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Social Evolution and Cheating in Plant Pathogens

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Abstract

Plant pathogens are a critical component of the microbiome that exist as populations undergoing ecological and evolutionary processes within their host. Many aspects of virulence rely on social interactions mediated through multiple forms of public goods, including quorum-sensing signals, exoenzymes, and effectors. Virulence and disease progression involve life-history decisions that have social implications with large effects on both host and microbe fitness, such as the timing of key transitions. Considering the molecular basis of sequential stages of plant–pathogen interactions highlights many opportunities for pathogens to cheat, and there is evidence for ample variation in virulence. Case studies reveal systems where cheating has been demonstrated and others where it is likely occurring. Harnessing the social interactions of pathogens, along with leveraging novel sensing and -omics technologies to understand microbial fitness in the field, will enable us to better manage plant microbiomes in the interest of plant health.

INTRODUCTION

Virulence: the severity of disease symptoms on a host

Cheating: in cooperative interactions, when a strategy increases its own fitness while decreasing the fitness of its interaction partner

Free-loading: a form of cheating in which an individual benefits from but does not contribute to a public good

Coincidental virulence: pathogens that do not complete their life cycle in the host and thus do not experience selection on the traits involved

A plant is a complex and dynamic environment for the microbes that colonize it. An individual plant is vast and ensconces itself in a milieu of diverse chemical compounds, within which it comprises multiple types of surfaces atop rapidly responsive cellular processes that integrate information about the above- and belowground environment. A bacteria, fungus, or virus that interacts with a plant goes through many lifetimes during this interaction, given that their generation time may be on the scale of hours; this fact means that evolution can occur within these microbes during a single generation of their longer-lived host. Furthermore, even if a single cell or viral particle initially encounters a plant alone, if this individual is successful it will soon become a population. Populations and communities of microbes interact with one another as well as with their shared host, and these social interactions set up all manner of opportunities for cooperation and conflict (102, 125, 143, 147). The plant host can be considered a public good and its exploitation can result in a tragedy of the commons (68), wherein short-term gain through virulence outweighs long-term prosperity. Conversely, many of the mechanisms that pathogens leverage during infection act cooperatively to alter the environment and are thus also public goods and potentially subject to cheating; e.g., a free-loading former pathogen might reduce the harm to the shared plant host. Therefore, cooperation and conflict between pathogens may result in worse outcomes for the plant, or these processes may lessen pathogen virulence. Understanding the principles and mechanisms that shape microbial social interactions thus has profound implications for how we understand and manage plant disease.

This review first describes the theoretical frameworks underpinning the evolution of virulence and social evolution, including evolutionary game theory. I next explore the extent to which the social interactions of cooperation and cheating between microbes could impact plant–pathogen interactions by considering opportunities for cheating as a result of the molecular and physiological processes that occur during infection and disease progression. For these cases of selection to result in evolutionary effects, there must be variation in virulence strategies, so I consider mechanisms underlying this variation. I then turn to case studies and consider the evidence for cooperation and cheating in pathogens along with the impact of these processes on plant disease. Finally, I consider biocontrol and how humans can leverage an understanding of microbial social evolution to more effectively manage disease.

THE THEORY OF VIRULENCE AND SOCIAL EVOLUTION

Predicting how virulence, the severity of a disease caused by a pathogen, evolves requires thinking from the microbial perspective (25, 43, 44, 90). Pathogens that cause disease may do so for two distinct reasons that have dramatically different evolutionary implications. Coincidental virulence occurs when a microbe that typically inhabits another environment encounters and is able to infect a host but does not transmit from that host to others (23). Although we currently lack examples of coincidental virulence in pathogens of plants, there are several examples that affect humans. The soil bacteria *Clostridium botulinum* and *Clostridium tetani* cannot be transmitted between humans, but the neurotoxins they produce cause botulism and tetanus, respectively (7). In these cases, infection is an evolutionary dead-end that is maladaptive for the microbe, and there is no selective pressure favoring microbial mutations that enhance virulence; understanding these types of virulence requires knowledge of the selection pressures experienced in other parts of the pathogen's life cycle. For pathogens that are transmitted between hosts, particularly obligate pathogens that cannot reproduce outside their host, early conceptual models predicted that virulence would always be selected to decline because the pathogen requires the host (described in 90). However,

more rigorous theory has clearly shown that the key pathogen life-history trait under selection is the transmission rate between hosts (5, 25, 44). In some cases, there is a trade-off between virulence and transmission, and when this occurs initially high virulence evolves to a lower, optimal level. In other cases, increasing virulence may increase transmission rates, e.g., if producing spores harms the host but enhances dispersal of the microbe. Initial models assume that a single, clonal pathogen population is interacting with a host, but theoreticians have also considered cases of coinfection and superinfection (3, 101, 110).

Public goods games: a scenario whereby resources are shared among multiple individuals and a tragedy of the commons can arise

Social Evolution and Multilevel Selection Theory

Social evolution theory arose out of the early group selectionist view positing that traits could evolve "for the good of the species." In 1964, W.D. Hamilton first formally described the process of kin selection, wherein an allele that encodes a behavior that is costly to the individual actor can nonetheless be favored by natural selection if the behavior benefits individuals with copies of the same allele more than other individuals that lack the allele (65, 66). This theory has been generalized in several ways relevant to plant pathology, including multilevel selection and between-species cooperation. Multilevel selection theory applies when organisms experience selection within a group, such as when they are colonizing a host plant, as well as between groups, such as when they disperse between hosts (52). In 1975, D.S. Wilson first showed that in this trait-group model, alleles that promote group-level fitness but are costly within groups can spread (145); an experimental demonstration of this used bacteria that could both produce and detoxify an antibiotic (32). The flip-side of this process is the potential for cheating alleles to arise within a group and diminish group-level fitness. Cheating can occur in otherwise cooperative interactions within and between species (10) when an allele increases the fitness of an individual actor but decreases the fitness of its partner (58, 78). A famous instance of this phenomenon is the tragedy of the commons in which a public good is selfishly exploited by individuals within a group, even when this leads to the demise of the group (6, 68). An unfortunate consequence of evolution by natural selection is that it is intrinsically short-sighted, and higher-order effects of multilevel selection require more time to act and thus must be strong enough to overcome within-group selection for selfish behavior (44). Conflicts between individuals can readily arise whenever there are public goods, which in the case of plant pathogens include both shared resources derived from the plant host and compounds produced by the microbes involved.

