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How many generations does it take for phytophages to colonize invasive plants? Mathematical modeling predictions

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Abstract. Native phytophagous insects are often not adapted to novel chemistry of invasive plants, but over time they begin to adapt and feed on them. We simulated the spread of a mutant allele that enables phytophages to feed on invasive plant as effectively as on native plant. This simulation involved two insect populations associated with native and invasive plant species, with gene flow between them. Fitness was assigned using the Ricker function, which incorporated plant abundance, insect feeding efficiency, and competition between genotypes. For the mutation to become fixed in fewer than one hundred generations, invasive plant must be at least as abundant as native one. The effect of invasive plant relative abundance is larger than that of fitness differences in feeding efficiency of wild type phytophages between plants. The spread of this allele under natural selection is faster if it has come from standing genetic variation, rather than newly arisen mutation, or, in the latter case, if there is assortative mating.

Key words: mathematical model, population dynamics, biological invasions, insect phytophages.

Сколько поколений нужно фитофагам для освоения инвазивных растений? Прогнозы математического моделирования

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Резюме. Аборигенные насекомые-фитофаги часто не адаптированы к защитным веществам инвазивных растений, но через некоторое время они адаптируются к ним и начинают питаться инвазивными видами. Проведено моделирование распространения мутантного аллеля, который позволяет фитофагу питаться инвазивным растением так же эффективно, как и аборигенным растением, в двух популяциях насекомых, ассоциированных с аборигенным и инвазивным видами растений, с потоком генов между ними. Для оценки приспособленности использовали функцию Рикера с включением таких факторов, как обилие растений, эффективность питания насекомых и конкуренция между генотипами. Инвазивное растение должно быть по крайней мере таким же многочисленным, как и местное, для фиксации мутации менее чем за сто поколений. Эффект относительной численности инвазивного растения сильнее, чем эффект различий в эффективности питания фитофагов дикого типа на разных растениях. Распространение этого аллеля под действием естественного отбора происходит быстрее, если он исходно присутствовал в популяции как элемент генетической вариации, а не появился в результате вновь возникшей мутации, или, в последнем случае, если имеет место ассортативное спаривание.

Ключевые слова: математическое моделирование, динамика популяции, биологические инвазии, насекомые-фитофаги.

Introduction

Alien plants in secondary ranges are often spared from the pressure of phytophages [Cappuccino, Carpenter, 2005; Liu, Stiling, 2006], which allows them investing less resources in defense and more in growth and reproduction. The enemy release hypothesis uses this fact to explain the success of invasive species [Blossey, 2011; Heger, Jeschke, 2014]. Over time, native phytophages can adapt to feeding on invasive species [Carroll et al., 2005; Siemann et al., 2006; Brändle et al., 2008]. However, the duration of this period varies widely and is impossible to predict.

Some non-native plant species adapt to the new environment quickly and begin experiencing higher levels of herbivory, while others experience lower levels of pressure for many years. For example, alien Piper aduncum and P. umbellatum (Piperaceae), introduced to Papua New Guinea less than 50 years ago, had the same species richness and abundance of caterpillars as the native P. micropiper [Novotny et al., 2003]. On the other hand, the non-native Reynoutria japonica (Polygonaceae), introduced to North America and Europe in the 18th century, experiences less herbivory and pathogen attack than the native R. scandens when comparing leaf damage and herbivore abundance and diversity [Williams, Sahli, 2016]. Meta-analysis has shown that the time since introduction of a non-native plant species is a significant predictor of the enemy release from phytophages, and recently introduced species tend to experience less pressure from herbivores, but this effect diminishes over 50–200 years [Hawkes, 2007].

However not all researches reveal relationship between time since introduction and herbivory [Carpenter, Cappuccino, 2005]. Several factors influence the rate at which reciprocal interactions develop between introduced plants and native herbivores. Herbivore adaptation to an introduced plant is facilitated by the presence of native

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plants with similar chemical profiles. Comparing the chemical profiles of all plants in a community can be a daunting task, although phylogenetic relatedness can serve as a proxy of such similarity, with fewer native relatives of the introduced plant being associated with lower herbivore accumulation [Connor et al., 1980; Cappuccino, Carpenter, 2005]. In addition, the rate at which alien plants recruit an assemblage of herbivores depends on the native pool of phytophagous insects and the balance between specialists and generalists in the community [Cornell, Hawkins, 2003]. It was shown that the arthropod communities associated with an annual crop plant species in Japan exhibited an increasing proportion of family specialists over time since introduction [Andow, Imura, 1994].

In addition, limited knowledge about the introduction history of many species, often overlooking herbivores during the early colonization phases, assessment of the true distribution of the invasive plant and conduction of comprehensive studies of herbivore population changes over time complicate studying local herbivore adaptation to invasive plants. To address this, mathematical modeling can be used to study the population dynamics of herbivores.

We built a mathematical model to determine the conditions under which a mutation allowing more efficient consumption of the invasive plant can spread in the specialized herbivore population and how long this process takes.

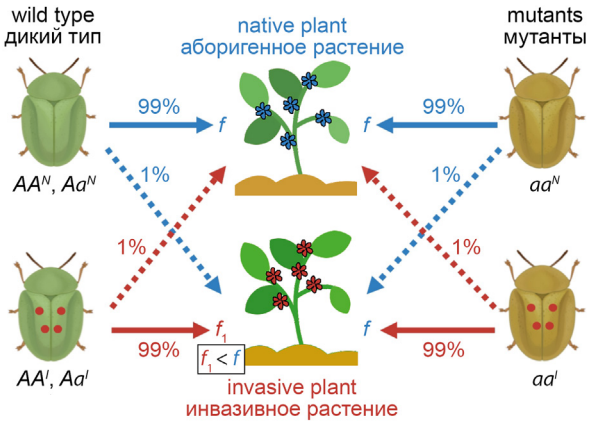

Fig. 1. Scheme of interactions between phytophages and their host plants in our model. There are two plant species – native and invasive, and two ecological races of the phytophage – the wild type and mutants. Each ecological race is represented by two populations, one feeding on the native plant, and the other on the invasive plant (beetles from this population are marked with dots on the elytra). *f* is the coefficient reflecting the efficiency of feeding of the phytophage on the plant; for the wild type on the invasive plant, the coefficient f_1 is used, which is lower than *f*. In each generation, 99% of each population remains on its host plant (solid arrows), and 1% of the population migrates to an alternative plant (dashed arrows).

