Eco-evolutionary agriculture: a study in crop rotations

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Abstract

Since its origins, thousands of years ago, agriculture has been challenged by the presence of evolving plant pathogens. In response, current practices have started relying on computational tools to design efficient prospective planning, but further efforts for multi-criteria assessment are needed. Here, we present a methodology for developing cultivation strategies optimal for control or eradication of pathogens. This approach can integrate both, traditionally used criteria in crop rotations and the analysis of host-pathogen coevolution systems where hosts are artificially selected. Our analysis shows that prospective planning can maximise cash yield in the long run by investing consecutively in soil quality during initial seasons. Importantly, rational application of crop rotation patterns can minimise yield loss in infected fields, despite the evolution of pathogen virulence. Our results provide strategies for optimal resource investment for increased food production and lead to further insights into minimisation of pesticide use in a society demanding efficient agriculture.

Keywords: agriculture, crop rotations, eco-evolutionary dynamics, plant-pathogen co-evolution

Introduction

Around ten thousand years ago, changes in climate conditions led to the emergence of agricultural practices in human hunter-gatherer communities around the globe [1]. This process of domestication – or artificial selection – has been refined along centuries through trial and error combined with experience, increasing the quantity and quality of the product. In the case of plant agriculture, the presence of pests has been a substantial threat to farmers [2]. The first farmers already tried to overcome the pest problem by employing field rotations, i.e., shifting cultivation techniques [3, 4]. As human population continues to multiply, current agriculture practices need to address a two-fold problem of the dearth of enough food supply and plant pathogens. Techniques such as slash-and-burn, pesticides and fertilisers are used for increasing yield as well as dealing with pests, but do not contribute to agricultural sustainability [5]. Thus, current research needs to focus on developing cropping techniques which increase yield and minimise the environmental impact [6]. Farmers have now started relying on data-based computational tools to design agriculture strategies. Among others, the computational tools involve decision support models for choosing optimal cropping plans and crop rotation decisions [7, 8]. The models guide allocating crops depending on their characteristics – botanical family, market demand, or soil demands –, considering spatial distribution and temporal successions. However, these models are presented as static decisions and the optimisation procedure often lacks multicriteria assessment [9] one such being plant pathogens.

In evolutionary biology, models on host-pathogen coevolution have contributed to understanding the relationship between some pathogens and their host crops, for example, regarding the specificity of the interaction [10, 11]. Plant-pathogen coevolution is a natural selection process happening in tandem with artificial selection of domestication, and the interface between them is worth studying. Recently, authors have highlighted the use of plant-pathogen coevolution in formulating disease management strategies and avoiding the increase of infectivity in pathogens [12, 13, 14, 15, 16]. When observing evolutionary dynamics, a change in the crop type acts as a per-

50 turbation leading to frequent selective sweep-like dynamics. Tracking the frequency
51 and speed of such sweeps would be useful in detecting periods of lower fitness and
52 reduced population size; in which the pathogen could be pushed to extinction [17].
53 Thus, including plant-pathogen coevolution – a natural selection process – in the
54 study of crop rotations would be a new approach for tackling agricultural problems.

55 Here, we aim to design a cultivating strategy optimal for pathogen eradication,
56 integrating criteria traditionally used on crop rotations, and host and pathogen coevo-
57 lution. We have developed an optimisation model for crop rotation patterns which ap-
58 points soil quality and cash yield value as variables of interest. When infection occurs,
59 ecological dynamics play out in the short term. Host-pathogen competitive dynam-
60 ics predicts yield loss depending on crop resistance, as well as changes in pathogen
61 load. Evolution of host resistance and pathogen virulence is considered by mutation
62 of the pathogen into strains which are able to infect the host more efficiently. While
63 some crop rotation patterns are optimal in pathogen-free scenarios, different patterns
64 are shown to be robust to infections, despite minor damages or sub-optimal yield
65 output. Improvement strategies are proposed by (i) introducing engineered resistant
66 variants of the original crops and analysing the corresponding pathogen adaptation
67 following previous models [18]; and (ii) proposing new rotation sequences which show
68 better performance in infection conditions.

69 **Model and Results**

70 Plants have a variety of responses to pest infestations such as susceptibility, tolerance
71 and resistance. In agriculture, farmers have used this variability for thousands of years
72 to control the spread of pathogens over the field. This simple yet powerful concept
73 is formalised below, using a theoretical model which optimises rotation patterns and
74 predicts infection dynamics.

