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ORIGINAL PAPER

Diploid versus haploid models of neutral speciation

David M. Schneider[1] · **Elizabeth M. Baptestini**[1] · **Marcus A. M. de Aguiar**[1]

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Abstract Neutral models of speciation based on isolation by distance and assortative mating, termed topopatric, have shown to be successful in describing abundance distributions and species–area relationships. Previous works have considered this type of process in the context of haploid genomes. Here we discuss the implementation of two schemes of dominance to analyze the effects of diploidy: a complete dominance model in which one allele dominates over the other and a perfect codominant model in which heterozygous genotypes give rise to a third phenotype. In the case of complete dominance, we observe that speciation requires stronger spatial inbreeding in comparison to the haploid model. For perfect codominance, instead, speciation demands stronger genetic assortativeness. Nevertheless, once speciation is established, the three models predict the same abundance distributions even at the quantitative level, revealing the robustness of the original mechanism to describe biodiversity features.

Keywords Models of dominance · Assortative mating · Individual-based simulations

1 Introduction

A key problem in evolutionary biology is to understand the mechanisms that drive speciation [1–3]. Although natural selection is the main driver of species changes, recently developed theories based on drift, migrations, and statistical fluctuations of the population size without selection, called neutral theories, proved to reproduce successfully the observed abundance distributions, which exhibit remarkable universal features [4–7].

Among the different mechanisms of speciation, allopatric speciation is considered the dominant form [8]. This process assumes that geographic separation of subpopulations by natural barriers is responsible for blocking or drastically reducing the gene flow between the

✉ David M. Schneider
schneide@ifi.unicamp.br

[1] Instituto de Física ‘Gleb Wataghin’, Universidade Estadual de Campinas, Unicamp, 13083-859 Campinas, SP, Brazil

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separated groups. As a consequence, the groups evolve independently either by selection or drift, acquiring incompatibilities leading to reproductive isolation. Sympatric speciation, on the other hand, is triggered by ecological interactions taking place in a single spatial domain and even in the same niche [9]. A proposed mechanism is the coupling between ecological and mating traits, which may lead to disruptive selection and ultimately to speciation [10–13].

A neutral mechanism of speciation relying on isolation by distance without geographic barriers or ecological interactions was recently demonstrated [14, 15]. The process, termed topopatric speciation, was shown to describe the universal features observed in abundance distributions and species–area relationships. In this context, assortative mating driven by spatial and genetic distances is sufficient to promote speciation [14, 16].

The formation of species in the topopatric model resembles a thermodynamic phase transition, in the course of which symmetry is broken, leading to the formation of quasi-homogeneous domains. Each domain is a cluster in the genetic space, a species, although the population remains homogeneously distributed in the physical space. Because the initial population consists of genetically identical individuals, the clusters in the genetic space branch from the initial point representing the whole population. At the same time, this process gives rise to the domains in the physical space, where species occupy adjacent areas but keep the overall population density constant. These structures appear as a result of the high diversity generated by mutations and the constraints on reproduction imposed by local mating and assortativity.

Other important processes can lead to pattern formation, such as Turing instability, caused by the diffusion of organisms. Cluster formation in populations of diffusing individuals was studied by Fuentes et al. [17]. Meyer et al. (1996) studied, analytically, a model for the dynamics of populations and epidemics that included birth and death processes in a system of independently diffusing individuals [18]. They observed that clustering in physical space is caused by spatial asymmetry between the birth and death processes; new organisms always appear near their parents, while death picks off individuals regardless of their location in space. Young et al. (2001) proposed a possible mechanism of clustering in which organisms such as plankton die and give birth with equal probabilities [19]. Numerical simulations have demonstrated that initially homogeneous populations organize in clusters, even in the absence of any interaction. Exact calculations of this model revealed that for dimensions $d \leq 2$, even though average concentrations of individuals remain constant, fluctuations can diverge as a function of time, giving rise for particular realizations of the model to cluster formation [20]. More recently, Lawson and Jensen (2007) analyzed a diffusive birth/death process with mutations employing field theory formalism and using dimensional analysis arguments to describe evolution as a super-Brownian motion for a continuous phenotype in the infinite population limit [21]. Their main conclusions established a difference between the evolution of phenotypes, for which strong local clustering is observed, and genotypes, for which distributions are more dispersed.

