Allelopathy as an evolutionary game

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Abstract
In plants, most competition is resource competition, where one plant simply pre-empts the resources away from its neighbors. Interference competition, as the name implies, is a form of direct interference to prevent resource access. Interference competition is common among animals that can physically fight, but in plants, one of the main mechanisms of interference competition is allelopathy. Allelopathic plants release cytotoxic chemicals into the environment which can increase their ability to compete with surrounding organisms for limited resources. The circumstances and conditions favoring the development and maintenance of allelochemicals, however, are not well understood. Particularly, despite the obvious benefits of allelopathy, current data suggest it seems to have only rarely evolved. To gain insight into the cost and benefit of allelopathy, we have developed a 2×2 matrix game to model the interaction between plants that produce allelochemicals and plants that do not. Production of an allelochemical introduces novel cost associated with both synthesis and detoxifying a toxic chemical but may also convey a competitive advantage. A plant that does not produce an allelochemical will suffer the cost of encountering one. Our model predicts three cases in which the evolutionarily stable strategies are different. In the first, the nonallelopathic plant is a stronger competitor, and not producing allelochemicals is the evolutionarily stable strategy. In the second, the allelopathic plant is the better competitor, and production of allelochemicals is the more beneficial strategy. In the last case, neither is the evolutionarily stable strategy. Instead, there are alternating stable states, depending on whether the allelopathic or nonallelopathic plant arrived first. The generated model reveals circumstances leading to the evolution of allelochemicals and sheds light on utilizing allelochemicals as part of weed management strategies. In particular, the wide region of alternative stable states in most parameterizations, combined with the fact that the absence of allelopathy is likely the ancestral state, provides an elegant answer to the question of why allelopathy seems to rarely evolve despite its obvious benefits. Allelopathic plants can indeed outcompete nonallelopathic plants, but this benefit is simply not great enough to allow them to go to fixation and spread through the population. Thus, most populations would remain purely nonallelopathic.
1 | INTRODUCTION

Competition is ubiquitous in the natural world, as there are finite resources available in a given time and space (Connell, 1983; Fowler, 1986; Schoener, 1983). Thus, competition generally reduces plant fitness when resources, such as light, space, water, and nutrients, are limiting (Friedman, 1971; Wilson, 1988). This type of competition for finite resources is broadly named resource competition and occurs when organisms compete by simply reducing the availability of resources to other organisms (Carothers & Jakić, 1984). Alternatively, interference competition occurs when one organism interferes with, and therefore reduces, the ability of the other to obtain a shared resource while not necessarily drawing down resource concentrations (Carothers & Jakić, 1984). Animals routinely face interference competition as they can physically fight over resources (Ford, 1979). Sessile plants primarily compete via resource competition. However, one of the major mechanisms of interference competition in plants is mediated chemically through allelopathy (Fuerst & Putnam, 1983). For example, one of the best documented examples is allelopathy by walnut trees (Juglans spp.), mediated by the allelochemical juglone, which is toxic to a variety of crop and horticultural species, including corn (Zea mays L.) and soybean (Glycine max [L.] Merr.) (Jose & Gillespie, 1998) and tomato (Solanum lycopersicum L.) and cucumber (Cucumis sativus L.) (Wilcove et al., 1998).

Allelopathy is the production of chemicals, called allelochemicals, that are released into the environment and negatively affect the growth and development of competing individuals (Latif et al., 2017). Although the term was first used in 1937, the effect has been recognized for thousands of years (Latif et al., 2017). A recent meta-analysis found that allelopathy reduced plant productivity by 25% across study conditions but further emphasized the influence of study design on the measured impact of allelopathy (Zhang et al., 2021), a large concern considering the difficulties in studying the competitive effects of allelopathy because of methodological difficulties. For example, for many years, experiments used soil additives such as activated charcoal that were thought to prevent the activity of allelochemicals with the goal of comparing how plants grew either with or without the presence of this form of interference competition. Unfortunately, it was later learned that activated charcoal also stimulates nutrient availability, and thus, many years of research showing the negative effects of allelochemicals were probably just detecting the positive effects of fertilization (see Inderjit & Callaway, 2003; Lau et al., 2008).