75 2.1 Crop rotation patterns

76 To establish a basic model of rotation patterns, we focus on sequential combinations
 77 of cash crops and cover crops. Cash crops are those which provide a product to
 78 be commercialised – e.g. maize –, whereas cover crops improve soil quality of the
 79 field but provide no direct substantial cash yield – e.g. clover. Because more soil
 80 quality provides more cash yield, both crop types are needed for successful farming:
 81 the aim of the basic model is to study which temporal patterns of cash and cover
 82 crops maximise the farmer’s benefit. For that, we have been inspired by a previous
 83 report assessing the optimal length of clover period, compared to maize, in a 9-year
 84 field experiment [19]. We work with pattern sequences of length $L = 10$, in contrast
 85 with common short rotations of $L \approx 3$, aiming to acquaint long-term patterns with
 86 increased optimisation.

87 The more cash yield a particular sequence provides the farmer, the more likely it
 88 is to be used repeatedly. This concept can be connected to the idea of differential
 89 survival. Better yielding sequences survive and reproduce i.e. are used again. Thus,
 90 we can connect the notion of fitness to the optimality of a sequence [20]. Here we
 91 attribute each of our rotation sequences a fitness value F , depending on their ability
 92 to maximise cash yield. In principle, the fitness function could be modified to optimise
 93 both final soil quality and cash yield, as discussed later on.

94 Each element in a rotation sequence corresponds to a harvesting season, mod-
 95 elled as a discrete time-step. During each time-step, there is a change in soil quality
 96 q_t and cash yield y_t , which varies depending on the host type $h_i, i = \{1, 2\}$. At the
 97 end of the sequence, F is set as the final cash yield (Fig. 1). Soil quality and cash
 98 yield per time step are crucial in obtaining the fitness value, taking the following form:

- Soil quality (q_t): Soil quality decreases following a logistic decay curve for n_1 consecutive h_1 cash crops, and increases with logistic growth for n_2 consecutive h_2 cover crops. The parameter β_i regulates the intensity and direction of the soil quality change given crop type i : we set $\beta_1 = -1.5$ for soil quality decrease by h_1 , and $\beta_2 = 1$ for soil quality increase by h_2 , considering that it

Figure 1: Soil quality and cash yield values of a rotation sequence along time.

Each time step corresponds to a harvesting season. Dots indicate discrete values for soil quality (circles) and cash (squares). Season crop type is indicated in orange for cash crops h_1 , and green for cover crops h_2 . Fitness F is equivalent to the cash yield value at $t = L$ (here $t = 10$).

is more difficult to improve soil quality than to decrease it (see Supplementary Material). We assume that the soil quality cannot increase indefinitely, reaching a saturation value – or carrying capacity – of K . We choose $K = 2$, for which approximately $n_2 = 4$ harvesting seasons are needed to reach it with $\beta_2 = 1$, similar to experimental work [19].

$$q_t = \frac{K q_{t-1} e^{\beta_i n_i}}{K + q_{t-1} (e^{\beta_i n_i} - 1)} \quad (1)$$

- Cash yield (y_t): Cash yield increases in proportion to the soil quality of the previous time-step q_{t-1} , regulated by the effective crop ratio δ_i , and the crop yield contribution γ_i . For cash crops we set $\gamma_1 = 1$, considering cash yield increases as much as the soil quality, in a 1:1 ratio. For cover crops, there is no cash yield, being $\gamma_2 = 0$. Overall cash yield is accumulated along the rotation

sequence time-steps.

$$y_t = y_{t-1} + \gamma_i q_{t-1} \quad (2)$$

- Fitness (F): Given that we consider fitness to be the total economical benefit that a sequence provides, we set it equivalent to the total accumulated cash yield (i.e. y_L).

Fitness of each sequence in a population of sequences of length $L = 10$ is computed according to the above-defined functions and analysed.

Results show that fitness distribution of the population of rotation sequences of $L = 10$ ($N = 1024$) has a non-zero (positive) skew: the proportion of sequences with high fitness values is small, indicating it is crucial to know the patterns underlying them (Fig. 2a). We have analysed the selection of sequences with a fitness value greater than two standard deviations from the mean, with the sequence population being reduced to $N = 16$. From this selection, we can observe the following patterns: (a) all sequences in the selection have an equal proportion of cover and cash crops; (b) all sequences in the selection end with a minimum of two cash crops; (c) most sequences start with cover crops, and the ones that start with cash, they set consecutive cover crops in the next seasons (Fig. 2b, 2c).

