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The Combined Effect of Haplodiplonty and Partial Clonality on Genotypic and Genetic Diversity in a Finite Mutating Population

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Abstract

Partial clonality is known to affect the genetic composition and evolutionary trajectory of diplontic (single, free-living diploid stage) populations. However, many partially clonal eukaryotes exhibit life cycles in which somatic development occurs in both haploid and diploid individuals (haplodiplontic life cycles). Here, we studied how haplodiplontic life cycles and partial clonality structurally constrain, as immutable parameters, the reshuffling of genetic diversity and its dynamics in populations over generations. We assessed the distribution of common population genetic indices at different proportions of haploids, rates of clonality, mutation rates, and sampling efforts. Our results showed that haplodiplontic life cycles alone in finite populations affect effective population sizes and the ranges of distributions of population genetic indices. With nonoverlapping generations, haplodiplonty allowed the evolution of 2 temporal genetic pools that may diverge in sympatry due to genetic drift under full sexuality and clonality. Partial clonality in these life cycles acted as a homogenizing force between those 2 pools. Moreover, the combined effects of proportion of haploids, rate of clonality, and the relative strength of mutation versus genetic drift impacts the distributions of population genetics indices, rendering it difficult to transpose and use knowledge accumulated from diplontic or haplontic species. Finally, we conclude by providing recommendations for sampling and analyzing the population genetics of partially clonal haplodiplontic taxa.

Subject area: Conservation genomics and biodiversity
Key words: mating system, asexuality, genetic and genotypic diversity, life cycle, linkage disequilibrium, F_{IS}

Introduction

Partial clonality, a reproductive system in which both asexual and sexual (including, selfing, inbreeding, and/or outcrossing) reproduction occur, is found across the majority of eukaryotes (Schön et al. 2009), including many taxa that are 1) integral to global processes (i.e., primary productivity), 2) foundation species in important ecosystems (e.g., corals or seagrasses: Gélin et al. 2017; Arnaud-Haond et al. 2019; algae: Krueger-Hadfield 2020; e.g., flowering plants: Stoeckel et al. 2006), and 3) human pathogens, agricultural pests, and invasive species (e.g., aphids, malaria, phytophthora: Lowe et al. 2004; De Meeûs et al. 2007; Barrett 2010, 2015; Song et al. 2013; Bonal et al. 2018; Gutekunst et al. 2018). In these taxa,

the balance between sexual and clonal events strongly influences the ecological success of a species (Halkett et al. 2005; Silvertown 2008) and the ability to track environmental change via phenotypic evolution (Orive et al. 2017). Thus, the evolutionary trajectories of partially clonal taxa may have major consequences for ecosystem functioning, and for human health and development. However, partial clonality has long been neglected in population genetic models (Avise 2015; Dia et al. 2014; Fehrer 2010; Yu et al. 2016), thereby restricting our understanding of the evolutionary consequences of a *very* common mixed reproductive mode across much of biodiversity.

All sexual eukaryotes, including partially clonal taxa, undergo a cyclic alternation between meiosis (diploid to haploid) and fertilization (haploid to diploid), but there is a tremendous variation in the amount of time spent in the haploid or diploid stage, and in the amount of somatic development that occurs in each ploidy stage. Although recent theoretical and empirical research since the pioneering work of Marshall and Weir (1979) has demonstrated a significant effect on the genetic composition and evolutionary trajectory of populations due to the simple alternation of clonal and sexual reproduction in diploid taxa (i.e., diplontic life cycles in which there is a single, free-living diploid stage; Reichel et al. 2016; Rouger et al. 2016; Stoeckel et al. 2021), many partially clonal eukaryotes exhibit life cycles in which somatic development occurs in both the haploid and diploid stages (Figure 1). Haplodiplonty in which there are *free-living, wholly independent* haploid *and* diploid stages is found across the major eukaryotic lineages (e.g., Stramenopiles: brown algae; Rhizaria: some foraminiferans; Opisthokonts: some fungi; Archaeplastida: green algae, red algae, ferns; Haptophytes: coccolithophores), suggesting this life cycle is remarkably stable on evolutionary time scales (e.g., Valero et al. 1992; Mable and Otto 1998). The effects of clonality in these life cycles remain largely understudied, restricting our understanding of the consequences of this reproductive mode across a large breadth of eukaryotic taxa. The lack of theoretical predictions of partial clonality on common population genetic summary metrics renders it challenging to explore in natural populations within an analogous framework that now exists for diplontic taxa (e.g., Arnaud-Haond et al. 2007; Stoeckel et al. 2021).

