and *Pythium* (13, 21, 26, 49, 55, 121). However, correlations over a wide range of composts deriving from different materials are not generally observed (88), perhaps because it is hard to obtain normalized mean FDA hydrolysis values for a comparison of different composts.

Microbial Consortia

Understanding compost microbiology is a necessary first step in relating suppression and population dynamics. However, the need to investigate population dynamics of microbial consortia, rather than single microbial species, severely limits the applicability of classical ecological models and tracing methodologies. Most of the past research on compost microbiology has been performed using cultivation-based techniques. For composts, the percentage of cultivable microbiota is unknown (56). Molecular tools offer alternative and complementary methods of monitoring microbial populations. Molecular fingerprinting techniques usually target information-bearing macromolecules such as DNA, RNA, or lipids (79). Profiling of the microbial community during the composting process has been performed using polymerase chain reaction (PCR)-based molecular methods, such as denaturing gradient gel electrophoresis (DGGE), clone library, and microarray. Distinctive community shifts during curing and the dominant species prevailing during the different curing stages have been identified, with γ-Proteobacteria and Bacteroidetes being the most abundant phyla in all cases (27). Different members of the nitrifying bacteria and cellulose- and macromolecule-degrading bacteria have been found throughout the compost-curing process. In contrast, bacterial pathogens were not detected, even after a year of curing. During the mid-curing stage, actinobacteria were dominant (96, 105). The bacterial diversity in a compost derived from marine animals was also studied by DGGE and dominance of Bacillaceae was reported (85). Recently, the relative abundance of the same bacterial taxa (i.e., γ- and β-Proteobacteria, Firmicutes, and Actinobacteria) was reported to be an important indicator of disease suppression exhibited by suppressive versus conducive soils, using a Phylochip-based metagenomics approach that incorporates a high-density 16S rDNA microarray analysis (30, 78). Unfortunately, these studies have almost exclusively focused on bacterial consortia, and there is very little knowledge of the fungal populations prevailing in suppressive composts, despite the isolation of very successful and effective fungal biocontrol agents from compost-amended media. Recently, the presence of many Basidomycetes in the compost environment has been described (7, 8). To the best of our knowledge, viruses have never been considered, but the following example may illustrate their potential: A five-strain bacteriophage mixture isolated from sewage effluent was applied to dairy manure compost inoculated with *Salmonella enterica*. The bacteriophage treatment resulted in a greater than

2-log reduction in *Salmonella* within 4 h at all moisture levels compared with controls (43).

Compost surface/niche microbiology, as opposed to bulk compost microbiology, has also been largely underinvestigated. We know little about surface/interface microbiology, or biofilm formation or colonization, e.g., of a piece of straw, a leaf vein, a rice husk or grape skins, or of seed decomposing and aging in a compost pile over time. Events occurring in the rhizosphere/rhizoplane and on seed surfaces of plants grown in the presence of compost should also be considered, as these are the sites of interplay between the compost microbial community, the pathogen and the plant (79). These surfaces harbor rather simplified microbial communities, and therefore molecular data on microbial community structures may be directly interpretable (78).

MECHANISMS OF PLANT DISEASE SUPPRESSION

Regarding disease-control targets, five possible mechanisms, attributed to either biotic or abiotic characteristics of the composts, have been identified: (*a*) successful competition for C and nutrients (such as Fe) by beneficial microorganisms; (*b*) production of antibiotics or other compounds that are toxic to pathogens; (*c*) successful predation/parasitism of pathogens by lytic bacteria and fungi; (*d*) activation of disease-resistance genes in plants by the compost microflora, and (*e*) improved plant nutrition and vigor, leading to enhanced disease resistance (48, 49). These mechanisms may exist separately or in combination. The first three mechanisms target the pathogen directly and reduce its survival and capacity to invade the plant, whereas the latter two act indirectly via the plant and affect disease progression in the host plant. Competition and production of antibiotics are mostly involved in general suppression effects, whereas predation, parasitism, and activation of disease resistance are more often manifested by specific microbial agents. Both diverse microbial consortia, such as plant growth–promoting rhizobacteria (PGPR), and specific microbial agents (usually showing endophytic growth) may lead to improved plant nutrition and vigor.