2.2 Patterns under infection: eco-evolutionary dynamics

In natural settings, the process of coevolution between the host plant and its pathogen can lead to the cyclic evolution of host resistance and parasite virulence, maintaining genetic diversity [14]. In agriculture, humans are the selecting agent: they decide which host grows in the next generation. While being economically important, the selected crop can be particularly vulnerable to pathogens which it has not coevolved with. Moreover, there are only a few major agricultural cash crops; resulting in less genetic diversity in host crops and more disease susceptibility [21]. In this section, we see how the introduction of a pathogen into our system modifies the fitness of the rotation sequences. We start with a simple infection scenario in which a pathogen p can infect cash crops h_1 , but not the cover crops h_2 . As an example, the fungi

Figure 2: Fitness distribution of the population of sequences of $L = 10$ and its optimal patterns. a) Fitness distribution of population of study ($N = 1024$). Dashed line indicates two standard deviation over the mean threshold for F . b) Crop rotation patterns of selected sequences over the threshold ($N = 16$). Gray-scaled squares on the right indicate fitness value relative to the selection. c) Frequency of cash h_1 and cover h_2 crops among the selection of sequences in each harvesting season.

125 *Fusarium graminearum* [22] is one such pathogen which infects cash crops as maize
126 or wheat, but does not infect cover crops such as clover.

127 **Ecological dynamics.** To include host-pathogen ecological dynamics, we adapt
128 the Lotka-Volterra competitive equations, based on [23]. Within a season, time is
129 continuous and dynamics are described by a system of ordinary differential equations,
130 corresponding to m host types and n pathogens (Eq.(3)).

$$\begin{aligned} \dot{h}_i &= h_i \sum_{j=1}^n -\sigma_i p_j \\ \dot{p}_j &= \sum_{i=1}^m \sigma_i h_i p_j - d_p p_j \end{aligned} \quad (3)$$

131 While the equations are for multiple hosts and pathogens, we start with two hosts and
132 a single pathogen ($m = 2$ and $n = 1$). Here h_i is the population density of crop i
133 and p_j is the pathogen density. The pathogen infectivity is set by σ_i ($\sigma_1 = 0.04$ for the
134 cash susceptible crop, $\sigma_2 = 0$ for the cover resistant crop), and d_p is the death rate of

the pathogen ($d_p = 0.5$). Due to the artificial setting of agriculture, we consider that without external perturbations and independently of the pathogen interaction, there is no birth nor death in host population during the season.

Between seasons, however, the system changes in discrete time-steps. At the end of each season, the crop is harvested, converted to yield and new crops are planted as per the rotation schedule. Due to the harvest, the pathogen population is disturbed. The pathogens are partially eliminated (ϵ) when the crop is harvested but some survive in the soil or in residues of the infected crops. We set $\epsilon = 0.5$, considering half of the pathogen population stays in the field. Overall, the model evolves following continuous time within seasons, and discrete jumps between seasons, being an example of a hybrid dynamical system [24] (Fig. 3).

Eco-evolutionary Dynamics. Substantial evolutionary changes can happen on ecological time-scales [25, 26, 27]; thus, we need to consider evolution in the host-pathogen interaction. In our case, we study the dynamics when there is evolution of pathogen virulence, and incorporate them in the already developed ecological dynamics. In literature, virulence refers to the pathogen capacity to establish an infection or the consequences for the host to be infected [28]. Here, by evolution of virulence, we refer to the change in the propensity of a pathogen to infect the host. Within a season, the pathogen reproduces, generates variation, and some of these variants may carry mutations that provide more virulence. To incorporate evolution in the ecological dynamics, we modify the previous equations (3) allowing the pathogen to mutate into strains which can exploit the cash host more efficiently, Eq. (4). The evolved pathogen cannot evolve to infect the cover crop since cash and cover are assumed to be phylogenetically distant.

$$\begin{aligned}\dot{h}_i &= \sum_{j=1}^{n=5} -W_{ji}\sigma_i h_i p_j \\ \dot{p}_j &= \sum_{k=1}^{n=5} Q_{kj} p_k \sum_{i=1}^{m=2} W_{ji}\sigma_i h_i - p_j d_p\end{aligned}\tag{4}$$

The new equations have two important elements: the transition matrix Q_{kj} and the fitness matrix W_{ji} . The transition matrix Q_{kj} corresponds to the probabilities of the pathogen to mutate into other strains. We define four possible mutants, separated by