Although multilevel selection theory and public goods games are powerful tools appropriate for modeling plant–pathogen interactions, the special cases that arise in simpler two-player games illustrate the range of strategies and outcomes possible in more realistic scenarios (8, 60, 72). Two-player games typically consider simplified strategies in which each partner can either cooperate or defect, and the partners accrue fitness payoffs depending on their joint behavior. The costs and benefits of cooperating and defecting then completely define the structure of the game and its evolutionary outcome. Note that defecting is not the same as cheating, as defecting is simply the behavior of not cooperating; for cheating to occur, defecting must increase the fitness of the defector while decreasing the fitness of its partner. The classic game in which cheating occurs is the prisoner's dilemma, whereby one partner increases its fitness by defecting and its partner has reduced fitness regardless of its strategy (8). In the prisoner's dilemma, both partners would obtain the highest fitness if they cooperated, but the evolutionarily stable strategy is for them to both defect. In the case of plant viruses, plants are commonly infected by two (or more) viruses at the same time, and these pairs of viruses can be categorized into the different types of two-player games (45). In no instances did the prisoner's dilemma occur and in some cases the evolutionary game showed context dependency in which the fitness payoff matrix depended on the host, the

viral genotypes, or the environment. In 64% of cases, both viruses had increased fitness during coinfection, 44% experienced a hawk–dove or snowdrift game, and 8% experienced a stag-hunt or coordination game. Ironically, the case where cooperation increases the fitness of both players is not widely considered in theoretical models because it is too easy to explain; an adaptive dynamics model allowing the fitness outcomes of a two-player game to evolve found that the prisoner's dilemma readily evolves into a mutually beneficial, conflict-free game (146). However, even when conflict is present, cheating does not always result in the extinction of the cooperative type. In the snowdrift/hawk–dove game, both cooperating and defecting strategies coexist at a stable equilibrium that is determined by the costs and benefits of the interaction; unlike the prisoner's dilemma, under this game spatial structure reduces cooperation (69). Coexistence occurs when fitness depends on the frequency of the strategies present and this frequency-dependent fitness readily occurs (124). The large literature on evolutionary game theory has explored mechanisms by which cooperation can be stabilized; these include repeated interactions, spatial structure, signals, and punishment (40).

Potential Impacts of Pathogen Cheating on Plant–Pathogen Interactions

The connection between microbial growth within a plant host and the virulence of that microbe, in terms of the actual damage to the host, is not always straightforward. Although theoretical models assume this relationship is positive, empirical data show that pathogen fitness and host damage can be positively correlated, negatively correlated, or show no relationship for numerous bacteria, fungi, and viruses (reviewed in 127). Thus, cheating by pathogens could result in any possible disease outcome for the host. To add further complexity, eco-evolutionary models of mutualism show that diversification of a cooperating lineage into a cooperator and a cheater—sometimes referred to as a third-party exploiter in the mutualism literature when it is a separate species (22)—can result in character displacement whereby the cooperator becomes even more cooperative (47). In three-species models, exploiters can result in evolutionary immunization, a situation in which the early presence of an exploiter leads to a shift in the equilibrium strategies of two cooperators such that the exploiter can no longer invade the community (48).

OPPORTUNITIES FOR CHEATING

For plant pathogens to cheat, there must be social interactions in which there is a public good that is being produced by either the microbe or the host. These public goods are compiled for reference in **Table 1**. Considering the stages of infection systematically, I highlight numerous potential opportunities for cheating (**Figure 1**), pointing to relevant theoretical models and work

Table 1 Public goods produced by plant pathogens

Disease symptom	Public good	Pathogens
Rot	Cell wall–degrading enzymes	*Pectobacterium carotovorum*, *Dickeya solani*
Wilt	EPSs, xanthan gum	*Ralstonia solanacearum*, *Xanthomonas oryzae* pv. *oryzae*
Gall	Habitat, opines	*Agrobacterium tumefaciens*
Water-soaking	EPSs, HopM1 effectors	*Pseudomonas syringae*, *Xanthomonas malvacearum*
Spots (HR)	Effectors	Most bacteria
	Quorum-sensing signal	Most bacteria
	Siderophore	Most bacteria

Abbreviations: EPSs, exopolysaccharides; HR, hypersensitive response.