Much of the research regarding mechanisms underlying the antagonistic relations between plant pathogens and soil microorganisms has been conducted with specific biological control agents, and these mechanisms have been extensively reviewed (4, 41, 69, 75, 102). In the case of suppressive compost, attention should be paid to the role of the microbial community and consortia of microorganisms. Each mechanism of suppression might be independently responsible for suppression of a specific pathosystem, but several mechanisms may function simultaneously in suppression of another disease. Nevertheless, most studies have explored each mechanism separately. Here, we describe and discuss representative examples.

Competition

Disease suppression based on competition could be related to microbial metabolic activities and is controlled by the availability and rate of utilization of nutrients and energy sources (20, 21). Chen et al. (21) found that samples taken from the low-temperature edge of compost piles were suppressive to cucumber damping-off caused by *Pythium ultimum*, whereas material removed from the higher-temperature center or core was conducive to disease. The microorganism populations in the low-temperature area were taking up nutrients and creating a nutrient sink. This nutrient sink was the principal mechanism of suppression, shown through destruction of the suppressive effect by addition of nutrients to the compost, and the presence of higher nutrient concentrations in the disease-conducive media (21).

Competition for C source was suggested as a mechanism of suppression of *Pythium aphanidermatum*. Oospores survived in disease-suppressive compost for over six months but could not germinate because of competition by the microbial populations. Consequently, disease did not develop (40, 74). In that study, a glucose and asparagine mixture was

incorporated into suppressive cattle manure compost and conducive peat. In the compost, the glucose was depleted within 12 h, concomitant with a rapid increase in respiration rate, whereas in the peat, most of the glucose remained after 24 h. Several consecutive amendments of the glucose/asparagine mixture to the compost negated the suppression phenomenon.

Nelson (84) suggested that interactions between the pathogen and competitors occur in the spermophere, with competition being for specific seed exudates. McKellar & Nelson (77) demonstrated that suppressive leaf composts contain microbial consortia that metabolize fatty acids produced by cotton seeds. These fatty acids stimulated germination of *Pythium ultimum* sporangia, and their metabolism corresponded with control of damping off. Furthermore, populations of fatty acid–metabolizing bacteria and actinobacteria were higher in microbial communities originated from suppressive as compared with conducive compost. The authors suggested that communities of compost-inhabiting microorganisms, colonizing cotton seeds within the first few hours after sowing in a *Pythium*-suppressive compost, play a major role in the suppression of *P. ultimum* sporangium germination, seed colonization, and damping off. Formation of the suppressive community on seeds, in the presence of compost, did not require the presence of the pathogen and was very rapid, taking place within 8 h from sowing (22, 77, 113). Results presented by Ofek et al. (89) supported the above hypothesis: In the presence or absence of *Pythium*, highly dense bacterial populations deriving from suppressive compost colonized the germinating seed coat and radicle. The bacterial communities of seeds germinating in the compost-amended medium were highly similar in composition, size, spatial distribution, and structure. This may further support a nonspecific, *Pythium*-independent mechanism of suppression.

Fusarium oxysporum, which is highly susceptible to competition for nutrients, was controlled by soil amendment with composts (4). Addition of compost to a conducive soil rendered the soil suppressive to *Fusarium* wilt of flax caused by *F. oxysporum* f. sp. *lini*. Microbial antagonism was responsible for the suppression based on nutrient competition, involving the total microflora of the soil and compost (100). Yogev et al. (119) characterized the suppressive ability of three plant residue–based composts toward the following formae speciales of *F. oxysporum*: *melonis*, *basilici*, *radicis-lycopersici*, and *radicis-cucumerinum*. Disease development in melon, tomato, and cucumber seedlings growing in the three composts was significantly less than that observed in peat. The tested formae speciales exhibited different rates of decline of the viable conidia incorporated into the composts, compared with the rate in the peat control, suggesting that different mechanisms, including competition, might be involved in the suppression of the different pathogens. Indeed, Yogev et al. (120) demonstrated that induced resistance could also be involved in *Fusarium* disease suppression by composted manure and tomato residues. A nonpathogenic *F. oxysporum* strain, designated F2, isolated from a suppressive compost, was reported to reduce *Verticillium* wilt in eggplant under greenhouse and field conditions (79). F2 was shown to colonize the root surface along the intercellular junctions, excluding *Verticillium dahliae* from that ecological niche. In parallel, quantitative PCR analysis showed that application of F2 reduces the levels of *V. dahliae* vascular colonization along with disease severity. In this case, parasitism, antibiosis, and induced resistance were ruled out as suppressive mechanisms. Therefore, it was suggested that competition for space or nutrients on the root surface is the main mechanism of action of F2 against *V. dahliae* (90).