Figure 3: Host-pathogen ecological dynamics, between and within seasons. a) Dynamics between seasons, following a hybrid system. After each harvesting season, initial host density $H = 50$ is reinitialized and pathogen is reduced according to parameter $\epsilon = 0.5$. b) Dynamics within a season, when there is a susceptible cash crop h_1 . Host density decreases due to the presence of the pathogen, while the pathogen load increases as long as there are enough crops to infect. c) Dynamics within a season, when there is a resistant cover crop h_2 . The resistant cover crop maintains its output throughout the season and remains unaffected by the presence of the pathogen; however, the pathogen dies out to the lack of a suitable host to grow on.

a genetic distance d from the original strain. Mutation can happen between strains which are one mutational step away ($d = 1$) with a transition probability $\mu = 0.1$.

$$Q_{kj} = \begin{matrix} & \begin{matrix} p_1 & p_2 & p_3 & p_4 & p_5 \end{matrix} \\ \begin{matrix} p_1 \\ p_2 \\ p_3 \\ p_4 \\ p_5 \end{matrix} & \begin{pmatrix} 1-\mu & \mu & 0 & 0 & 0 \\ \mu & 1-2\mu & \mu & 0 & 0 \\ 0 & \mu & 1-2\mu & \mu & 0 \\ 0 & 0 & \mu & 1-2\mu & \mu \\ 0 & 0 & 0 & \mu & 1-\mu \end{pmatrix} \end{matrix} \quad (5)$$

For the fitness matrix W_{ji} , we set the fitness of the original pathogen p_1 to $w_{11} = 1$ when infecting h_1 ; and to $w_{12} = 0$ when infecting h_2 . Each mutant increases the fitness proportional to the distance with respect to p_1 , so $w_{j1} = w_{11} + 0.25d$, with $w_{51} = 2$ being the maximum fitness. Infecting h_2 does not provide any fitness benefit, with $w_{j2} = 0$. The fitness matrix, when multiplied by the parameter σ , shows the increase in virulence in each mutated strain of the pathogen. Overall, eco-evolutionary dynamics results in elevated yield loss as compared to the solely ecological dynamics (Fig. 4).

2.3 Sequence improvement: pattern rearrangement

Under eco-evolutionary dynamics, infection causes yield loss in susceptible cash crops, which becomes more noticeable when pathogen virulence increases. Consequently, pathogen presence has repercussions on each rotation sequence fitness: accumulation of cash is diminished, depending on the proportion of crops that survive the infection by the end of the season, making the fitness value decrease.

We need to compute how much yield is lost due to the pest, and for that, we have included a new parameter: the effective crop ratio, or δ_i . It indicates the proportion of crops that contribute to the change in soil quality or cash yield. To represent the loss, we have set δ_i to be the ratio of the density of hosts remaining at the end of the season h_i and the initial host density H , Eq. (6),

$$\delta_i = \frac{h_i}{H}. \quad (6)$$

Figure 4: Ecological vs. eco-evolutionary dynamics of host-pathogen interaction.

a) Ecological dynamics, without pathogen evolution. Dynamics between seasons are represented, with infection starting at $t = 4$. In b), pathogen population is homogeneous, due to the absence of mutation. c) Eco-evolutionary dynamics with pathogen virulence evolution. Dynamics between seasons are represented, with infection starting at $t = 4$. Because of pathogen evolution, the impact of the infection in the last h_1 provokes higher host density lost, compared to a). In d), time evolution of pathogen shows that the relative abundance of fitter strains – in darker colours – increases along seasons.

173 Included in the equations of soil quality and cash yield, Eqs. (7), δ_i modifies the out-
174 come of the season.

$$\begin{aligned} q_t &= \frac{Kq_{t-1}e^{\delta_i\beta_i n_i}}{K + q_{t-1}(e^{\delta_i\beta_i n_i} - 1)} \\ y_t &= y_{t-1} + \delta_i\gamma_i q_{t-1}. \end{aligned} \quad (7)$$

175 Therefore, in neutral conditions there is no loss of host density and $\delta_i = 1$. In infection
176 conditions, $\delta_1 < 1$ because of host death due to the pathogen; and $\delta_2 = 1$ as h_2
177 density is not affected. The loss in host density is consequential for the final cash
178 value, influencing the fitness F of each sequence.

179 Implementing eco-evolutionary dynamics in the model shows that under different
180 infection scenarios (see Supplementary Material for infection at different time-points),
181 the optimal sequence varies. Hence, the sequence with higher fitness under neutrality
182 becomes non-optimal and can potentially be changed to a fitter sequence. Consid-
183 ering this, we next describe how to improve the initial sequence once infection has
184 occurred.