In many haplodiplontic life cycles, the independent haploid (often including separate sexes) and diploid individual stages can be

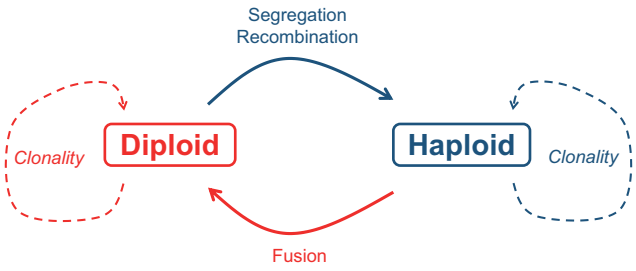

Figure 1. Simplified haplodiplontic life-cycle representative of those found across many eukaryotic lineages, including, as examples, algae (a polyphyletic group including multiple distinct eukaryotic groups of micro- and macroalgae), ferns, or fungi. The diploid (red) and haploid (blue) stages are spatially and temporally separated by the sexual processes of fusion (haploid to diploid; fertilization) and segregation and recombination (diploid to haploid; meiosis). Bell (1994) described these life cycles as an alternation of 2 vegetative cycles separated by 2 sexual processes (fusion and segregation/recombination). Thus, in addition to this sexual cycling (shown by solid lines), each ploidy stage may also be capable of asexual reproduction (clonality; shown by the dotted lines) through the fragmentation of thalli, the production of propagules, or mitotic divisions.

isomorphic. It is the case of *Gracilaria*, a partially clonal red algal genus, in which haploids and diploids are virtually indistinguishable (Kain and Destombe 1995). In other haplodiplontic life cycles, haploids and diploids can be strongly heteromorphic. This is the case of *Mastocarpus*, a partially clonal red macroalgal genus with an alternation crustose diploids and foliose haploids (Krueger-Hadfield 2020), and in kelps (Graham and Wilcox 2000) and ferns (Grusz 2016; Ebihara and Nitta 2019) where diploids are macroscopic and the haploids are microscopic. For many macroalgae, the haploids and diploids often live in the same environment, though intertidal zones are very dynamic environments on short spatial scales (Helmuth et al. 2011). In ferns, haploids and diploids can often occupy different and even separated habitats, posing questions as to which reproductive mode is found in each habitat and why such habitat disconnections exist (Nitta et al. 2017). Although different niches can more easily be explained for heteromorphic alternations of ploidy stages (e.g., bet-hedging, Lubchenco and Cubit 1980), it is not straightforward to explain how isomorphic alternations can be an evolutionary stable strategy when the life cycle stages are superficially similar (Valero et al. 1992). However, Hughes and Otto (1999) predicted the maintenance of haplodiplontic life cycles if there were even very subtle differences in the niche occupied by each of the stages. More studies investigating this hypothesis are warranted (Thornber 2006; Krueger-Hadfield 2020).

Empirical population genetic studies have been undertaken in remarkably few haplodiplontic taxa in which the life cycle has been thoroughly investigated by genotyping both haploid and diploid stages with codominant markers (reviewed in Valero et al. 2001; Krueger-Hadfield and Hoban 2016; Krueger-Hadfield 2020; Krueger-Hadfield et al. 2021). Yet, for partially clonal haplodiplontic taxa, there are unique consequences that stem from the spatiotemporal separation of meiosis and fertilization (e.g., Guillemin et al. 2008; Krueger-Hadfield et al. 2016, 2019). In those organisms, clonal reproduction can happen in any one of the free-living stages of the life cycle (in diploids or haploids, involving male, female, or hermaphrodite) with different consequences (see De Meeûs et al. 2007). And sometimes, one stage gets propagated and the other stages are potentially lost. For example, in the red macroalgal genus *Agarophyton* (formerly *Gracilaria*), a widespread invasion (Krueger-Hadfield et al. 2016, 2017) and farming practices (Guillemin et al. 2008) have led to the clonal propagation of diploids, where haploids have seemingly been lost. As diploids are the stage at which meiosis occurs, sexual reproduction can eventually be recovered. However, sex might be harder to recover in a haploid male or female only population (Guillemin et al. 2008; Krueger-Hadfield et al. 2016). We do not understand the longer-term consequences of clonality that occurs in one life cycle stage versus another despite the ubiquity of partially clonal, haplodiplontic taxa.