Parasitism

Mycoparasitism is a mechanism based on another organism feeding on a fungus. The parasitic activity of various microorganisms toward plant pathogens involves recognition of the pathogen by the antagonist and excretion of several cell wall–degrading enzymes to enable

the parasite to penetrate the hyphae of its host, the plant pathogen. This type of antagonism, which causes the death of the target organism, results in reduction of pathogen inoculum density. The occurrence of mycoparasites has been reported for several suppressive composts.

El-Masry et al. (34) showed that compost water extract (CWE) from several suppressive composts produced clear inhibition zones against all fungi tested. The microflora found in the CWE had an important role in suppressing the growth of *Pythium debaryanum*, *F. oxysporum* f. sp. *lycopersici*, and *Sclerotium bataticola*. The CWE did not contain antibiotics or siderophores. The presence of protease, chitinase, lipase, and β-1,3-glucanase (cell wall–degrading enzymes) in the CWE indicated a possible role for mycoparasitism.

Nelson et al. (83) identified specific strains of *Trichoderma* sp. and *Gliocladium virens* as the most effective fungal hyperparasites of *R. solani* present in tree bark compost. A few of the 230 other fungal species present also showed activity, but most were ineffective. Kwok et al. (65) described a synergistic interaction between *T. hamatum* and *Flavobacterium balastinum*. In another study (28), mature biosolids compost was found to be suppressive toward germination of sclerotia of *Sclerotium rolfsii*. However, during prolonged curing, suppression of sclerotial germination was reduced by more than 50%. When plant assays were conducted, suppression of disease development was also reduced by more than 60%. Correlations were found between the decrease and subsequent loss of suppression of sclerotial germination and the decrease in pH, dissolved organic carbon, and NH_4^+ concentrations and increase in NO_3^- concentration in the compost. It was concluded that parasitism of sclerotia by antagonistic fungi occurred only when the sclerotia were weakened by the presence of NH_3 at the higher pH. This study represents a case in which direct parasitism is the mechanism of suppression but it occurs only under the proper abiotic conditions (125). Profiling of the *Ascomycetes* populations of composts and attacked sclerotia revealed that the sclerotial environment can serve as bait for compost mycoparasitic populations. Novel mycoparasites *Thielavia* and *Petriella* were identified and isolated along with known mycoparasites, such as *Chaetomium*, *Geomyces*, *Penicillium*, and *Trichoderma*. However, a single species that could account for all of the naturally attacked sclerotia was not identified; rather, a variety of antagonists were revealed. Two types of mycoparasitic behavior were described for the antagonists examined in this work. *Trichoderma* acted as a primary mycoparasite, colonizing sclerotia of *S. rolfsii* regardless of external conditions. The *Penicillium* and *Petriella* isolates acted as weak, opportunistic antagonists, better able to colonize sclerotia when the external conditions were unfavorable for the latter's germination. Thus, compost extracts that inhibited germination of sclerotia also increased their susceptibility to attack by these mycoparasites.

Increased susceptibility of sclerotia of *S. rolfsii* to colonization by *Trichoderma* spp. has been reported as a consequence of treatment with sublethal concentrations of metham sodium (42). Weakening of sclerotia with sublethal heating has been reported to increase their microbial colonization and frequency of surface cracks (68). Hoynes et al. (53) increased the efficiency of *G. virens* biocontrol of *S. rolfsii* germination by a combined treatment with ammonium salts and urea.

Another interesting example (112) demonstrated that incorporation of kraft lignin into soil reduces the viability of *R. solani* sclerotia. They hypothesized that lignin-degrading basidiomycetes enriched with this amendment might play an important role in the control of *R. solani* sclerotia by degrading the melanin in the sclerotial cell walls, making the sclerotia more susceptible to antagonists such as *Trichoderma*, actinomycetes, and gram-negative bacteria. The presence of humic substance-degrading basidiomycetes in biosolids compost was shown by Grinhut et al. (38). Until recently, basidiomycetes were considered less

common in habitats such as agricultural soils. However, soil DNA amplification has revealed much greater diversity than was anticipated in this habitat on the basis of culture-based methods or surveys of fruiting bodies (71). Moreover, additional studies found the presence of many basidomycetes in the compost environment (7, 8). It could be postulated that these white-rot fungi might play an important role in humic substance turnover in nature as well as during composting, probably in the last stage of the process when the temperature decreases in the upper and cooler area of the compost heap, and may partially contribute to suppression.