185 Improving the current rotation sequence consists of comparing the focal optimal
186 sequence – the best under neutral conditions – with the whole population under in-
187 fection conditions. From the complete population of sequences, those ones which (i)
188 patterns match until infection season – included –, and (ii) have greater fitness under
189 infection, are considered candidate sequences for improvement. Since all infection
190 scenarios show similarities among the optimal patterns (see Supplementary Mate-
191 rial), few crop changes can already reduce the pest impact. As we assume no cost
192 for pattern changes, we select the fittest sequence from the candidates (Fig. 5).

193 2.4 Sequence improvement: introduction of resistant cash crop

194 To prevent yield loss, a different strategy could be the introduction of a variant of the
195 cash crop with resistance to the pathogen: h_1^* . Acquiring a new resistance is asso-
196 ciated with a cost, often a yield penalty [29, 30]. Thus, crop yield contribution would
197 be set to $\gamma_1^* = 0.8$ (in contrast to $\gamma_1 = 1$, for the standard cash crop). However, the

Figure 5: Pattern rearrangement strategies in infection conditions. In neutrality, the central sequence is calculated to be the fittest one ($F = 9.75$). However, when there is infection at $t = 4$ its fitness decays to $F' = 6.90$. In infection conditions, the top sequence (dashed box) is indicated to be the fittest ($F' = 8.78$); but because the pattern previous to the infection time (white background) is different, we cannot rearrange the pattern to be the best one. In consequence, bottom sequence (bold box) is the best option for minimising yield loss, as it is the fittest sequence ($F' = 7.27$) where the pattern until the harvest of the infection crop is the same.

198 pathogen, in turn, could evolve to infect the resistant variant. The pathogen infecting
199 multiple host types follows a gene-for-gene infection model [10].

200 Gene-for-gene dynamics has been widely described in plant-pathogen relation-
201 ships [10, 31]. It is based on the idea that there is a correspondence between the
202 genes providing resistance to the host and virulence for the pathogen. Carrying re-
203 sistance supposes a cost for the host, and equivalently the pathogen has a cost for
204 virulence. When acquiring the virulence allele, the pathogen does not lose its abil-
205 ity to infect the non-resistant host and thus, becomes a generalist. The contrasting
206 model would be the matching alleles, in which the mutated pathogen is not infective
207 any more for the host which genotype does not correspond specifically [18].

208 In our model, we modify the transition $Q_{\kappa j}^*$ and fitness W_{ji}^* matrices to include
209 gene-for-gene evolution of the pathogen. Each specialist pathogen p_j can mutate,
210 with a transition probability μ , into its corresponding generalist variant p_j^* . Further-
211 more, each strain of p_j and p_j^* can mutate into fitter strains of their type (Fig. 6). In
212 W_{ji}^* , parameter α corresponds to the gradient between matching alleles and gene-
213 for-gene infection models, as described in [18]. We also include a cost κ of carrying
214 a resistant allele for the mutated pathogen.

215 Replacing the previously susceptible cash crops h_1 with resistant cash crops h_1^* ,
216 the frequency of newly evolved pathogens p_{j^*} increases until it outcompetes the old
217 strains of pathogen p_j (Fig. 6c). Because of the cost of resistance of the evolved
218 pathogen and the initial cash host resistance, the effect on yield loss is diminished.
219 However, infection persists due to the adaptation of pathogen.

$$W_{ji}^* = \begin{matrix} & h_1 & h_1^* & h_2 \\ \begin{matrix} p_1 \\ p_2 \\ \dots \\ p_1^* \\ p_i^* \end{matrix} & \begin{pmatrix} 1 & 0 & 0 \\ 1.25 & 0 & 0 \\ \dots & \dots & \dots \\ a(1 - ak) & 1 - ak & 0 \\ a(\omega_{j1} - ak) & \omega_{j1} - ak & 0 \end{pmatrix} \end{matrix} \quad (8)$$

Figure 6: Pathogen gene-for-gene evolution and eco-evolutionary dynamics of host-pathogen interaction. a) Table for pathogen evolution, following gene-for-gene model of infection. b) Eco-evolutionary dynamics with pathogen virulence evolution, and adaptation to new host h_1^* . Dynamics between seasons are represented, with infection starting at $t = 4$. At $t = 7$, the new resistant host h_1^* is cultivated, preventing the increase of pathogen density. However, the pathogen mutates following the gene-for-gene model, being able to infect both h_1 and h_1^* , and at $t = 8$, there is a noticeable loss of host density. In c) strains of the original pathogen which only evolve in virulence – in grey – dominate pathogen population during the first seasons, in which only h_1 and h_2 are present. At $t = 7$, h_1^* is introduced: only the strains which acquire the generalist mutation – in brown – are able to reproduce, which causes a fast increase on their relative abundance until the fixation of the generalist trait in the last season.