The topopatric model considers finite populations with finite genomes in a spatially uniform environment. Speciation in this case is not driven by diffusion but by a tension created by diversity, generated by mutations and local assortative mating, which requires individuals to be sufficiently similar and nearby to a mate. A comprehensive theoretical understanding of the topopatric model, revealing the relative and quantitative importance of the different parameters, is still missing. Some efforts have been made in that direction through standard non-linear dynamics techniques and network dynamics (see section II). These investigations allowed quantification of the effects of both genetic assortative mating

and spatial inbreeding on the critical mutation rate needed to promote sufficient diversity to give rise to speciation.

The demonstration that speciation can happen even in homogeneous environments also suggests that speciation can be accelerated by the presence of partial barriers, selection, and gene interactions. Ring species are interesting examples of such, where geography plays a crucial role in physically shaping the ring but does not block dispersal or gene flow along the ring [22–25]. A recent study of cluster formation with similar interactions was performed by Scott et al. (2013) using a sympatric model [26]. In this model, phenotypes are represented by two continuous traits and the initial population is assumed to be uniformly distributed in the phenotypic space, i.e., to have high diversity. Cluster formation also occurs in phenotypic space as a consequence of assortative mating, but does not represent speciation, since the initial population is not a single species.

The neutral speciation model developed by de Aguiar et al. [14] relies on several limiting assumptions that should be relaxed for comparison with real ecologies. In particular, the model assumes individuals to be haploid and hermaphroditic. In a recent article, Baptestini et al. (2013) considered haploid males and females explicitly and studied the effects of sex separation in the process of speciation [27]. They found that for a particular carrying capacity, speciation occurs under similar conditions in the sex-separated case and in the hermaphroditic case. However, the number of species with sex separation decreases in comparison with the hermaphrodite model. Evolution in this case results in fewer but more populous and stable species.

In the present paper we analyze to what extent the predictions of the original model hold when individuals are endowed with diploid genomes. To account for the effects of diploidy and its consequences on neutral speciation, we consider two different scenarios, corresponding to the complete dominance of one of the alleles and to a perfect codominance, where the heterozygous genotypes display a third phenotype. We show that, in comparison to the haploid case, speciation requires stronger spatial inbreeding when dominance is complete and stronger genetic assortativity when dominance is incomplete. Surprisingly, for the parameter region where speciation happens for the three models (haploid, diploid with complete dominance, and diploid with perfect codominance) the process leads to very similar outcomes, with the same number of species formed and the same abundance distributions.

2 The model

In this section, we describe the individual-based model employed to simulate the neutral evolution of spatially distributed populations. This model represents an extension of the de Aguiar model (2009) for the case of diploid genomes.

2.1 Physical and genetic spaces

We consider an initial population of N individuals randomly distributed over a homogeneous environment, represented by a rectangular geographical domain subdivided into $L \times L$ regions with periodic boundary conditions. The number of individuals is fixed throughout the simulations, corresponding to an underlying fixed ecological capacity. Each individual in the population is located at a position (x, y) in the physical space, with $1 \leq x \leq L$, $1 \leq y \leq L$, and its genome is represented by one or two binary strings of B loci, for the haploid or the diploid

case, respectively. The k_{th} locus of the i_{th} individual is denoted by σ_k^i in the haploid case and by $\sigma^i_{k,n}$ for the diploid case, where n=1, 2 designates each of the parental copies. From the genotypes σ we define the phenotypes φ according to three different rules (Fig. 1). As the general scheme means a multiple trait model with each gene independently coding for one specific trait, for haploid individuals the representation of phenotypes and genotypes coincide, namely:

$$\phi\left(\sigma_k^i\right) = \sigma_k^i. \tag{1}$$

For diploid individuals, we distinguish between two dominance models. In the first case, we consider a complete dominance scheme in which the allele 0 is expressed over the allele 1:

$$\phi\left(\sigma_{k,1}^i, \sigma_{k,2}^i\right) = \sigma_{k,1}^i \times \sigma_{k,2}^i \tag{2}$$

and in the second case we consider a perfect codominance scheme in which neither allele is dominant over the other. Instead, the heterozygous genotype expresses a third phenotype representing an intermediate trait between the two homozygotes:

$$\phi\left(\sigma_{k,1}^i, \sigma_{k,2}^i\right) = \frac{1}{2}\left(\sigma_{k,1}^i + \sigma_{k,2}^i\right). \tag{3}$$

The key ingredient of the model is the introduction of assortative mating based on two critical mating distances [14]: one in physical space, and one in genetic space. In physical space, an individual can mate only with others living in the neighborhood of its location determined by the spatial mating distance S. This type of spatial mating restriction was considered by Wright [28, 29] and Kimura and Weiss [30] and may lead to significant genetic differences between geographically distant individuals of the same species. The effect of the spatial constraint on the outcome of speciation depends on the genome size B but in the limit of infinitely large genome speciation becomes possible even if the spatial constraint is removed [31]. In addition, it is assumed that individuals do not mate with others who have dramatically different phenotypes. This restriction can be considered a form of genetic assortative mating, as it has the same effect as behavioral mating preference. Assortative mating is a key ingredient of several models of sympatric

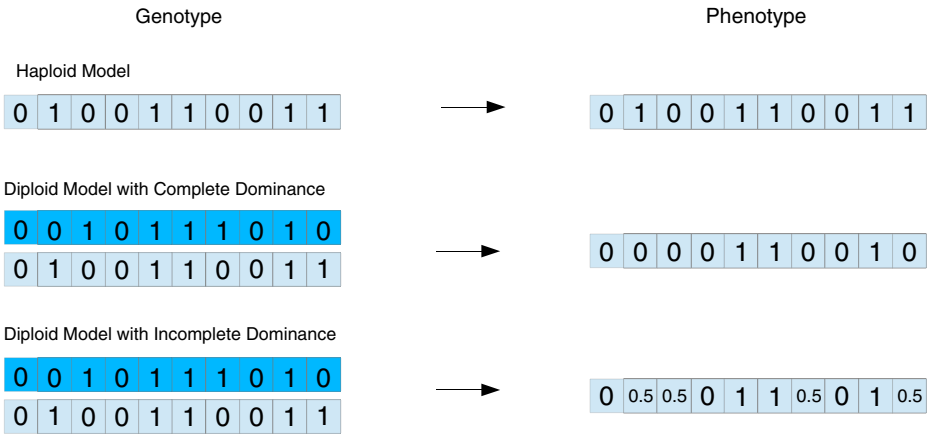

Fig. 1 Dominance rules relating genotypes to phenotypes

speciation [32–34] and empirical evidence of its role in speciation has been discussed [1, 35]. Reproductive isolation caused by this mechanism can be considered a multilocus generalization of the Bateson–Dobzhansky–Muller model in which individuals accumulate genetic incompatibilities [1]. As shown previously [14], genetic restriction on mating alone does not lead to speciation but it does keep different existing species genetically isolated from one another. Non-linear analysis of the underlying dynamics of the topopatric process of speciation was recently undertaken to give a quantitative description on how the genome size, the parameter controlling genetic assortative mating, and the mutation rate interplay [36–38]. Interestingly, the mechanism has a straightforward interpretation in terms of the 'error catastrophe' predicted by Eigen's quasi-species theory [39]. The effects of the spatial constraints are now being investigated by means of network dynamics techniques adapted to structured networks [40].