Antibiosis

Antibiosis—the production of antimicrobial compounds by antagonistic microorganisms—has also been suggested as a suppression mechanism. The conclusion that antibiotics are involved in suppression is based mainly on the ability of microbes isolated from suppressive compost to produce antibiotics in vitro as described in the following example: A *Trichoderma harzianum* strain, isolated from composted hardwood bark in Western Australia, was found to produce a metabolite with antifungal and plant growth–promoting activity. The structure and absolute configuration of the fungal compound, harzianic acid, were determined by X-ray diffraction studies. Harzianic acid showed antibiotic activity against *Pythium irregulare*, *Sclerotinia sclerotiorum*, and *R. solani* (115). Bradley & Punja (17) evaluated the potential of three different composts added to a rock-wool medium to reduce the development of root and stem rot under greenhouse conditions. In vitro antagonism assays between compost-isolated bacterial strains and *Fusarium* showed that strains of *Pseudomonas aeruginosa* exhibit the greatest antagonism. These bacteria were able to produce the antibiotic 2,4-diacetylphloroglucinol. They concluded that composts containing antibiotic-producing *P. aeruginosa* have the potential to suppress diseases caused by *Fusarium* species.

Induced Resistance

Activation of the plant's immune system and induction of local and systemic resistance have also emerged as mechanisms of suppression of diseases conferred by both foliar and soilborne pathogens (3, 52, 58, 64, 122, 123). It was believed that induction of systemic plant resistance by composts was a rather rare and variable phenomenon, but more recent data indicate that this may not be the case (88). The split-root system, which allows spatial separation of the pathogen from the compost-treated root part, and thus eliminates its direct effect on the pathogen, has been used to study plant-induced responses to soilborne diseases (67, 120, 122).

Plant immunity research has advanced considerably, and key genes mediating the activation of plant responses, as well as the signaling networks that operate during this perpetual interaction of plants with their environment, have been recognized (92, 114). Nevertheless, we still lack sufficient understanding of the integration or natural variation of the different lines of defense that apparently operate in the environment. The involvement of hormones in the defense-signaling pathways has been repeatedly confirmed, and it is apparent that we are facing a situation in which the recorded response depends, in many cases, on the plant species and the strategy of the pathogen. More informational gaps exist concerning the influence of the environmental parameters and how these formulate the plant response toward the most beneficial net result in terms of plant survival. Plant growth and plant defense run in parallel, and how the plant achieves this energy trade-off remains unclear.

Taking into consideration the complex nature of composts, the stimulation of several interconnected defense mechanisms by different inducing factors is to be expected. Information on the molecular basis of the protective role of composts is still limited, and the signaling pathways mediating the plant response remain unclear. An increased state of resistance, similar to the systemic acquired resistance (SAR) manifested by the

induction of plant defense–related genes that serve as hallmarks of induced immunity in plants, such as peroxidase, β-1,3-glucanases, and pathogenesis-related 1 gene (*pr1*) in the leaves of plants growing in compost media, has been reported in some cases (58, 91, 123). In the absence of pathogens, Kavroulakis et al. (61) described the elicitation of increased expression and the spatial induction pattern of certain *PR* genes in the roots of tomato plants grown on compost. Compost treatment has also been shown to result in primed plants, i.e., plants in an alert state, capable of more efficiently combating a great variety of pathogens and pests (97). Induced resistance in plants, exhibited as SAR, induced systemic resistance, or priming, has also been repeatedly reported as the mode of action for specific microbial agents that were originally isolated from compost media and act as biological control agents. Alfano et al. (5) found that *T. hamatum* 382 consistently modulates the expression of genes in tomato leaves without activation by a pathogen. However, except for *PR5*, the main markers of induced systemic resistance were not significantly induced. In a similar case, a nonpathogenic *Fusarium solani* strain (FsK), able to protect tomato plants both locally and systemically, had the ability to alter salicylic acid–mediated plant responses (60). Using mutant plant lines, the authors showed that the ethylene-signaling pathway is also required for the mode of action used by the fungus to confer resistance. Furthermore, composts affect plant vigor: Plant growth may be stimulated and plant vigor induced, even when the composts are not suppressive. It may be that the suppressive trait of composts represents a quantitative difference of certain thresholds in the signaling pathways that characterize each plant.