Despite limitations in the ability to experimentally study allelopathy, it has been implicated in the success of some invasive plants, highlighting the advantage of interference competition as a strategy (Callaway & Aschehoug, 2000). Invasion by nonnative species is ranked the second strongest risk to natural diversity (Keane & Crawley, 2002), and approximately half of 524 invasive species studied were found to be allelopathic (Kalisz et al., 2021). For example, Paterson’s curse (Echium plantagineum L.) is an invasive weed in Australia, affecting up to 30 Mha, whose invasion success is partially attributed to production of the allelochemical shikonin and its derivatives (Zhu et al., 2016). Indeed, one commonly invoked mechanism for invasion by nonnative species is the novel weapons hypothesis, which suggests invasive species are successful through use of competitive strategies for which native species have not coevolved counter strategies (Callaway & Ridenour, 2004; Prati & Bossdorf, 2004). This mechanism has been linked to the invasion success of allelopathic Policeman’s helmet (Impatiens glandulifera) (Ruckli, Hesse, et al., 2014), which releases a compound structurally similar to shikonin called 2-methoxy-1,4-naphthoquinone (2-MNQ) that elicits negative effects on herb germination and mycelium growth and is otherwise absent in soils without l. glandulifera, thus suggesting 2-MNQ may function as a “novel weapon” (Gruntman et al., 2014; Ruckli, Hesse, et al., 2014; Ruckli, Rusterholz, & Baur, 2014). Indeed, Zhang et al. (2021) found that the negative effect from a naturalized, allelopathic species was stronger on native plants than the negative effect from a native allelopathic species on other native species. From these studies, it may be possible that allelochemicals may have significant potential for genetically modified cropping systems to enhance the competitive ability of crop species over weeds.

Despite the potential advantages of allelochemicals as an evolved tool for interference competition—as evidenced by the predominance of invasive plants to be allelopathic (Kalisz et al., 2021)—production of allelochemicals seems to have rarely evolved in plants. Here, we report an evolutionary game theoretic model to probe the benefits and circumstances that might favor the evolution of allelochemicals to better understand why they might not be more common in plants. The advantage of the evolutionary modeling approach is that we can identify conditions where allelopathy might evolve or might not evolve. That is, the evolutionary approach does not take for granted that allelopathy should exist. Specifically, we ask: (1) What circumstances favor the production of allelochemicals? (2) How does the cost of producing an allelochemical affect fitness of the plant producing the allelochemical and plants competing with that plant? (3) When will allelopathic plants be stable in a population? Beyond the implications for evolutionary ecology, understanding the evolution of allelopathy has the potential to inform the design of applications for agriculture, from the integration of allelopathic crops into farming systems to the use of synthetic biology to create a crop that produces its own allelochemical-based weed control.

KEYWORDS
allelopathy, evolutionarily stable strategy, game theory, modeling
2 | MATERIALS AND METHODS

2.1 | Model development

We developed a $2 \times 2$ matrix game of interactions among a plant player with (+$A$) and without (–$A$) allelopathy. We assumed that competition creates benefits of available resources ($B$), that the cost ($C$) to the player of producing allelochemicals is the sum of the costs of production of the allelochemical and detoxification to prevent autotoxicity, and that allelochemicals impose some different cost to the opponent in the form of toxicity and/or detoxification ($T$). We further assumed that benefits were shared unequally, encompassed by a parameter, $a$, that represents the proportion of benefits the allelopathic plant receives when competing with a nonallelopathic plant. These parameters of the model should adhere to the following: $0 < B, C, T$ and $1/2 < a \leq 1$, $0 \leq p \leq 1$ where $p$ is the proportion of allelopathic plants in the population. We found this four-parameter model to be the simplest possible model that generates the evolution of allelopathy in ways that seem true to nature, though we describe two possible simpler alternatives in the Supporting Information that explore how the parameters $a$ and $T$ individually shape model solutions. We understand the limitations imposed by the simplicity of the model, but the four, simple parameters encompass complex, multifaceted biological possibilities, and the simplicity allows us to ask large-scale questions about the ecology and evolution of allelopathy.

Combining these parameters, we can derive the payoff, $G_{uv}$, across several competitive contexts, where $v$ is the focal plant strategy (+$A$ or –$A$) and $u$ is the neighboring plant strategy (+$A$ or –$A$). Finally, we also assume that there are two plants competing in something like a pot experiment, because we imagine this is the most likely way to empirically test our model in the future (e.g., Inderjit & Callaway, 2003; Lau et al., 2008). However, the equations below can be extended to any number of competing plants by simply replacing 2 with $N$, where $N$ is the number of competing plants.