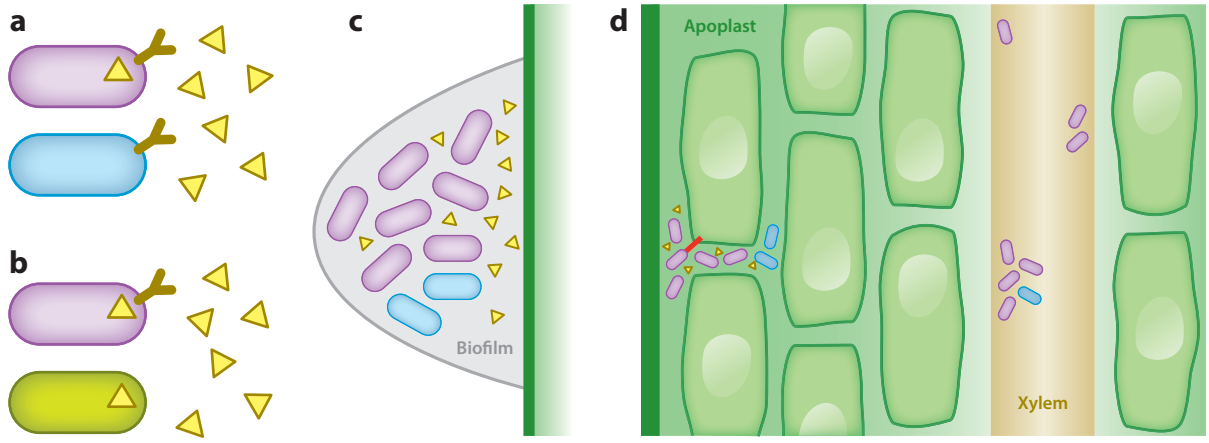

Figure 1

Opportunities for cheating in plant–pathogen interactions. (*a*) Public goods game in which the top cell both produces the public good (*triangles*) and is able to consume it (receptor), whereas the bottom cell consumes the public good but does not contribute to its production. The public good could be a quorum-sensing (QS) signal, exopolysaccharide, effector, exoenzyme, or any other molecule that can increase a cell's fitness. (*b*) Lying or coercion in a QS interaction, in which the bottom cell produces the signal but does not perceive it and thus does not engage in potentially costly cooperative actions such as the production of virulence factors. (*c*) Biofilm on a plant surface in which a subset of the cells (purple cells are producers and consumers, as in panels *a* and *b*) is producing a public good, such as an exoenzyme that weakens the plant cell wall. (*d*) Social interactions, and thus the potential for cheating, occur in the apoplast (e.g., *Agrobacterium tumefaciens*) and xylem (e.g., *Ralstonia solanacearum*). Apoplast: The red spike represents the type IV secretion system that injects the Ti (tumor-inducing) plasmid into a plant cell. Public goods (*triangles*) in the apoplast could be produced by either the plant cell (e.g., opines) or the bacteria (e.g., toxins). Xylem: Cells that proliferate more rapidly may result in earlier host death than those exhibiting more prudent within-host growth.

in other systems. These hypothetical scenarios indicate that there are abundant opportunities in which natural selection could favor cheating variants.

Sensing Opportunity: Signal Production, Perception, and Destruction

The earliest stages of interaction between hosts and potential pathogens occur through the exchange of signal molecules. Plants secrete numerous volatiles, nutrients, and diffusible small molecules into their rhizosphere and phyllosphere; microbes and the macrobes that vector them can be attracted to these host compounds. Because sensing host chemicals and moving toward them is critical for an interaction to occur, microbes could interfere with their competitors by degrading the host's signals or producing molecules that directly antagonize competitors. Quorum sensing (QS) is a mechanism that the majority of bacteria use to detect and respond to their biological environment (81, 125, 140). Many bacteria secrete a diverse array of small molecules [N-acyl-homoserine lactones (AHLs), furanosyl borate diester, γ-butyrolactone, oligopeptides, cyclic dipeptides, bradyoxetin, and butyrolactones] (140) and express a variety of virulence genes only when the concentration of particular molecules is above a certain threshold. The high diversity of these molecules could be due to antagonistic coevolution through cheating by eavesdropping on the signals of other microbes (38) or to character displacement because sharing signals would lead to noisy and perhaps ineffective responses. An example of a highly unique QS signal is the volatile signal molecule 3-OH palmitic acid methyl ester (3-OH PAME), which is only known to be produced by the bacterium *Ralstonia solanacearum* (50); this molecule is sensed by the gene *phcA*, which regulates the transcription of effectors, motility, secondary metabolism,

QS: quorum sensing

N-acyl-homoserine lactones (AHLs): the most common signal molecule used in quorum sensing

and iron scavenging (16, 57, 131). QS signals and their receptors are typically encoded in operons along with other coexpressed genes, such as the classic *luxR* and *luxI*, which encode the receptor for and the synthesis of an autoinducer (3-oxohexanoyl homoserine lactone; VAI-1) in *Vibrio fischeri* (54). Receptors that are used in eavesdropping on other species' signals occur alone in the genome, such as the *qscR* gene in the generalized pathogen *Pseudomonas aeruginosa*; this gene responds to AHLs that *P. aeruginosa* does not itself produce (137). Other receptors related to QS systems respond to compounds produced by the plant, and plants can respond to the production of some QS signals in bacteria (137). Quorum quenching occurs when QS signals are actively degraded and both plants and bacteria can produce enzymes targeting these signals. For example, plant flavonoids have been shown to directly bind to QS receptors in *P. aeruginosa*, suppressing virulence (112), and the soil bacterium *Ideonella* sp. 0-0013 produces a β-hydroxypalmitate methyl ester hydrolase (βHPMEH) that degrades the QS molecule 3-OH PAME and prevents virulence gene expression by *R. solanacearum* (132).

QS has been hypothesized to exist for purely selfish reasons rather than as a mechanism for social information exchange, given that these systems act as diffusion sensing systems: When a bacterium is alone and secreting a diffusible molecule, the local concentration of that molecule reflects whether the bacterium is proximate to a surface (121). Secreting attachment molecules or degradation enzymes in a high-diffusion environment would be costly and unlikely to provide a return on investment. However, the information that these extracellular signals provide could be considered a public good that any cell with a cognate receptor can potentially exploit. To understand how cheating might manifest during QS, we must consider how these signals influence bacterial fitness. If the cost of producing the signal is high, then cells could cheat by not producing the signal but still responding to it, if responding is adaptive. However, given that QS systems regulate many aspects of virulence, including other potentially costly, public goods–producing behaviors (see below), the cost of producing the signal might be low compared to responding to the signal, and signal-blind mutants that still produce the signal (liars) could cheat by coercing other cells to implement further actions contributing to virulence. Both of these forms of exploitation have been found to be theoretically possible (34). An elegant example using the plant and animal pathogen *P. aeruginosa* showed that both signal-negative and signal-blind QS mutants have higher fitness than wild-type bacteria in mouse burn wounds, with the signal-blind mutant experiencing stronger positive selection and more rapid increase from low frequency (125).