EFFECT ON SOIL MICROBIAL ECOLOGY

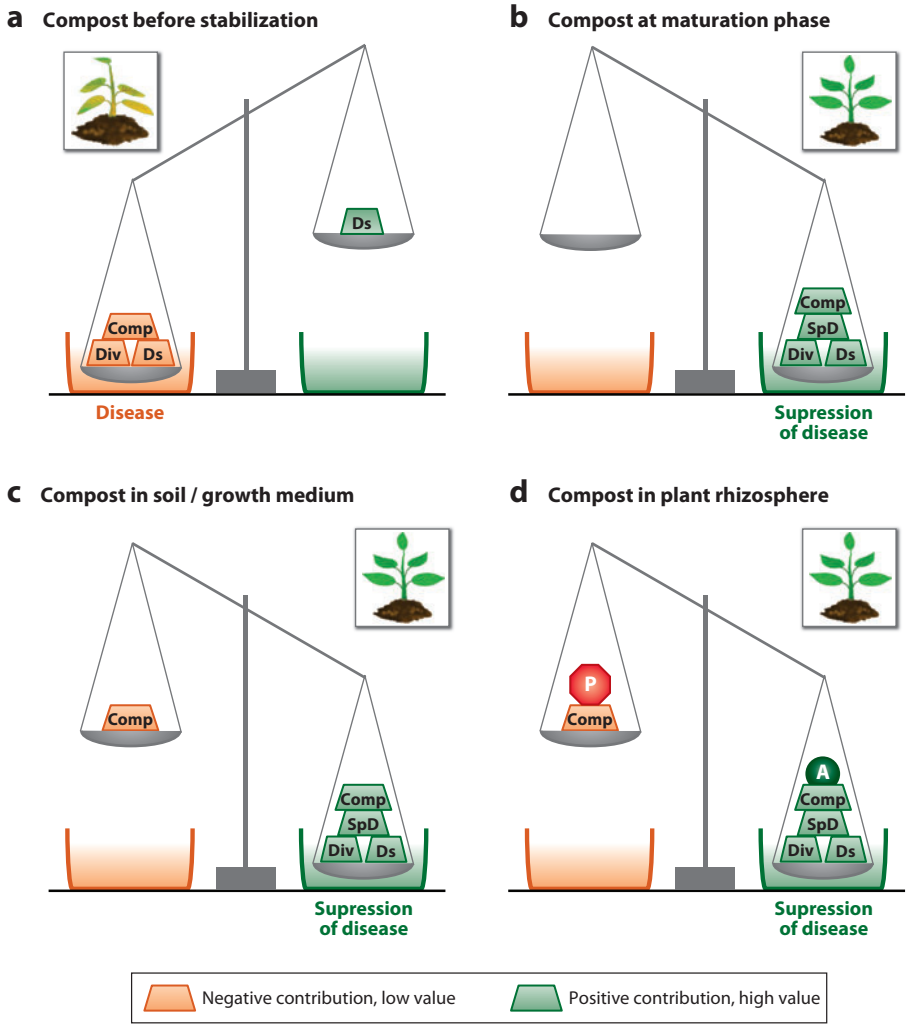
As discussed above, composition and dynamics of the microbial consortia harbored by composts depend heavily on the stage of the composting process, particularly the critical recolonization during the cooling phase, and probably on the raw materials used.