Over the past 2 decades, theoretical work has attempted to define the ecological and evolutionary conditions in which such biphasic life cycles and reproductive modes can emerge, be maintained, or disappear (Mable and Otto 1998; Hughes and Otto 1999; Vieira and Santos 2012; Scott and Rescan 2017). Life cycle evolution in the longer term depends on the dominance of mutations and the masking of deleterious and beneficial mutations (Perrot et al. 1991; Orr and Otto 1994; Otto 1994; Jenkins and Kirkpatrick 1995). Diploidy (i.e., diplontic life cycles) is expected to gain in fitness in the presence of partially recessive deleterious mutations and partially dominant beneficial mutations while haploidy (i.e., haplontic life cycles in which somatic development only occurs in the haploid stage) would be favored when partially

dominant deleterious mutations are common (reviewed in Valero et al. 1992). Diploidy is also expected to support higher mutation load protected by the masking of alleles that would otherwise be expressed in haploids and, therefore, would be favored at higher rates of recombination. Ultimately, for haplodiplontic life cycles in which both the haploid and diploid stages are long-lived, this results in variable mutation load and variable quantities of mutation across genetic backgrounds (Kondrashov and Crow 1991) with inequal effects on fitness depending on the stage considered (Scott and Rescan 2017), on the rate of recombination that exposes deleterious and beneficial mutations (Otto and Goldstein 1992), and on the efficiency with which they allow occupation of different ecological niches (Hughes and Otto 1999; Rescan et al. 2016). The evolution of different reproductive modes, such as exclusive sexuality versus partial clonality, follows the same logic.

If the evolution of life cycle and reproductive mode diversity is to be understood over evolutionary time scales, we must also have an understanding of the evolution of genetic diversity in populations over a few generations while considering the life cycle (i.e., haplodiplonty) and the reproductive mode (i.e., partial clonality) as immutable constraints (i.e., fixed quantitative parameters that cannot evolve at this time scale) to understand microevolution and population biology within species at contemporary time scales. When tackling questions in ecology, contemporary evolution, and the management of wild and cultivated populations (including epidemiology, conservation, invasion, global climate change), we have to deal with processes occurring at the scale of few tens to hundreds of generations within studied populations. Analyzing genetic diversity is a proven, efficient proxy to infer multiple biological and environmental features of the studied populations for less effort and with better reproducibility than direct observations (Wakeley 2005; Ellegren and Galtier 2016), especially considering life cycle and reproductive modes (Duminil et al. 2007). Thus, enabling the rational interpretation of values and ranges of values of genetic and genotypic indices that correspond to quantitative evolutionary forces (in our case, as the relative importance of haploid vs. diploid population sizes and the relative importance of clonal vs. sexual reproduction) over short time scales (i.e., a human generation) is essential for understanding ecology and microevolution of populations, and implications on their managements. Life cycles and reproductive modes structurally constrain genetic diversity and its dynamics in populations, which may result into fitness consequences under certain environmental conditions. This influence on fitness creates an opportunity for the establishment of a feedback loop extending the effect of life cycle at larger spatiotemporal scales, which may *in fact* influence the evolution of the life cycle and reproductive mode themselves. Thus, understanding the effects of life cycle and reproductive mode on genetic diversity in populations and its dynamics over a few generations is essential to understand larger evolutionary scales. Moreover, ecologists and population geneticists studying the demographic and genetics dynamics of populations and species on ecological time scales need to have clear expectations as to the combined effect of a particular life cycle and a given reproductive mode on the spatiotemporal distribution of genetic polymorphism to be able to recognize specific genetic signature of these biological processes.