Рис. 1. Схема взаимодействий между фитофагами и их кормовыми растениями в нашей модели. Есть два вида растений – аборигенное и инвазивное, а также две экологические расы фитофага – дикого типа и мутанты. Каждая экологическая раса представлена двумя популяциями, одна из которых питается на аборигенном растении, а другая – на инвазивном (жуки из данных популяции отмечены точками на надкрыльях). *f* – коэффициент, отражающий эффективность питания фитофага на растении; для дикого типа на инвазивном растении используется коэффициент f_1, который ниже, чем *f*. В каждом поколении 99% каждой популяции остается на своем кормовом растении (сплошные стрелки), а 1% популяции мигрирует на альтернативное растение (пунктирные стрелки).

The ability to feed effectively on another host plant can be achieved without decreasing the effectiveness of the initial host plant's digestion. So, in the absence of the invasive plant, this mutation is neutral, and in the presence of the invasive plant, it is beneficial. Neutral mutation can reach rather high frequency by genetic drift, which should facilitate fixation [Kimura, 1968]. The problem of spread of beneficial mutation was solved by Fisher [1930] in general, but we are interested in the specific case where we do not define adaptive value per se. Instead, we model differences in fitness using the Ricker function, which incorporates the factors influencing the spread of the mutation.

We hypothesized that the timeframe required for the spread of a mutant allele within a population is influenced by the abundance of the invasive species relative to the native one, differences in herbivore consumption efficiency of the invasive plant between mutant and wild type, and the initial frequency of a mutant allele. A high abundance of the invasive plant creates strong selective pressure, promoting the spread of the mutation that enhances the efficiency of consuming the invasive species. The more effective the consumption of the invasive plant by the mutant phenotype compared to the wild phenotype, the more advantageous the mutant allele is, resulting in a higher selection coefficient. We also hypothesized that assortative mating should facilitate the spread of mutation, since it increases the proportion of homozygotes.

Methods

The simulations were performed using the R programming environment [R Core Team, 2024].

We considered a deterministic model of plant-phytophage interactions in the presence of a native host plant and phylogenetically related invasive plant species affected by one species of phytophagous insect. The phytophage has two populations associated with these two plant species, whose sizes are modeled separately. The phytophage has two ecological races determined by a single diallelic locus: for a wild type, the feeding efficiency on the invasive species is lower on the native plant, a mutant feeds equally effective on both native and invasive plant species (Fig. 1). Complete dominance is assumed so wild-type homozygotes and heterozygotes have a wild-type phenotype and only mutant homozygotes exhibit a mutant phenotype.

The first stage of the model simulates mating and the transition to the next generation. The next stage involves the migration of phytophages between two populations; during this stage, they disperse across host plants, where the following stage–feeding–occurs. Phytophage nutrition and biomass gain are influenced by the host plant's abundance. The population size of each genotype on native and invasive plants is modeled by a separate equation, resulting in six equations for each stage.

Mating in the model is either panmictic (1.1–1.6) or fully assortative (1a.1–1a.6).

$$AA^N_{t+1} = X\left(G\left(AA^N_t\right), G\left(Aa^N_t\right), G\left(aa^N_t\right)\right), \quad (1.1)$$

$$Aa^N_{t+1} = X_1\left(G\left(AA^N_t\right), G\left(Aa^N_t\right), G\left(aa^N_t\right)\right), \quad (1.2)$$

$$aa_{t+1}^{N} = X\left(G\left(aa_t^N\right), G\left(Aa_t^N\right), G\left(AA_t^N\right)\right), \quad (1.3)$$

$$AA_{t+1}^{I} = X\left(G\left(AA_t^I\right), G\left(Aa_t^I\right), G\left(aa_t^I\right)\right), \quad (1.4)$$

$$Aa_{t+1}^{I} = X_1\left(G\left(AA_t^I\right), G\left(Aa_t^I\right), G\left(aa_t^I\right)\right), \quad (1.5)$$

$$aa_{(t+1)}^{I} = X\left(G\left(aa_t^I\right), G\left(Aa_t^I\right), G\left(AA_t^I\right)\right), \quad (1.6)$$

where

$$X(x,y,z) = \frac{(2\cdot x+y)^2}{4\cdot(x+y+z)},$$

$$X_1(x,\ y,\ z) = \frac{(2\cdot x+y)\cdot(2\cdot z+y)}{2\cdot(x+y+z)}.$$

The assortative mating is modeled following Li [1976].

$$AA_{t+1}^{N} = F_1\left(G\left(AA_t^N\right), G\left(Aa_t^N\right), G\left(aa_t^N\right)\right), \quad (1a.1)$$

$$Aa_{t+1}^{N} = F_2\left(G\left(AA_t^N\right), G\left(Aa_t^N\right), G\left(aa_t^N\right)\right), \quad (1a.2)$$

$$aa_{t+1}^{N} = F_3\left(G\left(AA_t^N\right), G\left(Aa_t^N\right), G\left(aa_t^N\right)\right), \quad (1a.3)$$

$$AA_{t+1}^{I} = F_1\left(G\left(AA_t^I\right), G\left(Aa_t^I\right), G\left(aa_t^I\right)\right), \quad (1a.4)$$

$$Aa_{t+1}^{I} = F_2\left(G\left(AA_t^I\right), G\left(Aa_t^I\right), G\left(aa_t^I\right)\right), \quad (1a.5)$$

$$aa_{(t+1)}^{I} = F_3\left(G\left(AA_t^I\right), G\left(Aa_t^I\right), G\left(aa_t^I\right)\right), \quad (1a.6)$$