Figure 1

The disease suppression balance. Biotic and abiotic factors play a role in the development of pathogen suppressive microbial consortia (SMC). We consider six main factors and assume that the development of SMC is favored by maintenance of high microbial density (Ds) concurrent with high microbial diversity (Div). It is enhanced by high spatial microenvironment diversity (SpD) and competition for nutrients and carbon (Comp). It is also favored by coevolution of beneficial microbes with plants [A (pathogen antagonists, mutualists, systemic resistance inducers)]. On the contrary, coevolution of the pathogen (P) with plants may deter the suppressive effect. This role changes at different stages of compost production and application, leading to either disease (*orange*: negative contribution, low values) or suppression of disease (*green*: positive contribution, high values). Four stages are described: (*a*) compost before stabilization (no selection for SMC, disease); (*b*) stabilized, maturation phase (selection for SMC-disease suppression); (*c*) compost applied to soil or growth medium (selection for SMC-disease suppression); and (*d*) compost in plant rhizosphere (selection for SMC-disease suppression). The figure shows that disease-suppressive conditions prevail during compost maturation and following application in soils and growth media (high Ds, Div, SpD, and high competitions for carbon and nutrients) but not before compost stabilization (fluctuations in Ds, poor Div with only thermotolerants present, abundant carbon and nutrients leading to poor competition). Management practices, such as fertilizer applications and rotation schemes (*not shown*), may moderate or even cancel compost suppressiveness by affecting these environmental factors (changes in carbon and nutrient availabilities) or plant-microbe interactions processes (A and P). Simple conceptual models of this kind, focusing on microbial ecology at interfaces, have an explanatory rather than a quantitative/predictive value. They are also not expected to integrate the whole complexity of interactions between microbial genetics, evolution theory, and plant-microbe interactions on which compost suppressive effects appear to be based from a top-down point of view. Conceptual models may, however, be improved and refined by the accumulating empirical data and aid resetting research priorities and hypothesis formulation.

During their maturity phase, composts act as supreme carriers for these microbial consortia. Therefore, regardless of the mode of action conveying plant protection and antagonistic activities, amendments of soil or growth media with these composts lead to direct disturbance of the indigenous microbial populations, including plant pathogens, and the establishment of new equilibria (**Figure 1**). At the same time, the plant itself plays a very important role in determining the diversity and activity of the microbial community that will eventually predominate in the rhizosphere (104, 117) through cross talk with rhizosphere microorganisms, secretion of plant exudates, and even the production or degradation of quorum-sensing signaling molecules (35). In fact, plant species appear to be a dominant factor influencing the composition of the microbial communities in the rhizosphere and maybe even the selection and activity of antagonists (11, 36) and of biocontrol agents with specific genetic abilities within microbial species (12).

The challenge is to derive conclusions about the suppressive function of microbial rhizosphere communities following compost application by studying community composition and structure over time. Metagenomics, in



which the metagenome (genetic material from an environmental sample rather than a single microorganism) is sequenced and studied, offers a powerful tool to address the complexity present in compost-amended rhizospheres. Nevertheless, although state of the art, these approaches only represent a snapshot in time and are not suitable for addressing the complex microbial community dynamics, as this is modulated by biological interactions that accompany the growth and defense of the plant. It is imperative to (1) focus on critical niches of complexity, such as seed, root, tissue, and aggregate surfaces and their interfaces, and, more importantly, (2) develop theoretical ecological considerations and, accordingly, design experiments to test specific hypotheses in order to link our analytical output to ecological significance and to disease-suppression effects. The tools for targeting specific microbial groups as well as functional genes, apart from ribosomal-DNA genes, are only valuable when they complement the testing of specific hypotheses, ecological theory considerations, or data indicating the nature of the microbial involvement. The identification of microbial agents that may induce suppressive effects has to be related to ecophysiology and compost microenvironment conditions that are prone to temporal and treatment changes. For example, a wide range of fungal and bacterial antagonists may not efficiently produce chitinases in the presence of simple sugars, such as glucose (29, 39, 70), and the physiological profiles of microbial agents change in the context of the microbial consortia with which they interact (36).