Approaching: Competition, Cooperation, and Cheating in the Rhizosphere

Epiphytic colonization is a key step for environmentally transmitted pathogens, and the vast majority of plant-associated microbes never make it past this environment. The rhizosphere, in particular, is the subject of intense study currently because roots act as the guts of a plant, playing critical roles in nutrient acquisition (77). There are many excellent reviews of this rapidly moving field (26, 56). The rhizosphere is characterized by a high degree of spatiotemporal heterogeneity, with hot spots of enzyme activity (133) and elemental fluxes (76). These hot spots represent sites of strong competition and provide opportunities for cooperation and cheating; spatial structure is one of the classic mechanisms by which cooperation within and between species can be stabilized (41, 46, 109, 124). Microbes engage in multiple public goods games within a local patch of rhizosphere, many of which include the plant as a partner (21, 71, 85, 128). Nitrogen and phosphorus availability can be enhanced by the secretion of extracellular enzymes (4, 108), and modeling suggests that cheating should readily arise whenever costs are high relative to the degree to which these public goods are able to diffuse away from the producer (4). Another critical aspect is the rhizosphere pH, which both plants and microbes can alter (18, 99), because pH exerts strong

selection on the composition of the microbiome (39) and determines metal availability. Metals, particularly iron, are often limiting micronutrients for microbial growth and many bacteria can produce diverse siderophores that enhance iron solubility (93). These siderophores typically require a cognate receptor to be taken up by the cell. The generalist pathogen *P. aeruginosa* produces multiple forms of the siderophore pyoverdine constitutively at low levels; when one of these molecules returns to the cell complexed with iron, the result is upregulation of this particular type of pyoverdine siderophore. In both human infections and soil, nonpyoverdine-producing strains are often recovered and in vitro competition assays have documented that these nonproducers are cheaters that benefit from the pyoverdine produced by co-occurring cooperators (24, 27, 58, 59, 62). Microbes can sometimes utilize other species' siderophores, as illustrated by *Pseudomonas putida*, which responds to iron availability modulated by the siderophores enterobactin and aerobactin produced by *Enterobacter cloacae* and is able to uptake iron using these heterologous complexes (94). In other cases, iron competition can restrict plant pathogens, as shown by in vitro experiments documenting the role of *Pseudomonas* spp. pyoverdines in inhibiting the growth of bacterial [*Pectobacterium* (formerly *Erwinia*) *carotovora*] and fungal pathogens (*Fusarium oxysporum*, *Gaeumannomyces graminis*, *Gaeumannomyces candidum*, *Pythium ultimum*) (93).

JA: jasmonic acid

Entering Into: Surface Attachment and Penetration

Fungal pathogens of plants vastly outnumber bacterial pathogens (138). One potential reason for this discrepancy is that physical barriers created by plants to protect their tissues are likely more effective against the much smaller bacteria (0.5–2 μm diameter, 1–10 μm length) than against large fungal hyphal cells (1–10 μm diameter, centimeters long). Many fungi, upon encountering a plant, produce specialized infection structures called appressoria that attach to the plant surface, secrete cell wall–degrading extracellular enzymes, and then give rise to an infection peg that uses turgor pressure localized at a small point to penetrate the plant (14). Bacterial cells can readily enter through multiple gates: (*a*) through stomates, which *Pseudomonas syringae* causes to initially open through the action of the jasmonic acid (JA) mimic coronatine (148); (*b*) through the porous lenticel tissue on stems, leaves, or roots (75); or (*c*) belowground at epidermal cracks that form during lateral root development (61). Soilborne bacterial pathogens that colonize a growing root can in theoretical models be left behind if their attachment and detachment dynamics are too slow (42). Bacterial attachment typically consists of two phases, an initially reversible, rapid binding followed by irreversible binding. Initial binding can occur when rotating flagella lodge in tissue crevices or adhere to plant surfaces because of weak binding through their repetitive structure (123). Some bacteria, including *Agrobacterium tumefaciens*, exhibit initial polar binding through UPP (unipolar polysaccharide) (100). Irreversible binding occurs through structures such as fimbriae (*Erwinia rhapontici*, *P. carotovora*, *P. syringae*, *Xanthomonas campestris*, and *R. solanacearum*), type IV pili (*P. syringae*), lipopolysaccharide (*R. solanacearum*, *A. tumefaciens*), cellulose (*A. tumefaciens*), and agglutinin (*P. putida*) (122). Adhesion is a competitive process that follows from the occupancy of limited sites, as deduced by early work modeling the rate at which avirulent *A. tumefacien*s inhibits colonization by virulent strains.

Many rhizosphere- and phyllosphere-inhabiting bacteria produce biofilms, an extracellular matrix binding cells together with an assortment of carbohydrates, proteins, and DNA. In addition to promoting binding to plant surfaces, biofilms can also provide protection to cells against predation, infection by bacteriophage, antibiotics, and dehydration (91, 144, 147). The secretion of biofilm matrix compounds therefore represents a public good and nonproducing mutants could have an advantage by exploiting producers. A famous example of this is the highly repeatable experimental evolution of biofilm cheaters in cultures of *Pseudomonas fluorescens* (120). Another

potential mechanism for cheating or exploitation could arise through interfering with biofilm adhesion or integrity through surfactants or degradation (141). The potential for exploitation could drive diversification of matrix compounds to reduce the potential that unrelated bacteria will invade a biofilm. Exopolysaccharides (EPSs) and cellulose are common components of biofilms, and although multiple forms of these molecules exist, with EPSs being particularly diverse, many bacteria can form mixed-species biofilms. Mathematical modeling shows that in conditions in which nutrients are flowing over a surface, the biofilm producer has higher fitness than a nonproducer because the producer cells end up on top (147); this may not apply to the rhizosphere and phyllosphere where nutrients may be coming directly from the surface of the plant tissue. A biofilm also serves the function of even further limiting diffusion of secreted molecules, so bacteria that secrete cell wall–degrading enzymes in this environment can be relatively certain that they will act locally. Even so, these enzymes are costly to produce and represent a public good by opening the door to everyone who is nearby.