First, when both plants produce allelochemicals, we argue that they will, on average, share the total benefit of the soil volume equally, $B/2$, but will also pay the cost of producing and detoxifying allelochemicals, $C$. Thus, the fitness payoff to a plant in a population of pure +$A$ plants is

$$G_{+A,+A} = \frac{B}{2} - C. \quad (1)$$

Second, in a mixed population of +$A$ and –$A$ plants, the +$A$ plant will pay the cost $C$ but will share the benefits $B$ differently. Instead of equally sharing the benefits, the player will get a proportion of benefits, $a$, that takes into account the competitive advantage of production of allelochemicals according to

$$G_{+A,-A} = aB - C. \quad (2)$$

Inversely, in the mixed population, the –$A$ plant receives when competing with a nonallelopathic plant. We developed a $2 \times 2$ matrix game of interactions among a plant player with (+$A$) and without (–$A$) allelopathy. We assumed that competition creates benefits of available resources ($B$), that the cost ($C$) to the player of producing allelochemicals is the sum of the costs of production of the allelochemical and detoxification to prevent autotoxicity, and that allelochemicals impose some different cost to the opponent in the form of toxicity and/or detoxification ($T$). We further assumed that benefits were shared unequally, encompassed by a parameter, $a$, that represents the proportion of benefits the allelopathic plant receives when competing with a nonallelopathic plant. These parameters of the model should adhere to the following: $0 < B, C, T$ and $1/2 < a \leq 1$, $0 \leq p \leq 1$ where $p$ is the proportion of allelopathic plants in the population. We found this four-parameter model to be the simplest possible model that generates the evolution of allelopathy in ways that seem true to nature, though we describe two possible simpler alternatives in the Supporting Information that explore how the parameters $a$ and $T$ individually shape model solutions. We understand the limitations imposed by the simplicity of the model, but the four, simple parameters encompass complex, multifaceted biological possibilities, and the simplicity allows us to ask large-scale questions about the ecology and evolution of allelopathy.

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Finally, in a pure population of –$A$ plants, because no plant produces allelochemicals, they merely share the benefits as $B/2$ and have no costs associated with allelochemicals, according to

$$G_{-A,-A} = \frac{B}{2}. \quad (4)$$

Combined, Equations 1–4 yield the payoff matrix shown as Figure 1.

2.2 | ESS definition

In a matrix game, an evolutionarily stable strategy (ESS) is identical to a Nash equilibrium where a participant cannot gain by changing strategy if the other participant’s strategy does not change (Apaloo et al., 2014; Maynard Smith & Price, 1973). Thus, a pure ESS is defined as the strategy that once adopted by members of a population cannot be invaded by any alternative strategy. Mixed ESSs are also permissible where multiple strategies either ecologically coexist through evolutionary time or form noncoexisting alternative stable states (sometimes also called priority effects). Here, in a $2 \times 2$ matrix game, if $G_{uv}$ is the fitness payoff of a focal plant species using strategy $v$ against a competing plant species using strategy $u$ such that $v \neq u$, then $v$ is a pure ESS if and only if $G_{uv} > G_{vu}$ and $G_{wu} > G_{uw}$. Alternatively, $u$ is a pure ESS when $G_{uv} > G_{vu}$ and $G_{wu} > G_{uw}$ (i.e., under the opposite inequalities). Most interestingly, under this definition, mixed ESS solutions are possible where the two strategies may coexist or for a system of alternative stable states (Maynard Smith, 1982). A mixed ESS occurs when $G_{uv} > G_{vu}$ and $G_{wu} > G_{uw}$. Alternative stable states occur when: $G_{uv} > G_{wu}$ and $G_{wu} > G_{vu}$. Together, a keen

\begin{center}
\begin{tabular}{|c|c|c|}
\hline
\textbf{Player} & \textbf{+A} & \textbf{–A} \\
\hline
\textbf{+A} & $\frac{B}{2} - C$ & $aB - C$ \\
\hline
\textbf{–A} & $(1 - a)B - T$ & $\frac{B}{2}$ \\
\hline
\end{tabular}
\end{center}

\textbf{FIGURE 1} Symmetric payoff matrix for competition between plants that either produce allelochemicals (+$A$) or not (–$A$). See text for parameter definitions.
observer will note that these four inequalities form all possible pairs of inequalities within each column of a $2 \times 2$ payoff matrix as drawn here (Figure 1).