A theoretical ecological framework for studying microbe-mediated plant disease suppression in the context of diffuse coevolution of microbes has been recently reviewed by Kinkel et al. (62). Diffuse coevolution leads to the selection of a specific trait by interactions with multiple species rather than a single species population. Kinkel and coauthors advocate that "microbial interactions within the soil community are likely the primary force imposing selection for the enhanced antagonistic activities crucial to disease suppression (62). They refer to empirical indications that are likely to drive selection for antagonistic populations in the framework of diffuse microbial coevolution-specific ecological and evolutionary forces, for example, the contribution of green manure to the active management of improved pathogen-inhibitory activity of *Streptomycetes* in soil (118). Selection for antagonistic phenotypes is viewed in light of the benefit that antagonistic traits, such as exoenzyme, antibiotic, or siderophore production, confer in the interaction with coexisting microbes. This offers a rare conceptual model for analyzing progress toward disease suppression, and we feel that this is an ecological framework worth exploring with respect to compost-mediated suppression of plant diseases. The role of compost applications in (*a*) increasing microbial density and diversity, (*b*) conveying average, but not excessive disturbance, and (*c*) resulting in slow-release nutrient availability is in accordance with a positive role in increasing antagonistic populations. However, three points need to be critically approached: (*a*) Apparently, the diffuse microbial coevolution framework is bound not to encompass all mechanisms and processes leading to disease suppression—consistent improvement through meta-analysis of empirical research and additional theoretical frameworks are needed; (*b*) even when the short microbial-turnover cycles are taken into account, direct effects following compost applications on plant disease suppression imply that time for coevolution is limited, and other mechanisms may be involved—the latter could perhaps include preshaped microbial communities during compost maturation or direct compost effects causing a drift in rhizosphere microbial population frequencies rather than coevolution effects; and (*c*) direct effects at the level of plant-induced resistance and microbe-plant cross talk certainly appear to involve the plant in complex tripartite interactions. Plant-microbe interactions leading to either coevolution or merely physiological adaptation are critical for shaping disease-suppressive effects in the rhizosphere. For example, the case of *Trichoderma* reflecting the multifaceted mode of action of an

organism originally isolated from compost (32, 69) and cases such as the avenacin/avenasinase system developed in oats/*Gaeumannomyces graminis* in regard to reflecting plant/pathogen coevolution (18) clearly indicate that a wider theoretical framework involving microbial coevolution and plant-microbe interactions should be considered.

BEYOND PHYLOGENETIC ANALYSIS: THE POSTGENOMIC ERA

The question remains: How can we pinpoint the common genetic factors that govern each specific case of plant protection? More research is needed to address the functionality of the effective biological network (54, 110). Fortunately, the phylogenetic approach to suppressive microbial groups appears to be a useful choice. Although not specifically investigated, there are no indications of an important role for horizontal gene transfer in most cases of microbially mediated disease suppression, contrary to other microbially mediated functions, such as degradation of pollutants. Transcriptome analysis, by using random shotgun DNA microarrays (76) and comparing suppressive to nonsuppressive compost-amended rhizosphere samples, represents a feasible approach. Meta-transcriptome analysis of complex soil microbial communities under contrasting conditions has also been used successfully (103), and such methods could find application in compost-treated soils as well. Advances in mass sequencing of environmental samples (including shotgun sequencing and pyrosequencing) as well as advances in bioinformatics may offer promising alternatives. To date, the microbial communities harbored by composts and compost-amended soils are too complex to enable full assembly of every individual genome present (10). Nevertheless, aside from providing genetic information on noncultured microorganisms, such approaches may give information on the genes responsible for important enzymatic functions in soil (107) and compost samples (66). Identifying specific genes in ecosystems that have a high degree of microbial diversity, such as compost, is challenging, but recent metagenome studies have demonstrated that it is possible to assign functional annotations to partial gene sequences from shotgun sequence reads with a reasonable degree of accuracy (6). These experiments could be further expanded to involve more than one plant species and, preferably, more than one soil/compost type. It should, however, be noted that for the moment, metagenomics analyses exclude eukaryotes, mainly owing to cost limitations. Such limitations pose some risk because the role of fungal strains contributing or potentially determining the suppressive phenomena may thus be masked. In addition, there is limited knowledge on the metabolites that are produced by the microbial consortia and the plant response to the presence of composts. Powerful metabolomics platforms exist today that could tackle the formidable task of deciphering the role of disease-suppressive metabolites produced by the microbial consortia or the plant in the presence of compost. Similarly, data are lacking on the proteins and enzymes present in suppressive versus nonsuppressive composts. Metaproteomics is yet another approach with great potential to provide suppressive compost-specific molecular markers. Overall, it is anticipated that, as in other cases of complex biological systems (e.g., 31), with the integration of data sets generated by postgenomic methodologies applied to suppressive compost, more secrets of the compostome, i.e., systems biology of the compost microbiome and plant-microbiome interactions, will be revealed to provide a meaningful and accurate interpretation of the complexity and dynamics of compost suppressiveness.