EPSs: exopolysaccharides

HR: hypersensitive response

Becoming One With: Colonization and Remodeling of Plant Tissues

While entering a tissue, pathogens are interacting with the plant immune system, which consists primarily of rapidly acting signal transduction cascades culminating in the hypersensitive response (HR) and localized cell death, along with the production of reactive oxygen species and the synthesis of antimicrobial compounds. Interactions of these processes with the plant microbiome as a whole are reviewed elsewhere (64). Plant defense responses occur upon successful perception of infection through pattern recognition receptors (19), and significant effort has gone into identifying the molecular basis of infection and resistance. Plants recognize and respond to highly conserved biochemical signatures of bacteria, fungi, viruses, and insects, including specific motifs of flagellin, the peptidoglycan cell wall, and chitin (19), and microbes have an array of strategies to prevent plant defense responses and alter the function of plant tissue for their own gain. In some cases, these interactions are based on public goods and could thus be exploited by potential cheaters. Vectored microbes, such as the citrus greening agent α-proteobacterium *Candidatus* Liberibacter and most viruses, enter plant tissue via mechanical wounding by their animal carriers and therefore bypass the social interactions described up to this point.

In the apoplast, bacteria and fungi secrete effectors that alter host pathways to promote further infection (142). For example, the fungus *Phytophthora sojae* secretes the glucanase PsXEG1, along with an inactive decoy paralog PsXLP1 that has enhanced binding to the soybean host's apoplastic glucanase inhibitor protein, GmGIP1 (97). To the extent that these effectors' effects are shared among infecting cells, they may represent public goods. Bacteria also use QS-related proteins while colonizing the apoplast, such as the γ-proteobacterial pathogen *Xanthomonas axonopodis* pv. *glycines* (Xag), which has the QS receptor LuxR protein XagR that responds to a plant compound that increases during infection. When XagR is triggered, Xag switches off the production of adhesion genes and turns on biosurfactant production, thus enabling cells to spread within the apoplast (137). Homologous plant-responsive QS proteins are also found in the plant-associated genera *Pseudomonas*, *Dickeya*, *Rhodospirillum*, *Citreicella*, *Rhizobium*, *Ensifer* (*Sinorhizobium*), and *Agrobacterium* (137). In this case, the decision to boldly colonize the apoplast may be an example of a coordinated behavior, and it is possible that switching too soon or too late could have negative consequences for the population as a whole. In addition, the biosurfactants could be a public good, although we lack information on the fitness consequences of producing these molecules in competitive environments.

Some bacteria are able to reach the vascular system and thus be transported throughout the plant, through either insect vectors, as in the cases of *Xylella fastidiosa* and *Pantoea stewartii* subsp.

stewartii, or root colonization, as in the case of *R. solanacearum*. Viruses vectored by phloem-feeding insects can also colonize systemically. In the vasculature, bacteria risk prematurely clogging their habitat, and in the early stages of *X. fastidiosa* infection plants are symptomless with xylem vessels containing isolated cells or small colonies, while *P. stewartii* subsp. *stewartii* has specificity for parts of the protoxylem mediated by the EPS stewartan and *R. solanacearum* uses the type IV pilin PilA for adhesion and twitching motility (36). In the case of both *X. fastidiosa* and *R. solanacearum*, at high densities QS systems induce a physiological shift to higher virulence and rapid death of the host through wilting caused by bacterial proliferation and clogging of the xylem. Intriguingly, mutants that are hypervirulent and cause disease symptoms earlier have higher fitness than more prudent strains (36). The timing of this transition may thus represent a classic social dilemma with the potential for cheating. Similarly, in the soft-rot bacterium *P. carotovora* subsp. *carotovora*, QS regulates the production of cell wall–degrading enzymes and the decision to induce virulence is thus a social behavior (140). These extracellular enzymes may also represent a public good, and it would be interesting to know whether there is variation in their timing or level of expression.

Opines: a specialized class of nutrients produced by plant cells infected by *Agrobacterium* T-DNA

SA: salicylic acid

Plant pathogens have multiple ways of meeting their nutritional needs during infection. First, they can remodel plant metabolism to meet their carbon demands. The most dramatic example of this is the agent of crown gall *A. tumefaciens*, which inserts a tumor-inducing (Ti) plasmid containing the genes for biosynthesizing opines into its hosts' genome using a type IV secretion system. Engineered plant cells then secrete high levels of opines into the apoplast of galls and the bacteria have the genes required to catabolize these modified carbohydrates. Another example is the agent that causes rice bacterial blight, *Xanthomonas oryzae* pv. *oryzae*, which inserts multiple transcriptional activator–like effector (TALE) proteins PthXo1, AvrXa7, and PthXo3 that bind to the promoters of the host sucrose transporter genes *OsSWEET11* and *OsSWEET14* (135). Second, pathogens must cope with low levels of essential micronutrients such as iron; hosts have been hypothesized to purposely sequester these critical resources in the interest of nutritional immunity (73). As in the rhizosphere environment, secreted iron-chelating siderophores can help overcome this limitation. For example, *Erwinia chrysanthemi* produces two siderophores, achromobactin and chrysobactin, both of which contribute to virulence and population growth of the bacteria in African violet (53). Similarly, *X. campestris* pv. *campestris* produces xanthoferrin, which contributes to optimum virulence and growth in cabbage (114). In neither case are the siderophores required for virulence. In both host-secreted carbon compounds and bacterial-secreted siderophores, the nutritional resources are public goods, and there is potential for pathogens to cheat by consuming the resources without contributing to their production, assuming that this production entails a fitness cost to the microbe.

Pathogens have sophisticated mechanisms by which they interact with plant hormonal and defense signaling pathways. Many toxins interact with hormonal pathways (96), including the *P. syringae*'s peptide toxin syringolin A (SylA), which inhibits salicylic acid (SA) signal perception (104), and the various *Streptomyces* species' potential JA mimic coronafacic acid (CFA)-L-Ile (17). To the extent that hormonal system modification promoting infection by pathogens is systemic, these actions could create public goods that cheating mutants or unrelated microbes could exploit. When considered from the viewpoint of the pathogen, systemic acquired resistance and induced systemic resistance, phenomena in which interaction with an elicitor primes a plant for faster and more severe defense responses, could serve to preserve the host resources for the first infecting pathogen, assuming that they can successfully evade local responses. Thus, it is possible that pathogens could experience natural selection for traits that enhance systemic resistance even while they attempt to dampen local resistance.