To impose genetic proximity on mating organisms [41, 42], we restrict the number of distinct traits to be no more than the phenotypic mating distance G. The phenotypic distance between individuals i and j is measured by the Hamming distances:

$$d(i,j) = \sum_k \left|\varphi\left(\sigma_k^i\right) - \varphi\left(\sigma_k^j\right)\right| = \sum_k \left|\sigma_k^i - \sigma_k^j\right| \tag{4}$$

for haploid individuals and

$$d(i,j) = \sum \left|\varphi\left(\sigma_{k,1}^i, \sigma_{k,2}^i\right) - \varphi\left(\sigma_{k,1}^j, \sigma_{k,2}^j\right)\right| \tag{5}$$

for diploid individuals. In both cases, mating is allowed if $d(i, j) \leq G$.

2.2 Time evolution

The evolution of each generation is divided into τ time steps, in which a single individual reproduces. After one generation, the entire population has been replaced. We start with the i_{th} individual that attempts to reproduce and is successful with a probability $1-Q$ (Q being a parameter modeling the attempts to mate with incompatible individuals or failure to find a mate in the mating season) and identify all individuals in its spatial mating neighborhood, specified by the radius S. From these individuals, those whose phenotypic distance d is less or equal to G are pre-selected.

From this sub-group, an individual j is randomly chosen to be the mate. There is no mating preference within the sub-group of compatible individuals, including no restriction on mating between relatives. The genome of the offspring is obtained by recombination of the genomes of the two individuals as follows (see Figs. 2 and 3): a random position k in the parental genomes is chosen to cross over and new genomes are produced; one of these genomes is taken with equal probability as the offspring's genome, which is further subjected to mutations at a rate μ per gene. The offspring is placed at the position (x_i, y_i), or moves to a neighboring site with probability D. After reproduction, the originating parent expires and the label i is assigned to the offspring. More than one individual is allowed per site, although the average population density is typically less than 1.

Reproduction of the i_{th} individual can fail with probability Q. In this case, the individual i dies without leaving a descendant and another individual, chosen at random within the spatial

Fig. 2 Mating scheme employed in the haploid model

neighborhood of radius S, reproduces instead of it. The offspring generated takes the label i and is placed at the position of the original individual or somewhere in its neighborhood with probability D.

During the process of selecting a mate with the restriction imposed by spatial and phenotypic proximity, it is possible that the number of mates available to the reproducing individual becomes very small, possibly zero, preventing it from finding a mate. To avoid this situation, we introduce a parameter P, representing the *minimum number of potential mates*. Given S and G, if the number of mates available to the individual is smaller than P, we relax the spatial constraint by increasing S→S+1 for the present mating season only, i.e., the individual increases the search area in order to have more choice. If the number of available mates is still smaller than P, the process is repeated until S increases up to 10 units, with no cost to the individual. If the number of mates is still smaller than P, the organism does not reproduce and a neighbor is picked at random to reproduce in its turn.