where

$$F_1(x,y,z) = \frac{\frac{\left(\frac{2\cdot x+y}{2\cdot(x+y+z)}\right)^2}{1-\frac{z}{(x+y+z)}}\cdot(x+y+z)}{\frac{\left(\frac{2\cdot x+y}{2\cdot(x+y+z)}\right)^2}{1-\frac{z}{(x+y+z)}} + 2\cdot\frac{\frac{y^2+2\cdot x\cdot y}{4\cdot(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{\frac{4\cdot y^2}{(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{z}{(x+y+z)}},$$

$$F_2(x,y,z) = \frac{2\cdot\frac{\frac{y^2+2\cdot x\cdot y}{4\cdot(x+y+z)^2}}{1-\frac{z}{(x+y+z)}}\cdot(x+y+z)}{\frac{\left(\frac{2\cdot x+y}{2\cdot(x+y+z)}\right)^2}{1-\frac{z}{(x+y+z)}} + 2\cdot\frac{\frac{y^2+2\cdot x\cdot y}{4\cdot(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{\frac{4\cdot y^2}{(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{z}{(x+y+z)}},$$

$$F_3(x,y,z) = \frac{\left(\frac{\frac{4\cdot y^2}{(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{z}{(x+y+z)}\right)\cdot(x+y+z)}{\frac{\left(\frac{2\cdot x+y}{2\cdot(x+y+z)}\right)^2}{1-\frac{z}{(x+y+z)}} + 2\cdot\frac{\frac{y^2+2\cdot x\cdot y}{4\cdot(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{\frac{4\cdot y^2}{(x+y+z)^2}}{1-\frac{z}{(x+y+z)}} + \frac{z}{(x+y+z)}}.$$

The input to the mating function is the output of a logistic growth function, based on the modified Ricker equation [Ricker, 1954], with growth rate determined by feeding efficiency. This growth function models changes in the relative fitness of a given genotype, which depends on plant abundance, insect feeding efficiency on the plant, and the abundance of other genotypes on the same plant. This logistic growth of fitness can be interpreted in two ways: (1) as correlation of feeding efficiency with fecundity during the mating stage–better-nourished individuals are likely to produce more eggs; (2) as parthenogenetic reproduction on the host plant, followed by a round of sexual reproduction (as seen in aphids) during the mating stage. The Ricker equation was chosen as it is simple yet effective function that allows us to account for the necessary parameters: plant abundance, insect feeding efficiency on the plant, and competition between genotypes, assuming that the environment's carrying capacity is determined by plant abundance.

$$G\left(AA_t^N\right) = AA_t^N\cdot R\left(N, D\left(AA_t^N\right), D\left(Aa_t^N\right), D\left(aa_t^N\right)\right),$$

$$G\left(Aa_t^N\right) = Aa_t^N\cdot R\left(N, D\left(AA_t^N\right), D\left(Aa_t^N\right), D\left(aa_t^N\right)\right),$$

$$G\left(aa_t^N\right) = aa_t^N\cdot R\left(N, D\left(AA_t^N\right), D\left(Aa_t^N\right), D\left(aa_t^N\right)\right),$$

$$G\left(AA_t^I\right) = AA_t^I\cdot R_1\left(I, D\left(AA_t^I\right), D\left(Aa_t^I\right), D\left(aa_t^I\right)\right),$$

$$G\left(Aa_t^I\right) = Aa_t^I\cdot R_1\left(I, D\left(AA_t^I\right), D\left(Aa_t^I\right), D\left(aa_t^I\right)\right),$$

$$G\left(aa_t^I\right) = aa_t^I\cdot R\left(I, D\left(AA_t^I\right), D\left(Aa_t^I\right), D\left(aa_t^I\right)\right),$$

where

$$R(w,x,y,z) = \exp\left(f\cdot w - \frac{x+y+z}{w}\right),$$

$$R_1(w,x,y,z) = \exp\left(f_1\cdot w - \frac{x+y+z}{w}\right).$$

AA_t^N, Aa_t^N, aa_t^N are quantities of wild type, heterozygote and mutant genotypes on native plant species at the time t, respectively; N is abundance of the native plant, which also determines the carrying capacity of the respective herbivore population; AA_t^I, Aa_t^I, aa_t^I are quantities of wild type, heterozygote and mutant genotypes on invasive plant species, respectively; I is abundance of the invasive plant, which also determines the carrying capacity of the respective herbivore population; f is a proxy for feeding efficiency of a phytophage on the plant, for a wild type on invasive plant f_1 is used which is lower than f.

As arguments for the growth function, we use the abundances of genotypes on the host plant, which depend on post-mating dispersal. During this dispersal phase, a minor proportion of phytophages, both wild type and mutant, leave the host plant and migrate to other plant species (2.1–2.6).

$$D\left(AA_t^N\right) = M\left(AA_t^N, AA_t^I\right), \quad (2.1)$$

$$D\left(Aa_t^N\right) = M\left(Aa_t^N, Aa_t^I\right), \quad (2.2)$$

$$D\left(aa_t^N\right) = M\left(aa_t^N, aa_t^I\right), \quad (2.3)$$

$$D\left(AA_t^I\right) = M\left(AA_t^I, AA_t^N\right), \quad (2.4)$$

$$D\left(Aa_t^I\right) = M\left(Aa_t^I, Aa_t^N\right), \quad (2.5)$$

$$D\left(aa_t^I\right) = M\left(aa_t^I, aa_t^N\right), \quad (2.6)$$

where

$$M(x, y) = (1 - m)\cdot x + m\cdot y,$$

m is a proportion of phytohages migrating to the other plant species.

We do not include the down-regulation of plant abundance by phytophages since insects often do not significantly affect plant population dynamics [Crawley, 1989]. Despite this, using the Ricker equation, we assume that the carrying capacity of the environment is determined by the abundance of the plant. Instead, we ran our model with different relative abundances of invasive plant. Initially we also modeled expansion of the invasive plant outcompeting the native relative, but the results were the same as when we considered only minor relative abundance of native plant at the beginning. So we decided to use fixed proportions of host plant species to investigate the effect of the relative abundance of the invasive plant on phytophage dynamics.