Here, we studied how the respective fixed population sizes of haploid and diploid stages in a haplodiplontic life cycle influence the range of population genetic indices expected under various rates of clonality. We simulated evolving populations following a modified Wright–Fisher model tracking the evolution of genotype frequencies at multiple loci to include haploid and diploid life cycle stages and the possibility for genotypes to reproduce through clonality. Numerical results were analyzed to quantify the effects of the respective sizes of haploid and diploid stages at fixed total population size on the range of genetic and genotypic diversities we expect to observe in finite, mutating, partially clonal, haplodiplontic populations, such as those found among the algae, ferns, or fungi. We discuss those results and propose recommendations with which to interpret population genetics indices in such taxa.

Materials and methods

Approach

We tracked the evolution of genotype frequencies along generations using individual-based simulations to assess the range of values of population genetic indices as function of proportion of haploids and rates of clonality (Figure 2). Individual-based simulations followed a classical Wright–Fisher model extended to explicitly formalize panmictic-sexual and clonal reproductive modes in a life cycle in which individuals alternated through the sexual cycle between a long-lived haploid stage and a diploid stage with nonoverlapping generations. In other words, there were haploid or diploid stages that existed through time without an overlap between generations (i.e., t, $t + 1$, $t + 2$, etc.) of the 2 subpopulations. Each individual was composed of 100 loci segregating independently from diploid to haploid and fusing from haploid to diploid as a consequence of the spatiotemporally separated sexual cycle. Each locus possessed a maximum 10 different alleles that evolved by mutation following a stepwise K-allele model (KAM). In this mutation scheme, allele 1 mutates with the same probability either into allele 2 or allele 10, allele 2 mutates into allele 1 or 3, etc. The stepwise K-allele model has the advantage of simulating the behavior of both microsatellites and SNPs well (Weir and Cockerham 1984; Putman and Carbone 2014) and best approximates the "*disturbing factor of gene frequencies*" (sensu Wright 1931, including migration and mutation) in populations of finite size. Haploid or diploid subpopulations were able to reproduce through clonality at the same fixed rates of clonality, c, which determined the number of individuals that were randomly chosen with replacement to create descendants in the next generation with the same ploidy stage and genotype (i.e., genetically identical to their ancestor at all loci, except for mutations). Mutation occurred at the same rate, u, in both clonal and sexual haploid and diploid descendants just prior to reproduction in the next generation. Immediately after a mutation occurred and just prior to reproduction, we tracked and computed population genetic indices on adult genetic pools (Figure 2). By nature, this life cycle implies that haploids and diploids may evolve into 2 independent, genetically isolated pools either with an alternation of generations when strictly sexual or with a constant ploidy when strictly clonal.

We started all simulations with genotypes drawn at random with uniform allele frequencies to assess the steady-state distributions of population genetic indices expected at equilibrium between evolutionary forces. With no available prior on the number of generations needed to reach the steady-state distribution of genetic diversity, we computed population genetics on adult genetic pools after 10 000 burn-in generations. The distribution of population genetic indices were computed from 100 replicated simulations with parameter sets defined as quantitative values of 1) N, number of individuals per subpopulation (N = 10 000); 2) ph, the proportion of haploids (ph = 0.2, 0.4, 0.6, 0.8); 3) c, the rate of clonality (c = 0.0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 0.99, 1.0); and