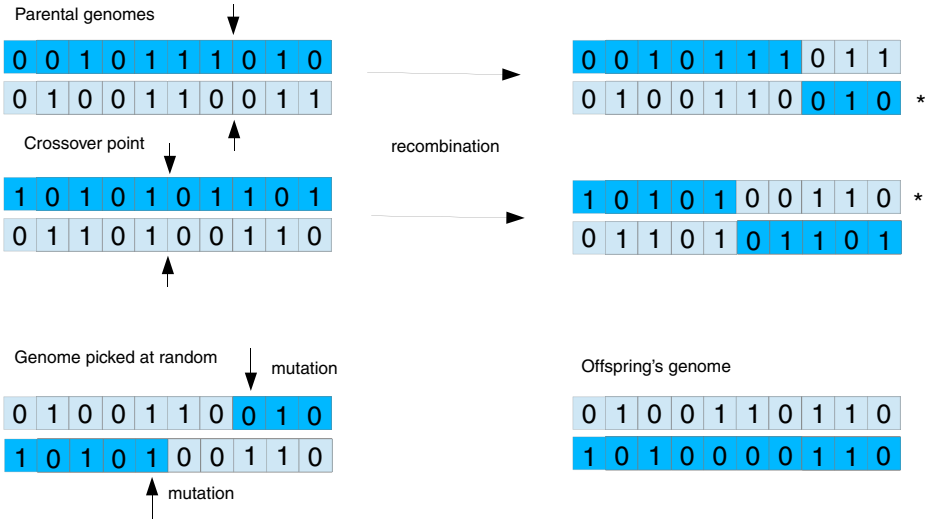

Fig. 3 Mating scheme employed in the diploid models

2.3 Species

For the purposes of our simulations, a species is defined as a group of individuals related by potential gene flow. This is equivalent to Mayr's Biological Species Concept (BSC) that states that "species are groups of interbreeding natural populations that are reproductively isolated from other such groups" [1, 43]. Notice that this definition does not require all individuals of the species to be able to mate with all others; there might exist individuals in the population that can be conspecific while also being incompatible, as long as they can exchange genes indirectly through other conspecifics, as in the case of ring species. For example, three individuals i, j, and k whose genetic distances satisfy $d(i, j) < G$, $d(j, k) < G$ but $d(i, k) > G$ belong to the same species.

In order to classify the individuals in the population into species, the following algorithm is applied: we start with individual number 1 (arbitrarily chosen) and assign it to the first species, named as species 1. We then collect all other individuals whose phenotypic distance to individual 1 is less than G and assign them to species 1. For each of these newly collected individuals in species 1, we check whether there are unassigned individuals whose phenotypic distance to them is less than G. The individuals satisfying this condition are assigned to species 1 as well. We take then the new individuals of species 1 and check again whether there are unassigned individuals whose phenotypic distance is less than G. The individuals satisfying this condition are also added to species 1 and so on. Once we find that no more individuals are added, species 1 is complete. It is a cohesive group that is genetically isolated from the unassigned individuals. If there are no unassigned individuals, there is only one species. Otherwise, we take one unassigned individual and assign it to the second species, named as species 2 and repeat the procedure. It is straightforward to prove that the species obtained in this way do not depend on which individuals are chosen. Notice that the only criterion used to define species is the phenotype similarity. No information about the spatial location of the individuals is taken into account.

3 Methods and results

We performed simulations of the models for different values of the parameters S and G ($4 \leq S \leq 8$, with $\Delta S = 1$ and $8 \leq G \leq 48$, with $\Delta G = 4$). For each S and G we ran the programs for 6000 time steps and used the last 4000 steps to calculate the corresponding ANS (Average Number of Species). Populations consist of $N = 2000$ individuals randomly placed on a 128×128 grid (geographical domain). The mutation rate, dispersion probability, and *minimum number of potential mates* P are taken from [14], corresponding to $\mu = 0.001$, $D = 0.01$, and $P = 4$, respectively. The parameter Q is set to 0.3. Initial populations consist of identical individuals, having genomes composed by 0's at all loci (genome size: $B = 125$). In the haploid model, this applies to the single copy of the genome and in the diploid models to both parental copies. A typical snapshot of the population after species were formed is displayed in Fig. 4.

To compare the outcomes of the two dominance models with the haploid model, we represent the ANS in a bidimensional space having the spatial critical mating distance S along the abscissa and the phenotypic critical mating distance G along the ordinate. Abundances are calculated by averaging the results of 90 snapshots of the population in a single run, starting at time 3000 and separated by 100 time steps (the total time elapsed in each simulation was 12,000 time steps). Results are shown in Fig. 5.