Thus, the model has three parameters: 1) difference in exponential growth between wild type and mutant on the invasive plant; 2) rate of migration to the other plant species; 3 relative abundance of the invasive plant.

The initial proportion of a mutant allele on the native plant was modeled in two ways: a) a new mutation appeared only in a low proportion of heterozygotes; b) an old neutral allele which had reached significant prevalence by the genetic drift with equilibrium genotypes distribution since it didn't affect fitness in the absence of the invasive plant. The initial frequencies of all genotypes on the invasive plant were considered to be zero, which corresponds to the absence of phytophages on the invasive plant, which they had not yet detected.

Since mutant allele is beneficial in the presence of invasive plant, it would finally replace the wild type allele after a number of generations, so the question of interest is the rate of its spread. We categorized possible outcomes of models in each phytophage population after a number of generations into three types: the elimination of the wild-type allele (when its frequency dropped below 0.05), the elimination of the mutant allele (when its frequency dropped below 0.05 and wild-type allele predominate with a frequency of more than 0.95), or the coexistence of both phenotypes (when the frequencies of both alleles exceeded 0.05). Different outcomes are possible in populations from different plants, resulting in nine theoretically possible combinations.

To examine the effect of the relative abundance of invasive and native species on a mutant spread, we increased this relative abundance stepwise by 0.01 from 0 to 1. Additionally, we manipulated the growth efficiency of phytophages with the wild phenotype on invasive plants, while keeping the consumption efficiencies of phytophages with the mutant phenotype on native and invasive plants (f) constant and equal to 1.7. Specifically, we varied the fitness cost of wild-type phytophages when feeding on invasive plants, representing how much less effectively they convert invasive plant biomass into their own biomass compared to mutant phytophages. We varied the fitness of wild phenotype phytophages on invasive plants (f_1) from 0 to 1.7 by 0.01, which corresponded to a fitness cost range of 0% to 100%, expressed as $(f - f_1)/f$.

To examine the effect of phytophages migration from one population to the other, we changed its rate in every direction, using three sets of parameter values. In our base model we used a symmetrical migration rate: 1% of phytophages move from the native to the invasive plant and vice versa. Also, we tested increased symmetrical migration rate (10% in both directions) and preference of the mutant phenotype for invasive species (2a.1–2a.6): mutants migrate from the native to the invasive plant at a 10% rate and from the invasive plant to the native at 1% rate, whereas wild phenotypes migrate in both directions at 1% rate.

$$D\left(AA_t^N\right) = M\left(G\left(AA_t^N\right), G\left(AA_t^I\right)\right), \quad (2a.1)$$

$$D\left(Aa_t^N\right) = M\left(G\left(Aa_t^N\right), G\left(Aa_t^I\right)\right), \quad (2a.2)$$

$$D\left(aa_t^N\right) = M_1\left(G\left(aa_t^N\right), G\left(aa_t^I\right)\right), \quad (2a.3)$$

$$D\left(AA_t^I\right) = M\left(G\left(AA_t^I\right), G\left(AA_t^N\right)\right), \quad (2a.4)$$

$$D\left(Aa_t^I\right) = M\left(G\left(Aa_t^I\right), G\left(Aa_t^N\right)\right), \quad (2a.5)$$

$$D\left(aa_t^I\right) = M_2\left(G\left(aa_t^I\right), G\left(aa_t^N\right)\right), \quad (2a.6)$$

where

$$M(x, y) = (1 - m) \cdot x + m \cdot y,$$
$$M_1(x, y) = (1 - m_1) \cdot x + m \cdot y,$$
$$M_2(x, y) = (1 - m) \cdot x + m_1 \cdot y,$$

m_1 is higher than m and reflects a higher migration rate of mutant phenotype to the invasive plant.

We constructed diagrams to visualize the dependency of simulation results on the relative abundance of native and invasive plants, as well as the fitness cost of wild-type phytophages when feeding on invasive plants.

In our model, the extinction of phytophage populations on one of the plants is impossible, since they are replenished through migration. However, the successful colonization of the invasive species depends on the propagation of the mutant allele. Without the spread of the mutant allele, natural selection would favor mechanisms that prevent the transition of wild type phytophages to the invasive plant. That is why we considered the probability of fixation of the mutant allele after different time intervals in populations under different parameters.

Results

Panmictic mating. The dynamics of genotype frequencies shows that spread of the mutant allele in the majority of parameter sets is faster on invasive than on native plant (Fig. 2). Under these specific model parameters (relative abundance of the invasive plant to the native is 1 : 1; the wild phenotype fitness on the invasive plant is 15% lower than on the native plant; initial Hardy-Weinberg equilibrium in the population with mutant allele frequency 0.1), we observed the coexistence of the mutant and wild phenotypes in both populations during the first 150 generations. However, after 200 generations, the wild-type allele was eliminated in the population living on the invasive plant, while both alleles remained present in the population living on the native plant. Remarkably, after 500 generations, the mutant allele became fixed in both populations, indicating its advantage over the wild-type allele.

In the case of a newly arisen mutation (the initial frequency of mutant homozygotes is zero and heterozygotes are extremely low), our simulations show that after 200 generations, the mutant allele can reach a frequency greater than 0.05 only in the population inhabiting the invasive species (Fig. 3, lower row). This outcome is observed when the relative abundance of the invasive plant is very high, and the wild-type phenotype experiences a significant fitness cost when feeding on invasive plants. However, after 500 generations, the mutant allele can achieve enough frequency not only on the invasive plant but also on the native plant, potentially displacing the wild-type allele from both plant types. To attain these states, a high relative abundance of the invasive plant and substantial differences in fitness between the two phenotypes are still required. When there are very large differences in fitness between the mutant and wild-type alleles, and the abundance of the invasive plant is not sufficiently high, the spread of the mutant allele slows down, apparently due to the limited environmental capacity, which constrains rapid population growth.