Discussion

Translational evolutionary biology is an emerging field where fundamental concepts from evolutionary biology can be used in an applied setting to make practical changes in society [32, 33]. Just as with the search for novel antibiotics, the search for novel agricultural strategies has caught the interest of evolutionary biologists. Particularly, it has been discussed how evolutionary principles can be applied to pest management in agroecosystems. Our work complements previous attempts on coupling plant genetics with resistance deployment strategies [16, 34], bringing into focus plant-pathogen dynamics and pathogen evolution in the context of crop rotation sequences.

We present a model for assessing how different patterns of cash and cover crop rotations influence long-term yield outcome. Our results highlight only a few patterns, from all possible crop sequences, maximising the yield after ten seasons of harvest. These patterns consist, overall, of investing in soil quality during the first seasons by cultivating cover crops, and once the carrying capacity of the soil is reached, introducing cash crops. For an accurate interpretation of these results, one must regard the conditions we have chosen. The initial soil quality, for example, is set at half of the carrying capacity, considering it an average value: different values could change the number of initial seasons invested in cover crops. Besides, using cover crops more efficient at increasing soil quality – this is, increasing their β_i – would increase the proportion of cash crops to be cultivated, augmenting the final cash yield. On the other hand, we have decided to maximise cash yield at the end of the sequence, for which last seasons are always dedicated to cash crops and soil quality is depleted. To allow continued use of the same sequence, we could optimise both soil quality and cash yield, so the final and initial soil quality have the same value. This new constraint would lead to a type of pattern possibly more regular in time, but reduced cash yield.

During the harvesting seasons, pathogens may invade the field and damage the crops, diminishing the expected yield. Using plant-pathogen dynamics, we have tracked the ecology and evolution of the infection in discrete and continuous time, predicting yield loss. Reported computational tools [7, 8, 9] rely on historical data

249 to predict which is the best decision; instead, our model gives *de novo* assessment
 250 integrating features of the crop field, economy and evolution (i.e. soil quality, cash
 251 yield and pathogen dynamics, respectively). Moreover, we bridge evolutionary biol-
 252 ogy knowledge applied to agricultural strategies, whereas previous theoretical models
 253 are mostly focused on epidemiology [16, 35] and do not model explicitly pathogen dy-
 254 namics and crop rotations sequences.

255 Farmers, looking for deployment strategies, can be guided by our improvement
 256 proposals: (i) pattern rearrangement and (ii) introduction of a resistant host. The first
 257 works purely at the ecological scale: it shows that by few changes in the optimal se-
 258 quence based on reordering or adding cover crops, host resistance attenuates the
 259 increase of the pathogen load. This strategy can diminish the total yield loss, even
 260 when the number of cash crop seasons is reduced. The second approach integrates
 261 eco-evolutionary knowledge making use of resistant varieties of the crops arranged
 262 in the right pattern. It shows that using a new variant of cash crop that has resis-
 263 tance can help to overcome the pest during a certain time period. However, in the
 264 long term, the pathogen can adapt and infect the new crop type as well. We have
 265 implemented the co-evolutionary process following the gene-for-gene model of plant-
 266 pathogen interactions [10]. Previous models describe the gene-for-gene approach in
 267 continuous evolutionary processes, whereas here it is used in the hybrid dynamical
 268 system across multiple harvesting seasons.

269 The agricultural strategies we propose fit previous work affirming that crop sea-
 270 sonality can slow down adaptation and promote evolutionary branching [36]. How-
 271 ever, these strategies do not eradicate the infection, as in agriculture most of the crop
 272 pathogens evolve rapidly. This fast adaptation phenomenon occurs because high
 273 planting density and genetic uniformity of host increase the effective population size
 274 and genetic diversity of the pathogen [37]. Current research works on cultivar mix-
 275 tures and their spatial distribution to diversify host genetics [38, 39, 40]. Our model
 276 considers a monoculture in a single field per season, but to complement these re-
 277 search lines, it could be extended by including more variation in host types, spatial
 278 structure, and between-field pathogen migration. Also, an increase in the number of

279 host types and the number of pathogens could lead to a model exhibiting complex,
280 and even chaotic, dynamics [41], which would be interesting to investigate.