Not all plant pathogens cause death, and many of those that do first exist as biotrophs before killing their host and transitioning to necrotrophs. This is clearly a critical life-history decision for

the pathogen that could represent a final social dilemma. In addition to possessing many effectors to inhibit plant innate immunity and the HR leading to cell death (106), pathogens also possess effectors that hasten the end. For example, *Colletotrichum higginsianum* infecting *Arabidopsis thaliana* shows three temporally distinct waves of effector gene expression with the final wave promoting cell death. Similarly, when infecting soybean, *P. sojae* first expresses effectors that inhibit cell death and then expresses a set of effectors that elicit cell death; misexpression at the wrong time reduces *P. sojae* virulence. Similar patterns are found in *Phytophthora capsici* and *Blumeria graminis* f. sp. *hordei* (135). Depending on how the speed of host death impacts pathogen transmission, selection could favor more or less restrained killing, depending on the potential conflict between within-host and between-host selection. If dispersal occurs only after host death and the number of susceptible hosts declines with time, then selection could favor early killing even if fewer pathogen propagules are produced.

ABUNDANT GENETIC (AND PERHAPS EPIGENETIC) VARIATION FOR VIRULENCE

Rapid shifts in virulence and the high diversity of plant-infecting microbes suggest that there is ample potential for the microbial social interactions described above to play important roles in plant disease because the timescale of diversity generation is short enough relative to the duration of an infection to contribute to its dynamics through eco-evolutionary processes. It has long been recognized that many pathogens rapidly lose virulence when brought into culture (20, 28, 126), variously termed "phenotypic degeneration, phenotypic instability, phenotypic deterioration, dual phenomenon, saltation and attenuation" (28, p. 213). Bacteria and fungi often carry virulence genes on horizontally transmissible genetic elements, ranging from the Ti plasmid of *A. tumefaciens* (118) and virulence islands across bacterial genomes (63) to various mobile accessory plasmids of *F. oxysporum* (136, 139). In addition, viruses can enhance or diminish virulence. Multiple filamentous bacteriophages infecting the bacterium *R. solanacearum* have been characterized, some of which increase virulence (e.g., ϕRSS1) and others (e.g., ϕRs551) repress virulence; in both cases, the phage modulates the bacterial virulence master regulator *phcA* (149). There are many examples of mycoviruses that reduce fungal pathogen virulence, including *Botrytis cinerea* (67); *Sclerotium rolfsii*, which contains sequences of multiple families of mycovirus (150); and *F. oxysporum* f. sp. *dianthi* (89); conversely, the mycoviruses FcMV1 and FcMV2 both increase the virulence of the pine pitch canker pathogen *Fusarium circinatum*, with consistent effects across 14 fungal strains; this translates into enhanced disease progression on Monterey pine (*Pinus radiata* D. Don) (107). Alternatively, loss of virulence may be due to epigenetic changes that are readily reversible upon exposure to a host (80, 126). Consistent with this hypothesis, bisulfite sequencing of *B. cinerea* after growth in culture compared to after infection of *A. thaliana* showed many epigenetic changes (20).

EVIDENCE FOR COOPERATION AND CHEATING IN PLANT–PATHOGEN INTERACTIONS

Given the potential for cheating at multiple stages of host–pathogen interactions, I review evidence that this cheating occurs within pathogens and examine four case studies: *Pseudomonas savastanoi* pv. *glycinea* with a truncated QS gene; *P. syringae* without the typical type III secretion system (T3SS); and *A. tumefaciens* without the Ti virulence plasmid, as well as *R. solanacearum* virulence modulated by multiple bacteriophages. A more nuanced evolutionary consideration of cheating comes from experimental evolution of the nonpathogenic *P. fluorescens* and is explored in the sidebar titled The Case of *Pseudomonas fluorescens*.

THE CASE OF *PSEUDOMONAS FLUORESCENS*

We can use *Pseudomonas fluorescens*, a nonpathogenic soil bacteria, to arrive at an integrated understanding of microbial populations that acknowledges their individuality while recognizing their sociality. When *P. fluorescens* is grown in static media, wrinkly spreader mutants quickly arise and form a biofilm at the air–broth interface. Upon sustained incubation, cells with a smooth colony morphology arise within the biofilm that do not contribute to the biofilm structure. These cells reduce the buoyancy of the biofilm, decreasing the fitness of the biofilm producers, and have higher fitness when competing head to head with wrinkly spreaders, thus satisfying the definition of cheating (120). Experimental evolution and genetic analysis of this system explored fundamental questions about cooperation and cheating, adaptive diversification, and the underlying molecular mechanisms. Rainey & Kerr (119) then proposed a completely novel interpretation, i.e., that cheaters might be considered a proto-germline because they are able to disperse and give rise to new mats. This highlights the importance of considering the entire life cycle when formulating one's evolutionary intuition, a caution particularly relevant to the study of pathogens because transmission is critical to their fitness yet challenging to measure in the field.

Case Study: *Pseudomonas savastanoi* pv. *glycinea* Lacking Quorum Sensing

Isolates of *P. savastanoi* pv. *glycinea* from Brazil and New Zealand were found to share a point mutation in the QS LuxR homolog ahlR (37). Molecular genetic analysis determined that this QS system regulates phosphate solubilization and swarming motility. Further surveys found that ~50% of isolates from Illinois possessed this mutation, suggesting that QS defective cells may coexist with QS intact cells. This is suggestive circumstantial evidence, but to determine whether these ahlR mutants are cheaters, competition experiments in rhizosphere and in planta that measure the fitness of each strain both alone and in competition are required. In addition, additional genetic data that trace the evolutionary history of this mutation are required to reveal whether the mutation has arisen multiple times in local populations or whether the mutation represents a distinct lineage that may be specializing on a particular host habitat. We also require inoculation experiments that document how this mutation impacts the virulence of the pathogen on its host in relationship to microbial fitness.