3 | RESULTS

3.1 | Pure evolutionarily stable strategies

For $+A$ to be a pure ESS, $+A$ needs to be able to (i) invade a population of $-A$ and (ii) resist invasion from $-A$. According to the ESS definition, this occurs when

$$aB - C > \frac{B}{2} \text{ and } C > B(1 - a) - T.$$  \hfill (5a)

Equation 5a can be rearranged into isoclines in $B$ and $C$ space to find

$$B > \frac{2C}{2a - 1} \text{ and } B > \frac{2(C - T)}{2a - 1}.$$  \hfill (5b)

Alternatively, for $-A$ to be ESS, $-A$ needs to be able to (i) invade a population of $+A$ and (ii) resist invasion from $+A$.

$$\frac{B}{2} > aB - C \text{ and } (1 - a)B - T > \frac{B}{2} - C.$$  \hfill (6a)

Equations 6a can be rearranged to find

$$B < \frac{2C}{2a - 1} \text{ and } B < \frac{2(C - T)}{2a - 1}.$$  \hfill (6b)

Notice that Equations (5) and (6) are simply opposite inequalities.

3.2 | Mixed evolutionarily stable strategies

The mixed strategy, where there are alternating stable states such that either $-A$ or $+A$ can resist invasion from the other strategy occurs when

$$\frac{B}{2} < aB - C \text{ and } (1 - a)B - T > \frac{B}{2} - C.$$  \hfill (7a)

Equations 7a can be rearranged to find

$$B < \frac{2C}{2a - 1} \text{ and } B > \frac{2(C - T)}{2a - 1}.$$  \hfill (7b)

The isoclines in Equations (5)–(7) create two parallel lines, each with slope $\frac{1}{2}$, but that either intercept the $y$ axis at 0 or at $\frac{2T}{2a - 1}$. Thus, depending on the values of $a$ and $T$, we can plot the entire solution space graphically in positive $B$ and $C$ phase space (Figure 2). For $+A$ to be the ESS, the parameters need to be above both isolines. For $-A$ to be the ESS, the parameters need to be below both isolines. Between the two lines, which will never cross as they have the same slope, there is a region of alternative stable states, also sometimes called a priority effect. In the region of alternative stable states, either
strategy might occur, but the answer depends on the history of the system. That is, whichever strategy was there first becomes the ESS.

In our model, coexistence is never possible. For it to be so, would require

\[ \frac{2C}{2a - 1} < B < \frac{2(C - T)}{2a - 1}. \]  

(8)

Because \( T > 0 \) by definition, these conditions can never be met. This suggests that within a population, all plants of a species will either produce or not produce allelochemicals.

Allelopathic plants gain a competitive advantage only when \( B > \frac{2C}{2a - 1} \), which can offset the cost of producing allelochemicals beyond just the cost of toxicity on the neighboring plant (Figure 2). However, if \( B < \frac{2(C - T)}{2a - 1} \), then nonallelopathic \( \rightarrow A \) plants gain the competitive advantage because the benefits of allelopathy do not outweigh the costs to the allelopathic plant, or the allelopathic chemical is simply not toxic enough to generate a benefit (i.e., low \( T \)). This region of pure \( \rightarrow A \) as the ESS expands as \( a \) increases (Figure 2). When \( a > 0.5 \), but \( \frac{2C}{2a - 1} < B < \frac{2(C - T)}{2a - 1} \), there is an interesting region between the two isolines of alternative stable states where only one strategy can exist at a time, but which one occurs depends on the initial conditions (i.e., on this history of colonization and/or mutation). This area of alternative stable states also expands with increasing \( T \) but decreasing \( a \).