If the genotype frequencies in a native plant population initially follow Hardy-Weinberg equilibrium, three possible scenarios can occur. When there is minimal fitness difference between the phenotypes, or when the relative abundance of invasive plants is low, both phenotypes coexist on both plant species. Only in the narrow range of nearly identical abundances of both plant species the wild-type allele is eliminated on the invasive plant while both phenotypes still coexist on the native plant. Lastly,

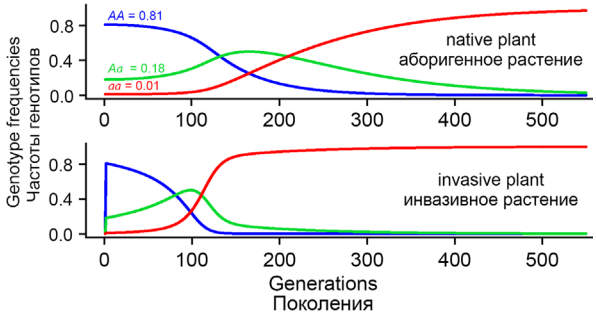

Fig. 2. Dynamics of allele frequencies over 500 generations. Relative abundance of the invasive plant is 0.5. The wild phenotype fitness on the invasive plant is 1.45 (15% fitness loss), the fitness of the mutant on both invasive and native, as well as the feeding efficiency of the wild phenotype on the native plants, is 1.7. On the invasive plant initial genotype frequencies are zero.

Рис. 2. Динамика частот аллелей за 500 поколений. Относительная доля инвазивного растения составляет 0.5. Эффективность питания дикого фенотипа на инвазивном растении составляет 1.45 (потеря приспособленности на 15%), эффективность питания мутанта на инвазивном и аборигенном растениях, а также эффективность питания дикого фенотипа на аборигенном растении составляют 1.7. На инвазивном растении начальные частоты генотипов равны нулю.

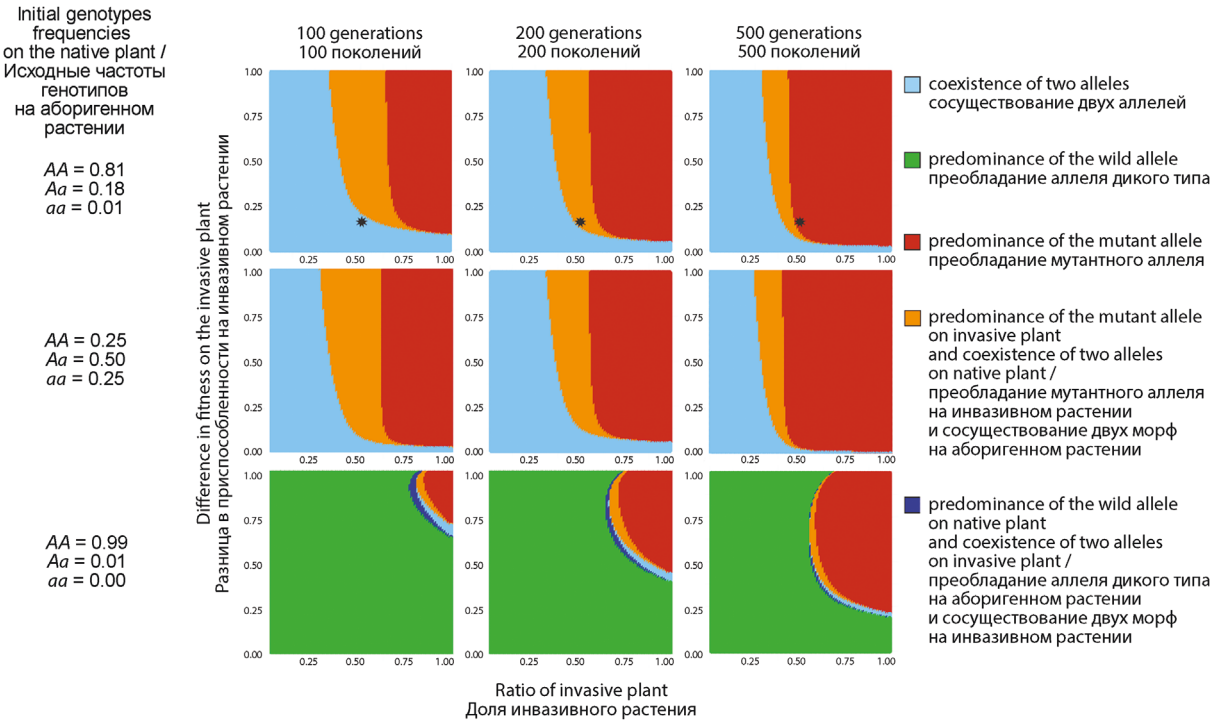

Fig. 3. Influence of the invasive plant's relative abundance and the decrease of wild type phenotype feeding efficiency on the invasive plant relative to the feeding efficiency on the native plant on the ratio of the allele frequencies after 100, 200, and 500 generations at different initial genotype frequencies (the upper and the middle rows correspond to different variants of the Hardy-Weinberg equilibrium, the lower row corresponds to a rare mutation in the heterozygote). On the invasive plant initial genotype frequencies are zero. The feeding efficiency of the mutant on both invasive and native plants, as well as the feeding efficiency of the wild phenotype on the native plant, is 1.7. Asterisks indicate the states reflected in Figure 2.

Рис. 3. Влияние доли инвазивного растения и уменьшения эффективности питания дикого фенотипа в сравнении с эффективностью питания на аборигенном растении на соотношение частот аллелей после 100, 200 и 500 поколений при различных начальных частотах генотипов (верхние два ряда соответствуют различным вариантам равновесия Харди – Вайнберга, нижний ряд соответствует редкой мутации в гетерозиготе). На инвазивном растении начальные частоты генотипов равны нулю. Эффективность питания мутанта на инвазивном и аборигенном растениях, а также эффективность питания дикого фенотипа на аборигенном растении составляют 1.7. Звездочками отмечены параметры, отраженные на рисунке 2.