Case Study: Avirulent *Agrobacterium tumefaciens*

The α-proteobacterium *A. tumefaciens* can induce crown gall using genes encoded on the Ti plasmid (13, 118). Surprisingly, nonpathogenic *A. tumefaciens* isolates are often isolated from tumors in nature (15), with strains seemingly impossible to recover from apple tumors. Nonpathogenic *A. tumefaciens* strains can also colonize from the environment: Of 5,419 isolates from 12 hosts inoculated under greenhouse conditions, 12% of the recovered isolates were nonpathogenic and only 0.1% of these were mutants of the inoculated strain; the remainder were of unknown origin and presumed from the environment (92). Finally, strains lacking the Ti plasmid arise readily in culture when populations are exposed to virulence gene inducers (51, 116). Some avirulent strains in nature lack Ti plasmids but contain plasmids encoding the ability to utilize the opines produced by tumors (118). Culture-based competition experiments demonstrated that carrying the Ti plasmids does not impose a fitness cost unless nutrients are very limited; however, under conditions in which the encoded virulence genes are expressed, the plasmid-lacking genotype has substantially higher fitness (116). When competition is conducted between Ti+ and Ti− strains with octopine in the media, the Ti+ strain has a fitness advantage (117). A model demonstrates that

strains lacking *vir* genes but able to utilize opines competitively displace virulent strains locally, but virulent strains contribute to larger group sizes and thus are able to persist through the balance of within-group and between-group selection (117). In mature infections, high rates of Ti plasmid conjugation are triggered by conjugal opines, which could further enhance the fitness of the plasmid (13).

Case Study: *Pseudomonas syringae* with Xenologous Type III Secretion System

P. syringae was voted the most important bacterial plant pathogen in 2012, as it is both a species of high economic importance and a model for fundamental aspects of plant pathology (98). However, Mansfield et al. (98) note, "It seems a little unfair that a team of pathovars has been voted for an award, a bit like a relay team winning the 400-m individual Olympic gold medal." The T3SS first characterized in *P. syringae* plays a crucial role in virulence on its many hosts (111), but in 2008 a subclade of avirulent strains isolated from diverse hosts and locations was described in which all strains possess an atypical T3SS (105). Additional surveys found that this clade 2c of *P. syringae* is common on healthy leaves and grows as a leaf epiphyte to densities as high or higher than virulent strains (33) but has reduced fitness as a leaf endophyte (84). The critical test of whether these strains are cheaters was the competition assays between multiple clade 2c isolates originating from wild *A. thaliana* across the Midwest and virulent *P. syringae* strains. When inoculated alone, the virulent strains had higher fitness in terms of viable colony forming units than the avirulent strains, but when coinoculated the avirulent strain had equal or higher relative fitness than the virulent strain and caused a fitness reduction of the virulent strain (11). Knocking out the atypical T3SS had no effect on growth in planta but did reduce fitness in low-nutrient conditions (11), suggesting that this clade is specialized in growth in oligotrophic environments in addition to opportunistically cheating plant-pathogenic clades. It is intriguing that these strains co-occur widely in nature, and future work is needed to reveal whether these cheaters impact diseases of crops caused by *P. syringae* or whether they could be harnessed as biocontrol agents. A parallel case of cheating was documented in the animal pathogen *P. aeruginosa*, wherein T3SS-negative strains are often isolated from human patients; in mixed infections in a mouse model, these nonsecreting strains had higher fitness that depended on the co-occurring T3SS-positive strain producing an effector (35).

Case Study: *Ralstonia solanacearum* and Phage-Modulated Virulence

R. solanacearum causes vascular wilt of multiple plants and its virulence regulation depends on a unique QS signal. At low population density, *R. solanacearum* expresses genes for adhesion and rapid growth, whereas at high population density it undergoes physiological shifts under the control of the master regulatory gene *phcA* to utilize only specific carbon sources and produce EPSs and other virulence factors (95). A genome-scale metabolic reconstruction analyzed with constraint-based and metabolic flux analysis encompassing 365 biochemical and transport reactions and 174 environmental exchange reactions found a trade-off between growth and EPS production. This led to the prediction of higher fitness for the *phcA* regulatory mutant, which was confirmed by in vitro competition experiments (115). The timing of this shift could represent a social dilemma because virulence is costly and cells that remain in the rapidly growing state for longer could potentially be cheaters. Intriguingly, multiple filamentous bacteriophages contain genes that repress *phcA* and therefore reduce virulence (1, 2). Competition experiments in planta demonstrate that in the case of ϕRs551, infection with a phage possessing the *phcA*-repressing gene increases bacterial fitness (2)—presumably by failing to contribute to the public good of EPSs.

What is currently not known is whether this reduces the total fitness of the community and/or transmission rates and thus represents a case in which within-host and between-host selection are in conflict. For the vector-borne vascular pathogen *X. fastidiosa*, the exact opposite scenario occurs wherein mutants defective in QS rapidly outcompete the wild type (31). It is unclear why this type of signal-insensitive strain does not dominate in wild populations; social evolution theory predicts that very strong between-host selection would be required for the overall transmission to be higher for prudent strains, such as when vectors strongly prefer hosts with lower bacterial loads.

COMMUNITY-LEVEL SOCIAL INTERACTIONS

As shown in the case studies above, social interactions within closely related pathogens can occur through diverse mechanisms. The commonality is the production and consumption of public goods, which can give rise to a tragedy of the commons scenario in which cheating can occur. Cooperation and cheating can occur readily between species (10) and plant pathogens may engage in these community-level social dilemmas as well as competitive and antagonistic interactions (for a broad consideration of the evolutionary dynamics of these systems, see 3). There is ample evidence for polymicrobial infections of plants in nature being the rule rather than the exception, with examples of both multiple types of virus, bacteria, or fungi and cross-kingdom coinfection (49, 86, 134). However, there are no clear predictions for how coinfection impacts the overall virulence to the host, and examples range widely from decreased to increased disease progression (134). In several cases, coinfection by a biotroph and a necrotroph can be synergistic, such as when one pathogen induces SA signaling and thereby depresses JA signaling through cross talk, which results in systemic induced susceptibility (12).