4 | DISCUSSION

In this study, we developed and analyzed a model of the evolution of allelopathy between two competing plants as an evolutionary game to examine the conditions under which the production and deployment of allelochemicals becomes a favorable competitive strategy. The model has four simple parameters that describe costs and benefits among players. Somewhat intuitively, allelopathy can only evolve when the benefits to the allelopathic plant outweigh the costs, but the model outlines these precise conditions in four-dimensional phase space (Figure 2). For example, in the case of an extremely toxic allelochemical (i.e., large \( T \)), that also happens to be metabolically costly to produce (i.e., large \( C \)), the model makes it clear that there must be relatively high benefits (e.g., high \( B \), very fertile environments) and confer a very large competitive advantage (large \( a \)). Indeed, except where \( a \) approaches 1 and \( T \) is large, we see large regions of alternative stable states, and relatively small regions where \( \rightarrow A \) is the pure ESS. Assuming that \( \rightarrow A \) is the ancestral condition, we argue that this might explain why allelopathy has been relatively rare to evolve, despite the obvious advantage. That is, in the region of alternative stable states, any \( \rightarrow A \) mutants would simply not be able to invade the ancestral \( \rightarrow A \) population because of their priority effect advantage. The relative rarity of allelopathy in nature might indicate natural environments found on this planet exist closer to the upper left region of Figure 2, though future work should investigate whether the biochemical cost of production and the cost of detoxification are substantial energetic costs to plants to narrow down the region of parameter space that exists in natural plant communities. There may be some biochemical constraints that place the plant kingdom in this part of the phase space, and this would be an important area for plant biologists to explore further. It is also possible that allelopathy is more common than current knowledge suggests.

One way of reducing the cost of producing novel allelochemicals, \( C \), is to harness existing metabolic frameworks. Many species producing naphthoquinone-based compounds, for example, have independently evolved to do so from 1,4-dihydroxy-2-naphthoic acid (DHNA), an intermediate of the phylloquinone (vitamin \( K_1 \)) pathway (Meyer et al., 2020; Widhalm & Rhodes, 2016). Examples include juglone in black walnut trees (McCoy et al., 2018), lawsone and 2-MNQ in the Balsaminaceae (e.g., \textit{Impatiens} species) (Zenk & Leistner, 1967), lawsone and lapachol in the Bignoniaceae (Hussain et al., 2007), anthraquinones like alizarin made by Rubiaceae species (Yamazaki et al., 2013), and anthracesasmoned by sesame (\textit{Sesamum indicum}, Pedaliaceae) (Furumoto & Hoshikuma, 2011). Interestingly, juglone, lawsone, and 2-MNQ are all implicated as allelochemicals (Block et al., 2019; Dana & Lerner, 2001; Ruckli, Hesse, et al., 2014). This indicates that DHNA derived from the phylloquinone pathway, which is present in all plants, likely provides a lower cost path for plants to synthesize allelochemicals.

Over time, the cost of allelochemical toxicity, \( T \), to \( \rightarrow A \) plants could be mitigated by evolution of mechanisms to tolerate or detoxify the allelochemical. Therefore, the competitive disadvantage of not producing the allelochemical to \( \rightarrow A \) plants would dissipate; however, the cost of detoxification, which is also part of \( T \), would likely remain nonzero. We hypothesize that the evolution of \( T \) can draw inferences from evolution of herbicide resistances in plants, which occur via mutations in herbicide target sites (target-site resistance) or non-target sites (non-target-site resistance) (Gaines et al., 2020). In an analogous scenario of non-target-site resistance, the allelochemical itself or the toxicity arising from the allelochemical could be metabolically counteracted through biochemical modification and/or compartmentalization of the allelochemical or its modified product. Thus, non-target-site resistance is referred to as “metabolism-based resistance” (Hatzios, 2004). Metabolism-based resistance to herbicides is primarily achieved via four gene families: cytochrome P450 monoxygenases, glutathione transferases (GSTs), glycosyltransferases, and/or ABC transporters (Yuan et al., 2007). It is likely that plants that evolve in proximity to an allelopathic plant use similar methods to tolerate allelochemicals. GSTs function by covalently linking glutathione (GSH) with compounds that are hydrophobic and electrophilic (Cummins et al., 2011); some also function as carriers that transport GSH conjugates to vacuoles for detoxification (Sun et al., 2012). Black-grass (\textit{Alopecurus myosuroides}) is a weed species that has evolved resistance to multiple herbicides by over expressing a single GST, AmGSTF1. Heterologous overexpression AmGSTF1 in \textit{Arabidopsis thaliana} was shown to be sufficient to confer resistance to multiple herbicides (Cummins et al., 2013). Moreover, \textit{Arabidopsis} seedlings grown in vitro in the presence of GSH in juglone-containing media were found to display root growth phenotypes...
indistinguishable from wild type (Meyer et al., 2020). Beyond conjugation with GSH, glycosylation appears to be a major mechanism of detoxification of specialized metabolites (le Roy et al., 2016). Indeed, much of the juglone found in black walnut is glycosylated (Müller & Leistner, 1978), suggesting that one of the mechanisms black walnut uses to tolerate producing and storing an autotoxic compound is through glycosylation. Reduced uptake or increased export could also confer some tolerance to allelopathic exposure. Mutations in transport proteins have been shown to confer resistance to herbicides through decreased uptake (reviewed in Conte & Lloyd, 2011). Additionally, fungi and bacteria have been shown to be able to degrade structurally diverse, toxic chemicals from a variety of plant families (Pedras & Ahiahonu, 2005; Rettenmaier et al., 1983; Yu et al., 2019). Studies from microorganisms may provide more insight into mechanisms plants use to tolerate allelochemicals or provide guidance for transgenic strategies to convey resistance to allelochemicals.