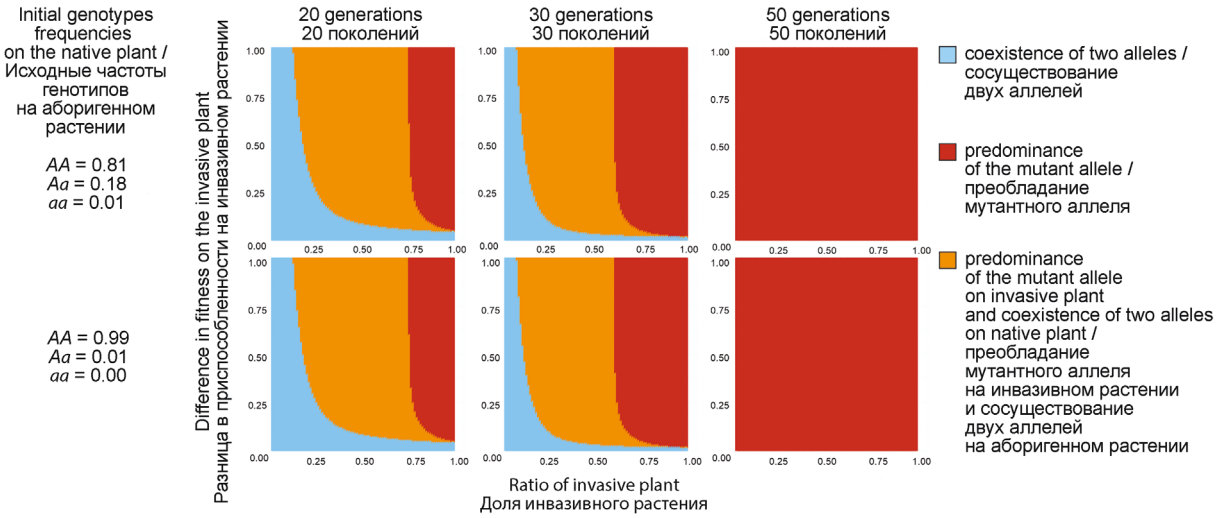

Fig. 4. Influence of the invasive plant's relative abundance and the decrease of wild type phenotype feeding efficiency on the invasive plant relative to the feeding efficiency on the native plant on the ratio of the allele frequencies for a model with assortative mating after 20, 30, and 50 generations at different initial genotype frequencies (the upper row corresponds to the Hardy-Weinberg equilibrium, the lower row corresponds to a rare mutation in the heterozygote). On the invasive plant initial genotype frequencies are zero. The feeding efficiency of the mutant on both invasive and native, as well as the feeding efficiency of the wild phenotype on the native plant, is 1.7.

Рис. 4. Влияние доли инвазивного растения и снижения эффективности питания дикого фенотипа на инвазивном растении в сравнении с эффективностью питания на аборигенном растении на соотношение частот аллелей для модели с ассортативным скрещиванием после 20, 30 и 50 поколений при различных начальных частотах генотипов (верхний ряд соответствует равновесию Харди – Вайнберга, нижний ряд соответствует редкой мутации в гетерозиготе). На инвазивном растении начальные частоты генотипов равны нулю. Эффективность питания мутанта на инвазивном и аборигенном растениях, а также эффективность питания дикого фенотипа на аборигенном растении составляет 1.7.

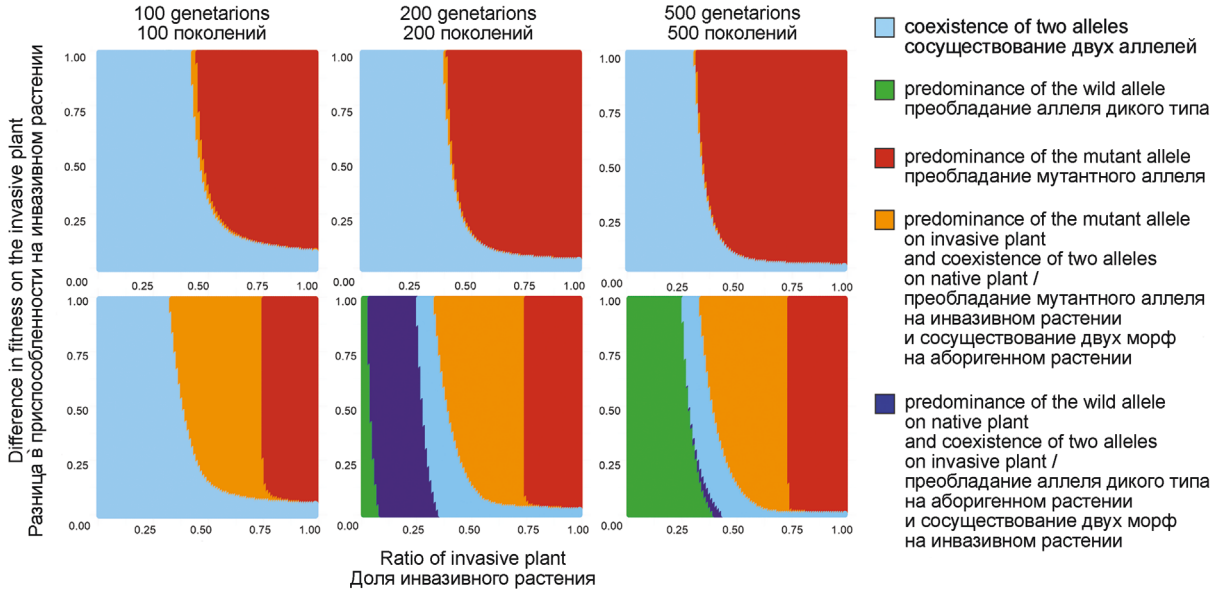

Fig. 5. Influence of the invasive plant's relative abundance and the decrease of wild type phenotype feeding efficiency on the invasive plant relative to the feeding efficiency of the native plant on the ratio of the allele frequencies for a model with panmictic mating after 100, 200, and 500 generations at different initial genotype frequencies with different modes of mutant migration (the upper row corresponds to 10% of the mutant population changing host plant, the lower row corresponds to 10% of the mutant population switching to the invasive plant from the native, and 1% from invasive to native). Initial genotype frequencies on the native plant are $AA = 0.81$, $Aa = 0.18$, $aa = 0.01$. On the invasive plant initial genotype frequencies are zero. The feeding efficiency of the mutant on both invasive and native plants, as well as the feeding efficiency of the wild phenotype on the native plant, is 1.7.