281 Overall, our model can advise on strategies for maximising the gain of yield in cash
282 crops, while using cover crops for soil improvement and control of pathogen spread.
283 Further insights on rational resistance patterns could lead to new approaches for
284 reducing pesticide use: the application of pesticides on strategic points where the
285 pathogen density is low could both improve the efficiency of pesticides by increas-
286 ing pest clearance and reducing the used amount of pesticide. These could possibly
287 help disentangle the effect of crop rotations and pesticide use, in controlling pests
288 and increasing yield. Experimental settings focus on crop rotations as the main fac-
289 tor for pest control, when put together with resistance variants and pesticides [22].
290 Agroecosystems rely on human artificial selection for controlling the outcome of the
291 harvest: we can profit from an evolutionary outlook bringing new tools towards a more
292 sustainable farming. Ideas such as the one presented in this research are merely the
293 first steps towards achieving this goal.

294 **Code availability.** Appropriate Python computer code describing the model is
295 available at <https://github.com/tecoevo/agriculture>.

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299 **Supplementary material**

300 **4.1 β parameter space**

301 The beta parameter indicates how fast a particular crop increases or decreases, ac-
302 cording to its sign, the soil quality. Depending on its value, we expect the optimal
303 patterns to be different: if cover crops improve soil quality rapidly, and cash crops
304 decrease it very slowly, we would expect that optimal patterns have few cover crops,
305 compared to the number of cash crops. In our case of study, we set $\beta_1 = -1.5$ for

306 cash crops (h_1) and $\beta_2 = 1$ for cover crops (h_2), making soil quality decrease faster
 307 when one cash crop is cultivated, than the increase cover crops bring during one
 308 season. Using these β values, the optimal sequences presented a ratio of cash:cover
 309 crops of 1:1.

310

311 Here, we analyse the β parameter space in case of (i) the ratio of cash cover
 312 crops in the selection – sequences with fitness above two standard deviations over
 313 the global mean –, and (ii) the maximum fitness values of this selection. We do both
 314 analyses in the neutral scenario (without infection), and we explore the ratios com-
 315 ing from the combinations of values $\beta_1 = \{-0.5, -1, -1.5, -2\}$, $\beta_2 = \{0.5, 1, 1.5, 2\}$.
 316 The average values of the selection for each combination of β values is shown in a
 317 heatmap (Fig. SI.1).

318

319 Low β_1 and high β_2 lead to a higher proportion of cash crops in the rotation se-
 320 quence and a general increase of fitness values. This can be interpreted as cover
 321 crops being very efficient, so few of them are needed to achieve values close to the
 322 carrying capacity of soil. Consequently, as fitness depends on the maximisation of
 323 cash, an increase on the proportion of cash crops – if the soil quality is high – brings
 324 to higher fitness. Combinations in which the absolute value of β_1 equals β_2 , the ratio
 325 cash:cover is approximatively 1, leading to patterns and fitness maximum similar to
 326 the ones studied in the paper. Those cases in which β_1 is greater than β_2 tend to have
 327 low fitness maximum and low ratio cash:cover. Because of cover crops improving the
 328 soil quality slowly, many of them are needed to take profit of cultivating cash crops,
 329 reducing the cash season and thus, the economic outcome, or fitness.

330 4.2 Optimal sequence pattern depending on the time of infection

331 In sections 2.2 and 2.4, we have analysed eco-evolutionary dynamics showing a spe-
 332 cific time of infection ($t = 4$). However, infection could occur at any season or time,
 333 and this could change the optimal sequence patterns. Here, we analyse the differ-

Figure SI.1: Fitness maximum and ratio cash:cover for different values of β_1 and β_2 . Each patch in the heat map represents the mean value of (a) fitness maximum and (b) ratio cash:cover in the selection of optimal sequences – with fitness value higher than two standard deviations above the mean – in different combinations of β_i values, represented on the axis. The combination of values used for the results in the main text ($\beta_1 = -1.5, \beta_2 = 1$) is highlighted with a black square.

ences between the optimal patterns at different times of infection. Similarly to other sections, we have taken a subset of the population of sequences: those which fitness value F is over two standard deviations from the mean of the population. For each selection, we have plotted the frequencies of cash crops in each season (Fig. SI.2). During seasons in which frequency of cash equals 0 it is optimal to cultivate cover; per contra, if the frequency of cash is 1, it is always optimal to cultivate cash. When the frequency of cash is 0.5, the convenience of cash or cover depends on the crops cultivated before. However, we can take those cases as seasons in which choosing a particular type of crop will not change drastically the final outcome of the rotation.