CONCLUSIONS

Working with nature is the new paradigm in applied life sciences. In phytopathology, this has been reflected by research in developing alternatives to replace wide-range biocides. Plant disease–suppressive properties in composts appear too frequently (in approximately

50% of tested composts) to be attributed to coincidence, and they are clearly related to microbial function. However, compost and rhizosphere microbiology, microbial ecology in particular, present a high level of dynamic complexity. Until recently, our methodological tools have allowed taking only detailed snapshots of this complexity. Despite the use of sophisticated statistical analytical means, our understanding of the system dynamics is far from sufficient. The development of metagenomic techniques may prove more helpful in this regard. However, the difficulty of linking laboratory high-throughput data to ecological theory still remains. Thus, we should ask whether predictability and consistency in compost-derived plant disease suppression are feasible or even practical goals. Hegel described three dialectical stages in approaching the nature of reality: thesis, antithesis, and the tension between them resolved by synthesis. It is surprising how this repetitive process fits into the humble microbial world of composts, soil rhizospheres, and potting media when we search for the outcome of microbial dynamics and plant-microbe interactions. We have referred to cases in which the disease-suppression outcome is predictable and the processes leading to it are well understood. Most of these cases involve the activity of specific microbial agents and/or a partial understanding of induced systemic resistance but not general suppression effects involving cumulative activity of favored microbial communities.

It is also clear, however, that working with nature demands a compromise in terms of outcome control, as the outcome is not biocide enforcement but a result of the synthesis of natural dynamics and balances. A practical approach toward enabling growers to choose the appropriate compost for a specific disease without jeopardizing the crop they wish to cultivate should be sought. Multiple routes that converge on the production of effective composts are available, for example, developing suppressive composts that are additionally fortified with specific microbial agents or developing criteria for the quality control of suppressive composts on the basis of pure compost characteristics. Empirical research has led to the development of valuable compost-specific pathogen products that may be put to practical use following optimization of production routines. In parallel and in the long run, however, the most promising approach is to focus on microbial ecology in composts, on the role of microenvironments in the root zone and rhizosphere, and on a molecular understanding of compost-induced immunity in plants. The real challenge lies in applying today's knowledge on plant immunity (available mainly for model plants and certain biocontrol agents) to understanding integration and regulation of the multidimensional signaling cross-communication of biotic and abiotic parameters in a real ecosystem, consisting of the plant, the microbial community of the compost, the pathogen, and the microbial community of the soil in an ever-changing abiotic environment under field conditions. Apart from developing consistently suppressive composts for plant diseases, side developments in the fields of functional microbial ecology and fundamental plant-microbe interaction mechanisms will prove to be rewarding.

SUMMARY POINTS

1. Suppressive compost provides an environment in which plant disease development is reduced, even when the pathogen is introduced in the presence of a susceptible host.
2. Plant disease suppression by compost is a widespread and ubiquitous phenomenon that occurs when diverse types of compost are applied for the control of a variety of pathogens.

3. Plant disease suppression is the direct result of the activity of antagonistic microorganisms that naturally recolonize the compost during the cooling phase of the process.
4. Understanding compost microbiology is a necessary first step to better understand the link between pathogen suppression and microbial population dynamics.
5. Harnessing the natural phenomena of disease suppression for plant disease control demands persistence and a compromise regarding the outcome consistency. This is because the consequence is not complete control as achieved by biocidal enforcement but that of a subtle synthesis of natural microbial dynamics and balances.

FUTURE ISSUES

1. An ecological theory is required to guide research in microbial ecology in complex environments, such as compost. This gap limits hypothesis-driven research and interpretation of metadata.
2. Research should focus on critical niches of complexity, such as seed, root, tissue, and aggregate surfaces and their interfaces, for which innovative and robust experimental approaches are needed.
3. Data sets on suppressive composts generated by postgenomics methodologies beyond phylogenetics (based on rRNA or cultivation) should be produced. Interpretation and integration of data will unravel the secrets of the compostome, the compost microbiome, and plant-microbiome interactions. This will give novel insights into the complexity and dynamics of compost suppressiveness.
4. A systems biology approach is necessary to address the complex, simultaneous, and dynamic interactions of variable communities that affect plant health.
5. Assays need to be developed for the prediction of the capacity of an individual compost to suppress a specific plant pathogen or group of pathogens. Such assays could be useful for quality control and commercial utilization of suppressive composts.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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Errata

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