Рис. 5. Влияние доли инвазивного растения и снижения эффективности питания дикого фенотипа на инвазивном растении в сравнении с эффективностью питания на аборигенном на соотношение частот аллелей для модели с панмиктическим скрещиванием после 100, 200 и 500 поколений при различных начальных частотах генотипов с разными режимами миграции мутантов (верхний ряд соответствует миграции в каждом поколении 10% мутантной популяции с инвазивного на аборигенный и с аборигенного на инвазивный вид растений, нижний ряд соответствует симметричной миграции 10% мутантной популяции на инвазивный вид с аборигенного, и 1% с инвазивного на аборигенный). Начальные частоты генотипов на аборигенном растении: $AA = 0.81$, $Aa = 0.18$, $aa = 0.01$. На инвазивном растении начальные частоты генотипов равны нулю. Эффективность питания мутанта на инвазивном и аборигенном растениях, а также эффективность питания дикого фенотипа на аборигенном растении составляет 1.7.

the elimination of the wild-type allele can occur on both plant types when the frequency of the invasive plant exceeds 50%, almost irrespective of fitness difference. The probability of the third scenario tends to increase with the number of generations.

Assortative mating. In the case of fully assortative mating, fixation of the mutant allele occurs across the entire range under consideration within just 50 generations. Assortativity plays a crucial role in accelerating the emergence of mutant homozygotes and the spread of the mutant allele within both populations when a new mutation arises (Fig. 4, lower row). The behavior of the model under assortative mating becomes independent of the initial state of the populations as early as 20 generations – initial equilibrium frequencies and a newly arisen mutation lead to identical results.

Rate of migration to the other plant species. The spread of the mutant allele accelerates with increased gene flow between the two populations, i.e. a higher migration rate between two plant species (Fig. 5, upper row). This nearly eliminates the scenario in which the wild-type allele is eradicated on the invasive plant while both phenotypes still coexist on the native plant. In other words, if the mutant allele becomes fixed in the population on the invasive plant, it quickly establishes itself in the population on the native plant as well, facilitated by gene flow.

And if there is asymmetric migration, where mutant phytophages preferentially choose invasive plants distinct outcomes can be observed depending on the relative abundance of the invasive plant (Fig. 5, lower row). At high relative abundances of the invasive plant, the mutant allele reaches fixation on both plant species. When the relative abundances of invasive and native plants are comparable, the mutant allele prevails in the phytophage population on invasive species, while both alleles coexist on the native species phytophage populations. If the relative abundance of the invasive plant is low, after 100 generations, both alleles coexist in both populations. However, over time, the wild allele becomes prevalent first in the phytophage population on the native species, while both alleles coexist in the phytophage population on the invasive species, but then in both phytophage populations.

Discussion

In our model, the presence of the mutant allele does not reduce the fitness of phytophages on a native plant, so it actually enables the expansion of the trophic niche, representing a conditionally beneficial mutation. However, the spread of this advantageous mutation still depends on several conditions. So, we can discuss the role of the studied factors in terms of their effects on the tempo of the spread of the mutant allele.

Time. First of all, in most simulated conditions, the process of mutation fixation typically requires a large number of generations. Even 500 generations may not be sufficient for fixation of a newly emerged mutation within a panmictic population. The generation time of insects varies considerably among species, with many insects exhibiting univoltine or semivoltine life cycles [Numata, Shintani, 2023], i.e. with generation time of one year or even less, respectively. Even in multivoltine species, the expected number of generations per year is generally limited to no more than five in temperate climates [Buckley et al., 2017]. Considering these factors, it becomes apparent that it can take approximately 100 years for the spread of a pre-existing mutation, and the appearance of such a mutation itself can require a certain amount of time. This helps explain the observed period of low pressure of phytophages, which can extend from 50 to 200 years [Hawkes, 2007].

It should be noted that these studies show an increase in diversity or abundance of native insects on invasive plants, but this does not necessarily mean that these insects have already adapted to this plant [Gassmann et al., 2006].

Furthermore, it is important to note that host shift to a new plant can influence the number of generations per year. For instance, *Choristoneura rosaceana* (Harris, 1841) (Lepidoptera: Tortricidae) exhibits a univoltine or bivoltine cycle depending on the host plant, and a low-quality diet can favor diapause induction, leading to a univoltine life cycle instead of a bivoltine one [Hunter, McNeil, 1997]. This obviously should increase the spread of mutation, favoring use of invasive plant.

High relative abundance of the invasive plant. Despite the fact that in our model a mutation that enhances the ability to feed on an invasive species does not reduce fitness on a native plant, the spread of the mutant form is slow at low relative abundance of the invasive plant, particularly within the native plant population. Our modeling scenarios consistently demonstrate that replacing the wild type allele requires the invasive plant to be at least as abundant as the native species. Our model assumes constant abundances of the native and invasive species, but in reality, it is highly plausible for the invasive species to spread extensively over time [Petrosyan et al., 2023], potentially resulting in the complete replacement of native species [Vasilyeva, Papchenkov, 2011; Vervoort, Jacquemart, 2012]. Our model allows us to suggest that host shift to alien species is possible only after it has effectively outcompeted the native relative.