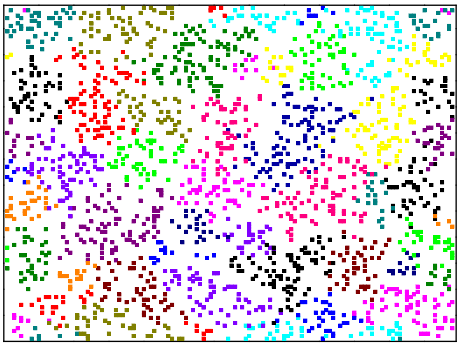

Fig. 4 Snapshot of a population after 2000 time steps for the haploid model with $G = 20$ and $S = 5$. *Colors* represent different species (some *colors* are repeated due to the large number of species formed)

4 Discussion

From the comparison of Fig. 5a and b, we observe that the contours corresponding to the number of species in the haploid model and in the diploid model with complete dominance behave in a similar way along the vertical direction (G axis). In the horizontal direction (S axis), the behavior changes significantly. For instance, in the range $6 \geq S \geq 7$, we observe the formation of several species in the haploid model, whereas in the diploid model with complete dominance, speciation does not occur. Speciation occurs only if individuals mate with others in closer proximity, namely, there is a need for a stronger inbreeding. A possible interpretation of these results is as follows: as the allele 0 dominates over the allele 1 and taking into account that at the beginning of the simulations all individuals have genomes composed by 0's only, for a phenotypic change to be expressed in the diploid model mutations need to affect both copies of the genome. The change therefore requires a higher mutation rate as compared to the haploid model. Since the mutation rate is the same in all simulations, the only way to increase the 'effective mutation rate' (the actual rate of allele change per locus) is by decreasing the size of the mating neighborhood [44]. Initial conditions in which one chromosome bears the recessive allele and the other the dominant one were also tested and show the same outcome, which can be explained by the need of the mutation to occur specifically in the chromosome with the recessive allele. However, if both chromosomes start with the recessive allele, the resulting contours are qualitatively similar to the case of haploid individuals. This happens because a single mutation in one of the chromosomes alters the phenotype, as if the genomes were haploid.

The comparison between Fig. 5a and c, on the other hand, shows the opposite behavior. The contours along the horizontal direction are similar to those of the haploid model. However, in the vertical direction of Fig. 5c there is a cutoff around G=27, above which species are not formed. This means that speciation needs stronger assortativity in comparison to the haploid model. As heterozygous loci display a different phenotype than any of the two homozygotes, mutations are now expected to affect phenotypes as if genomes were haploid. This explains why the number of species does not change, with respect to the haploid model, with the maximum search radius S. This time the changes are related to the critical mating genetic distance G and are due to the fact that in the diploid model with incomplete dominance there is a third phenotype with an intermediate value. The presence of this third phenotype makes the average genetic distance (per locus) between pairs of individuals become smaller in comparison to the corresponding average genetic distance of the haploid