### 4.1 Model assumptions and caveats

As ever, any model comes with some caveats. One large caveat is factors that are not included in our model can affect allelopathy. For example, in natural environments, allelopathy is affected by the ecology of the soil (Cipollini et al., 2012). Pseudomonas J1, a soil bacteria isolated from soil surrounding a black walnut, is capable of growing on soil bacteria have been shown to be able to degrade radish in sterile soil (Helsey, 1996). These and other studies show that degradation of allelochemicals by soil microbes is a factor in the toxicity of an allelochemical in a given environment. Such degradation would lead to a decrease in T, indicating that the parameter T for the same compound could be different in different soils and ecosystems. Similarly, the soil microbiome has been shown to have diverse effects on plant fitness (reviewed in Lakshmanan et al., 2014). In addition to directly harming nearby plants, allelopathy may also play a role in altering the microbial soil community to the benefit of the allelopathic plant. A recent study found that extracts of invasive, allelopathic Rhus typhina not only inhibited growth of marigold (Tagetes erecta) but also decreased carbon utilization and diversity of its surrounding soil microbiota, suggesting an interplay between direct and indirect allelopathic effects (Qu et al., 2021). Another example of a factor absent from our model is how allelopathy interacts with other plant interactions. For example, invasive garlic mustard (Alliaria petiolata) (M.Bieb.) Cavara & Grande has been shown to inhibit the interaction between seedlings of competitors and their mutualistic fungi (Stinson et al., 2006). Similarly, l. glandulifera invasion disrupts symbiotic associations between arbuscular mycorrhiza and native saplings (Ruckli, Rusterholz, & Baur, 2014), likely via the release of 2-MNQ, which was also shown to inhibit mycelium growth of ectomycorrhiza fungi (Ruckli, Hesse, et al., 2014). Conversely, some studies have suggested that the plant microbiome reduces the effect of allelochemicals on the plant (Mishra et al., 2013), in effect lowering the cost of toxicity/detoxification, T, to opponents. Paxillus involutus, a mycorrhizal fungi of black spruce (Picea mariana), has been shown to be able to degrade allelopathic compounds produced by Kalmia angustifolia, perhaps conveying some tolerance to black spruce (Zeng & Mallik, 2006). These examples demonstrate the complexity of studying allelopathy in field conditions that our model does not capture.