In one case, shared QS signals and coordinated metabolism were implicated in a disease consortium. Olive-knot disease results from the agent *P. savastanoi* pv. *savastanoi*, but the disease causes greater harm to the plant host when *Erwinia toletana* and *Pantoea agglomerans* co-occur (29, 74). All three bacteria produce similar QS AHLs and although an AHL nonproducing mutant of *P. savastanoi* pv. *savastanoi* is unable to cause disease alone, the presence of the other two bacteria can restore virulence (74). Furthermore, QS in *E. toletana* regulates an aldolase-encoding gene that is required for knot development and colocalization of the bacteria in planta (29). This intricate system demonstrates that cooperative social interactions between species can enhance disease; however, given that AHLs can be public goods, this system may be vulnerable to cheaters.

SUBVERTING OR LEVERAGING SOCIAL INTERACTIONS TO MANAGE PLANT DISEASE

Understanding the mechanisms that pathogens use to infect plants provides novel targets and strategies for managing disease, with QS attracting a lot of attention (87) because it acts so early in the interaction; circumventing the QS systems that pathogens use to induce virulence genes renders them just another commensal (or even beneficial) member of the microbiome. Some bacterial biocontrol strains secrete enzymes that degrade QS signals, a phenomenon known as quorum quenching. For example, reporter constructs demonstrated that *Rhodococcus erythropolis* is able to perform quorum quenching of the soft-rot agent *Pectobacterium atrosepticum* (30). With recent advances in sequencing technology, we do not even need to be able to culture the organisms carrying these protective systems, as demonstrated by a study that mined soil metagenome data for novel esterases and identified some that are able to degrade 3-OH PAME and inhibit EPS production in vitro by *R. solanacearum* (88). Plants can also produce quorum-quenching molecules,

and many synthetic compounds with quorum-quenching activity have been described (79). An active field of research is the identification and characterization of synthetic QS modulators (55). For example, some synthetic AHLs show activity preventing virulence in *P. carotovora* on potato and *P. syringae* B278A on bean (*Phaseolus vulgaris*) (113), and metal nanoparticles can inhibit QS in multiple gram-negative bacteria (70).

Although there are many examples of biocontrol between pairs of phylogenetically diverse microbes, some of the most effective biocontrol agents are close relatives of pathogens such as the cheaters described above. This could be due to high ecological similarity generating intense competition as well as the fact that toxins are often highly effective against close relatives; many toxins are encoded by mobile genetic elements that also encode resistance to the toxin, and these toxin–antitoxin systems can act to prevent loss or disruption of the genetic element. Two well-studied examples illustrate pairwise biocontrol by relatives. The nonpathogenic *F. oxysporum* strain Fo47 grows faster than the pathogenic strain *F. oxysporum* f. sp. *lycopersici* and has a competitive advantage in mixed inoculation of roots; Fo47 also germinates faster than the pathogenic strain *F. oxysporum* f. sp. *radicis-lycopersici* and has a competitive advantage on roots (103). The avirulent strain *Agrobacterium radiobacter* K84 serves as an effective form of biocontrol of *A. tumefaciens* because, in addition to competitively colonizing plant hosts and consuming opines, it also produces the antimicrobial agrocin (118). Work in the 1990s showed that this biocontrol system has the risk of horizontal transfer turning the biocontrol strain into a pathogen or the pathogen into an agrocin producer; this resulted in genetic engineering of a biocontrol strain derived from K84 that lacks conjugation ability and is widely used (118). In the first case, the biocontrol strain Fo47 could be a cheater if it benefits from the presence of the pathogen; in the second case, the biocontrol strain K84 is likely a cheater, assuming that it has fitness outcomes as described above in the *A. tumefaciens* case study.

Streptomyces bacteria are some of the most prolific antibiotic producers known, and many play a role in communities that create disease-suppressive soils that do not permit infection of host plants by pathogenic streptomycetes (83, 129). *Streptomyces* isolates responsible for suppression of disease-causing *Streptomyces* are promoted by high resource competition under monoculture, leading to interference competition (129), and *Streptomyces* communities under crop monocultures are more antagonistic to pathogens than *Streptomyces* communities that occur under diverse plant communities (9). Characterizing the resource niches in vitro showed that strains that invest in antibiotic resistance grow more slowly and use a narrower range of resources than more sensitive strains (130). The network structure of inhibition varies by the location where the strains are sampled (130). Because antibiotic production (and often detoxification) are public goods, cheating is predicted to play an important role in these communities; this would appear as strains that detoxify but do not produce antibiotics being derived evolutionarily from producing strains. However, modeling shows that an antibiotic-degrading strategy can coexist with antibiotic-producing and antibiotic-sensitive strategies even in well-mixed environments, provided that at least two antibiotics are involved (82).

SUMMARY POINTS

1. Many stages of infection by plant pathogens involve the production and/or consumption of public goods and thus provide opportunities for social dilemmas and cheating.
2. In most theoretical models, cheating does not completely displace cooperating and multiple strategies can coexist.

3. Plant pathogens have multiple genetic mechanisms that rapidly generate variation in virulence phenotypes; thus, if some of these phenotypes are able to cheat, they could be common.
4. Consideration of several case studies shows clear evidence for cheating by avirulent *A. tumefaciens* lacking Ti plasmids and avirulent *P. syringae* with atypical T3SSs.
5. Competition experiments, preferably in planta, are required to determine whether cheating is occurring, yet these are rarely conducted.
6. Biocontrol strains are often closely related to pathogens and may represent cheaters that exploit the pathogen.

FUTURE ISSUES

1. Culture-independent work and advances in metabolomics will enable us to search for cases of coincidental virulence in plant pathogens.
2. Social behaviors of plant pathogens, particularly QS, will continue to be exciting targets for managing disease.
3. Advances in the molecular bases of virulence and plant–microbe interactions will continue to yield tools that can be harnessed for agriculture, such as phages that alter virulence.
4. Experimental work with culture collections will continue to be critical in dissecting the basis and implications of variation in microbial fitness.
5. Emerging technologies to monitor microbial physiology and measure microbial fitness in the field will enable us to better understand their eco-evolutionary dynamics.
6. Multi-omics tools will enable us to tease apart the complex social interactions within plant microbiomes and allow us to predict how cooperation and cheating will impact plant disease.

DISCLOSURE STATEMENT

The author is 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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