Results qualitatively show that the patterns are similar between them. In all of them, there is a tendency to start with cover crops and invest in cash crops at the end of the season; whereas the intermediate positions have intermediate frequency values and are variable among the optimal selection. Interestingly, optimal sequences do put a resistant cover crop after the time of infection in most of the cases, so pathogen load can be reduced. This is not the case for infection in the later times ($t = 8, 9$), in

Figure SI.2: Frequency of cash crops in the optimal sequences at different times of infection. For each discrete time – i.e. season – of infection, the frequency of cash crops on the selection of optimal sequences (sequences with F over two standard deviations from the mean) is shown (orange bars). By subtracting the frequency of cash from 1, the frequency of cover crops can be inferred (corresponding to green in the plot). A frequency of 0 indicates that in all optimal sequences, cover crops are cultivated for that season. A frequency of 1 indicates that in all optimal sequences, cash crops are cultivated for that season. A frequency of 0.5 (dashed line) indicates that cover and cash crops are equally cultivated during that season. Other intermediate frequency values indicate that among optimal sequences, there is variation on the crop cultivated.

350 which keeping on investing in cash crops is yet the best strategy.

351

352 From this, we can observe that few changes in the optimal sequence in the neutral
353 scenario can lead to optimal sequences in infection, and diminish fitness loss. Thus,
354 the strategy of improvement based on pattern rearrangement is doable for all times
355 of infection.

356 4.3 α parameter space

357 The α parameter refers to the gradient between gene-for-gene (GfG) and matching
358 alleles (MA) models of infection. It was used in Agrawal and Lively (2002) [18] and
359 posterior literature [23] for analysing the differences in behaviour of their systems
360 between the two models of infection. Plant-pathogen coevolution is commonly asso-

ciated to GfG patterns [10], and for our study we have used $\alpha = 0.5$, which represents partial GfG. Here, we analyse how the pathogen strain relative abundance changes according the values $\alpha = \{1, 0.5, 0\}$, corresponding to complete MA, intermediate GfG and complete GfG, respectively.

We compare the relative abundance of the pathogen p_1 which can only infect h_1 crops, and p_1^* which can infect both h_1 and h_1^* . The analysis shows that the α value influences pathogen evolution. For $\alpha = 0$, the pathogen stays specialist – following the MA model – until the h_1^* crop is introduced; then a mutated strain p_1^* outcompetes the whole initial p_1 population. No mutation nor abundance of p_1^* occurs before. For $\alpha = 0.5$ we find an intermediate case: in the presence of h_1 there is already low frequency (< 0.1) of p_1^* , which increases when h_1^* is introduced. For $\alpha = 1$, frequency of p_1^* increases up to 0.2 before h_1^* is introduced. With the introduction of h_1^* , frequency of p_1^* increases and reaches 1.

4.4 Fixed parameters

In the model, there are several parameters which have fixed values. We have compiled them in Table 1.

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Table 1: List of fixed parameters used in the model

parameter	description	default value	reference
q_0	Initial soil quality	$q_0 = 1$	
y_0	Initial cash yield	$y_0 = 0$	
K	Carrying capacity of the soil	$K = 2$	
β_i	Soil contribution of host h_i	$\beta_1 = -1.5, \beta_2 = 1, \beta_1^* = -1.5$	
γ_i	Cash contribution of host h_i	$\gamma_1 = 1, \gamma_2 = 0, \gamma_1^* = 0.9$	
ϵ	Pathogen clearance	$\epsilon = 0.5$	
κ	Cost of resistance for the pathogen	$\kappa = 0.1$	[18, 23]
μ	Transition probability	$\mu = 0.1$	
α	GfG - MA gradient value	$\alpha = 0.5$	[18, 23]
σ	Infectivity	$\sigma = 0.04$	[18, 23]
H	Initial host density	$H = 50$	
d_p	Death rate of the pathogen	$d_p = 0.5$	[18, 23]

Figure SI.3: Pathogen evolution in different α values. Pathogen strain relative abundance is shown for three values of α ($\alpha = 0, 0.5, 1$). When $\alpha = 0$, the curve corresponds to a matching alleles (MA) model, in which the p_1^* strain – in brown – only evolves when h_1^* is present because it is specialised. On the contrary, $\alpha = 1$ reflects a pure gene for gene (GfG) model, where the p_1^* strain is a generalist and thus, already appears during the presence of h_1 . Value of $\alpha = 0.5$ corresponds to an intermediate MA-GfG, and it is the value used in the main results.

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