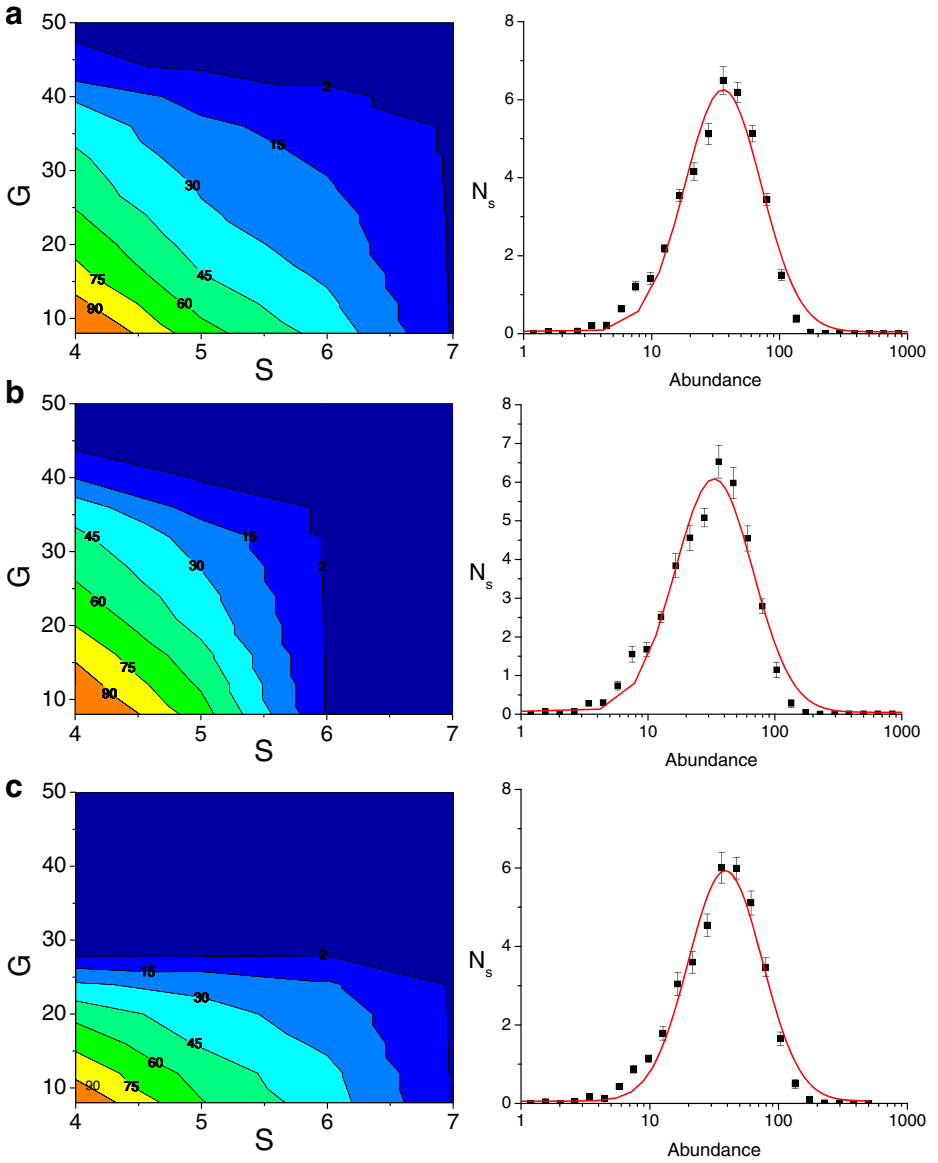

Fig. 5 Left: Average Number of Species (ANS) for different values of the critical mating distances. Right: Averaged Abundance Distribution (AAD) for S=5 and G=20 (*black squares* represent the averaged abundance data obtained from simulations, and solid curves correspond to log-normal fittings of the data; $y = A + \frac{y0}{w\sqrt{2\pi x}} \exp\left(-[\log(x/x_c)]^2/2w^2\right)$). **a** Haploid model ($y_0$=0.048 ± 0.096, A=490 ± 21, x_c= 58.0 ± 2.8, w = 0.69 ± 0.03, ANS = 42 ± 2). **b** Diploid model with complete dominance (y_0 = 0.049 ± 0.101, A = 455 ± 22, x_c = 55.0 ± 3.1, w = 0.71 ± 0.03, ANS = 42± 2). **c** Diploid model with incomplete dominance (y_0 = 0.058 ± 0.101, A = 470 ± 21, x_c = 60.0 ± 2.9, w = 0.66 ± 0.03, ANS = 39 ± 2)

model. Accordingly, with a given maximum genetic distance that, on average, would prevent haploid individuals to mate, for diploid individuals with incomplete dominance mating would still be allowed. Consequently, the presence of the third phenotype acts by increasing

the strength of the restriction that is necessary for the population to split into a given number of species (i.e., by decreasing the effective maximum genetic mating distance).

Interestingly, in the range where the three models predict speciation, biodiversity features are not sensitive to the genotype–phenotype map. The right panels of Fig. 5 display the abundance distributions obtained from the three models for S=20 and G=5. As can be seen from the graphs, there are no significant differences between the three distributions. This result is rather unexpected, since the implementation of assortative mating and the mechanism of reproduction in each case is quite different. Also, it shows that modeling the individuals with a single chromosome suffices to predict biodiversity patterns in more complex and general schemes of dominance.

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