### 4.2 Implications and applications of the model

Investigating the means by which plants reduce cost and increase fitness in the presence of allelochemicals will allow more predictable integration of allelopathy as part of weed management strategies in cropping systems. For example, intercropping is a common agricultural practice used in many parts of the world to improve land use efficiency, to mitigate the risk of a single crop failing, and to diversify farming income. Often, intercropping involves cocultivation of two or more cash crops, but in some cases, a cash crop is grown alongside a noncash crop to provide benefits, such as weed suppression, to the primary crop (Mohler & Stoner, 2009). In either case, intercropped species are grown in close enough proximity to allow biological interaction. Therefore, the allelopathic potential of each species should be considered when designing mixed cropping systems (Cheng & Cheng, 2015). For example, a study by Iqbal et al. (2007) showed that intercropping cotton (Gossypium hirsutum L., cv FH901) with allelopathic crops, including sorghum (Sorghum bicolor L), soybean, or sesame, was an effective strategy to control purple nutsedge (Cyperus rotundus L.), a common aggressive weed found in parts of South Asia. According to the matrix game presented here, the fitness payoff to both cotton and purple nutsedge (the -A species) would be expected to decrease as the toxicity, T, of allelochemicals produced by sorghum, soybean, or sesame (the +A species) increased. Indeed, seed cotton yield was found to decrease between 8% and 23% in all intercropping systems, compared with unmanaged cotton alone. Similarly, the presence of allelopathic species led to 70% to 96% reduced purple nutseed density (Iqbal et al., 2007). That control of purple nutsedge was found to be more effective in the second year of the study compared with the first year, which was suggested to be the result of residual allelochemicals leftover in the soil in year two (Iqbal et al., 2007). This is consistent with purple nutsedge paying an increased penalty, T, to detoxify higher levels of allelochemicals. Herbicide applications have increased over the last 25 years in many major cropping systems (Kniss, 2017). With this trend, so too has the number of weeds that have developed resistance to commonly used pesticides (Heap, 2020). To address the lack of new herbicidal modes of action needed to combat resistant weeds (Dayan et al., 2012), allelochemicals, which offer a wide diversity of new chemical structures, have been suggested as sources for developing novel herbicides (Cantrell et al., 2012; Macias et al., 2019). One attractive strategy is to engineer or breed production of allelochemicals into nonallelopathic cash crops, although autotoxity and the metabolic cost of biosynthesis must remain low enough to not significantly impact agricultural performance (Duke, 2003). If the cost, C, to the crop engineered to be +A is
too high, then it would not be an ESS and could be invaded by $-A$ species (i.e., weeds) (Equations 5a and 5b). At the same time, if the cost, C, to the $+A$ crop is too low, then it may allow it to become too easily capable of escaping and invading native populations of $-A$ species (Figure 2). If the crop were engineered to produce and detoxify an allelochemical such that it was in the realm of alternative stable states, the allelopathic crop would be able to resist invasion from $-A$ weeds, without the possibility of escape and invasion. Conversely, by purposefully engineering a less fit crop to fall outside the region where $+A$ is ESS (Figure 2), it would also provide a mechanism by which to prevent escape of the transgenic species. Such application could be useful in cover cropping where certain cover crops that are not controlled prior to planting cash crops can become weeds.

Finally, another interesting consideration in the evolution of allelopathy is the presence of allelobiosis. Allelobiosis is a relatively new term that describes communication between plants via nontoxic compounds (Ninkovic et al., 2010). For example, planting tomato (Lycopersicon esculentum) in proximity with sagebrush (Artemisia tridentata) resulted in increased production of proteinase inhibitors in tomato due to methyl jasmonate released by the sagebrush (Farmer & Ryan, 1990). Though allelobiosis has been most often demonstrated with volatile compounds, there are examples of this kind of plant–plant communication through the rhizosphere. Indeed, Li et al. (2016) found that allelobiosis and allelopathy coexist in interactions of weeds with allelopathic wheat. Root exudates from weed species were sufficient to induce allelopathy in the wheat, suggesting a chemical signal sensed by the wheat. Further work is necessary to detangle the effects of allelobiosis and allelopathy, especially in the case of inducible production of allelochemicals. As more information arises, allelobiosis could be an important factor to include in future efforts to expand the modeling of allelopathy as an ESS.

4.3 | Conclusion

Our model predicts three ESS cases, differing in the benefit and cost to the allelopathic plant. In the first, the nonallelopathic plant is a stronger competitor due to high metabolic costs to the allelopathic plant, and not producing allelochemicals is the ESS. In the second, the allelopathic plant is the better competitor, and production of allelochemicals is the more beneficial strategy. In the last case, the allelopathic and nonallelopathic plants are equal competitors but pay different costs resulting in alternative stable states depending on the history of the system. We find that despite the obvious benefits of allelopathy, there are relatively few conditions that lead to $+A$ as a pure ESS, and that if $-A$ is the ancestral state, the large regions dominated by priority effects would mean $+A$ mutants cannot successfully spread in a population. We argue that these results potentially help explain the relative rarity of allelopathy in nature. Additionally, the four parameters give insight into molecular mechanisms that future biochemical and molecular work could seek to better understand. Further empirical exploration of this model could lead to useful agricultural tools.

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CONFLICT OF INTEREST

The authors declare no competing interests.

AUTHOR CONTRIBUTIONS

R.M.M. conceived the project with guidance from G.G.M. and J.R.W. R.M.M. and G.G.M. performed the modeling. R.M.M., J.R.W., and G.G.M. analyzed the model solutions and wrote the paper.

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