Differences in fitness between mutant and wild type alleles. In our model, we assume that the wild phenotype has a lower ability to consume the invasive species, which is a plausible assumption considering that native insects are generally not adapted to novel plant chemistry [Cappuccino, Arnason, 2006; Lind, Parker, 2010]. Most forest insects perform worse on novel host trees [Bertheau et al., 2010]. However, in our model, we specifically focus on the appearance or distribution of the mutant phenotype that is capable of consuming both the invasive and native species equally. We consider the efficiency of consumption as a fitness component, as it directly affects the rate of population growth. A decrease in fitness when transitioning to a new plant can manifest as increased mortality [Faccoli, 2007; Kirichenko et al., 2008], decreased reproduction rate [Roininen, Tahvanainen, 1989], or impaired development [Keena, 2003]. The coefficient 'f' used in our model can encompass all these fitness effects. Thus, the difference in the efficiency of invasive species consumption between the mutant and the wild phenotype determines the utility of the mutation and consequently influences its rate of spread.

According to our simulation results, the difference in feeding efficiency has a lesser impact on the spread of

the mutant allele compared to the relative abundance of the invasive species. However, when the difference in the efficiency of consumption of the invasive species is minimal, the spread of the mutation is typically hindered.

Such slight decreases in fitness are more characteristic of polyphagous insects, while specialized insects may experience more dramatic changes in fitness upon host shift to a new plant. In a meta-analysis of forest insect fitness on novel and ancient host tree species, it was found that the difference in fitness between ancient and new host trees was significant for monophagous insects, moderate for oligophagous insects, and non-significant for polyphagous insects [Bertheau et al., 2010]. On the other hand, it is unlikely that a single mutation can fully restore fitness on an invasive plant to the level observed on a native plant, especially in cases with substantial differences in fitness between the two host plants. However, there are examples in nature when a single mutation has led to significant adaptations. For instance, a single amino-acid substitution in the Na^+, K^+-ATPase of the *Danaus plexippus* (Linnaeus, 1758) confers insensitivity to the cardenolide ouabain found in one of its host plants [Holzinger, Wink, 1996]. Another example is the adaptation of the polyphagous aphid *Myzus persicae* (Sulzer, 1776) to tobacco due to overexpression of CYP6CY3, resulting from the expansion of a dinucleotide microsatellite in the promoter region and a recent gene amplification, which arose as a recent, single evolutionary event [Bass et al., 2013].

Initial genotype frequency. The initial distribution of genotype frequencies has the strongest influence on the simulation results. In our model, the rapid and successful spread of a mutant allele is feasible only if it has already attained a substantial abundance in the initial population and the respective locus is in Hardy-Weinberg equilibrium, as the mutant allele does not affect fitness on the native plant and can freely spread within that population.

There are empirical examples demonstrating the presence of pseudo-neutral variability in natural populations, where individuals carry deleterious mutations with habitat-specific fitness effects [Kreslavsky-Smirnov, 1987; Kreslavsky, 1994]. One well-studied example is *Lochmaea capreae* (Linnaeus, 1758), where a particular allele causes mortality in homozygotes living on birch (Betula) but develops normally on willows (Salix). This allele does not confer any adaptive advantage on willow. Homozygotes of the alternative allele and heterozygotes, on the other hand, can develop successfully on both birch and willow [Kreslavsky, 1994].

Theoretical models suggest that loci that impact fitness in one habitat while being neutral or nearly neutral in others can potentially contribute to sympatric speciation [Kreslavsky, 1994; Kawecki, 1997]. However, in our model, we did not observe a situation where one allele became fixed on one plant while the other allele became fixed on the other plant. This outcome is explained by the gene flow between populations and the lack of reproductive barriers in our model.

Assortative mating. Assortative mating can facilitate the rapid spread of mutant alleles within a population [Parsons, 1962]. In our model, we used fully assortative mating, which significantly accelerated the spread of the mutant allele; however, such strict assortativity is rarely found in nature and more often there is just increased probability of mating between individuals with similar traits. Moreover, there is no reason to assume the immediate emergence and association of such mutant alleles with assortative mating ([Gavrilets, 2004]; but see alternative point of view in Servedio et al. [2011]). Nonetheless, assortativity can arise as a result of spatial factors, such as similar habitat preferences that lead to mating occurring on the same host plant [Edelaar et al., 2008].

In our model, the gene flow between populations of native and invasive plants was constant, and there was no situation leading to divergence, when one phenotype is widespread on one plant while the other – on the other plant. However, in reality, host shift to a new plant can have additional effects, such as altering the timing of reproduction, which can in turn reduce gene flow between populations and potentially lead to speciation [Forbes et al., 2017].

Behavioural adaptations. Higher migration rates between two plant species can enhance the spread of mutant alleles. If the migrations are asymmetric, with a higher migration rate of the mutant phenotype to the invasive species, it can be interpreted as a behavioral adaptation to the preferred plant. For successful host shifts, behavioral adaptations rather than physiological adaptations are often necessary [Bernays, Chapman, 1994].

A notable example is the host shift of *Ophraella notulata* (Fabricius, 1801) (Chrysomelidae) to a novel host plant, Iva frutescens (Asteraceae). This shift was facilitated by changes in behavior without an increase in the physiological capacity to utilize I. frutescens, despite it being a less digestible plant compared to the ancestral host, Ambrosia artemisiifolia [Gassmann et al., 2006].

Conclusion

Our model highlights several key factors that influence the rate of spread of conditionally beneficial mutant alleles allowing the expansion of the trophic niche in the context of host shift to the invasive plant.

The timing of reproduction and generation time of insects play significant roles in the spread of mutations. The process of mutation fixation typically requires a substantial number of generations. The relative abundance of the invasive plant is another crucial factor. Our modeling scenarios consistently show that displacing the wild type allele requires the invasive plant to be at least as abundant as the native species. The initial distribution of genotype frequencies strongly influences simulation results. For rapid and successful spread, the mutant allele needs to have already attained appreciable abundance in the initial population. Assortative mating can facilitate the spread of mutant alleles, although the immediate emergence and association of such alleles with assortative mating doesn't seem to be very plausible assumption.

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