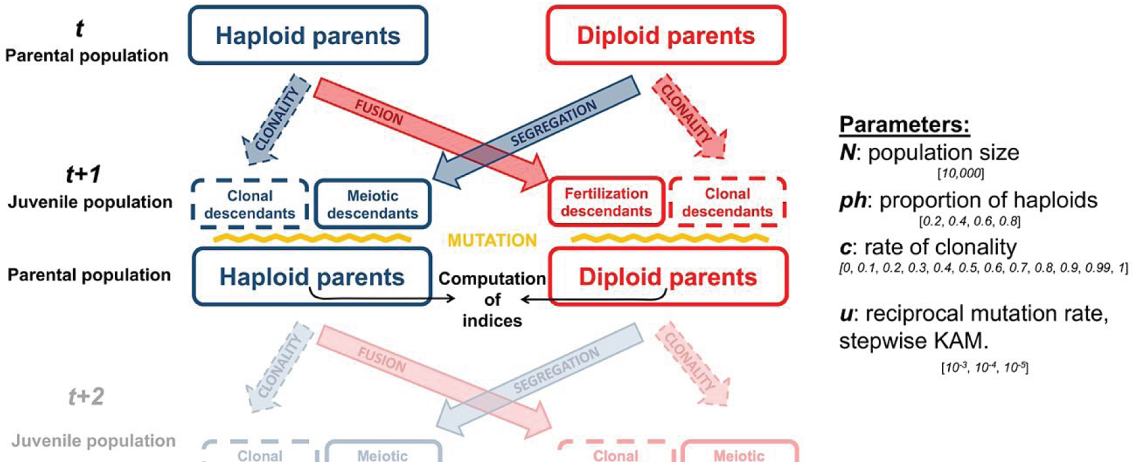

Figure 2. Algorithm diagram of the haplodiplontic simulator. Haploid (blue) and diploid (red) parents are shown at time *t*, followed by their descendants through fusion (for haploids: red solid line arrow; fertilization), segregation (for diploids: blue solid line arrow; meiosis), or clonality (blue or red dashed line arrow for haploids and diploids, respectively). We introduce mutation between the $t+1$ juvenile and parental populations. After the $t+1$ parental population is established, we compute the series of population genetic summary statistics commonly calculated for partially clonal taxa (Arnaud-Haond et al. 2007).

4) u, mutation rate ($u = 10^{-3}, 10^{-4}, 10^{-5}$). We, thus, assumed that population sizes of haploids and diploids, each remained constant over generations and that those sizes are controlled by external ecological constraints rather than resulting from evolutionary processes. Those assumptions make sense when interpreting reproductive mode and life cycle in contemporary populations in their ecological contexts rather than for understanding long-term evolution of reproductive mode and life cycle at the scale of species.

To disentangle the effects of the compartmentalization imposed by biphasic, haplodiplontic life cycles and the effects due to a specific ploidy stage, we also simulated diplontic (i.e., diploid-only life cycle; animal) and haplontic (i.e., haploid-only life cycle; charophyte) populations, noted as *D* and *H*, respectively, in which only one stage evolved (i.e., diplontic and haplontic life cycles, see Bell 1994 or Krueger-Hadfield 2020 for a review). In these single ploidy populations, sexual reproduction implies that segregation and fusion (diplontic) or fusion and segregation (haplontic) occurred with no spatiotemporal gap as in a haplodiplontic life cycle (Figure 1; see Bell 1994; Krueger-Hadfield 2020). That means, therefore, an infinite population size of the ephemeral haploid gametic or diploid zygotic stage between 2 successive generations.

To assess the combined effects of haplodiplontic life cycle and partial clonality on the distributions of genotypic and genetic diversity, we computed the estimation of effective population sizes measured from the Wright's standardized variance in allele frequency $Ne = 1/2F$ (Wright 1931; Waples 1989) using the bounded Nei and Tajima (1981) estimator of F, i.e., F_C:

$$F_C = \frac{\sum_{j=1}^{n}\sum_{i=1}^{K_j}\frac{(x_i-y_i)2}{\frac{(x_i+y_i)}{2}-x_iy_i}}{\sum_{j=1}^{n}K_j}$$

where n is the total number of loci, K_j is the number of alleles at locus j, x_i the allele frequency of allele i at locus j in the previous generation, and y_i the allele frequency of the same allele i at locus j in the current generation.

In diplontic populations, partial clonality is known to affect genotypic and genetic diversity (Halkett et al. 2005; Arnaud-Haond et al. 2007). Typical signature of partial clonality in diplontic populations is repeated genotypes leading to reduced genotypic diversity, linkage disequilibrium between loci and high variance of F_{IS} with a global excess of heterozygotes (Halkett et al. 2005; Arnaud-Haond et al. 2007, 2020; Stoeckel and Masson 2014; Stoeckel et al. 2021). We thus computed genotypic richness (R) and the size distribution of multilocus repeated genotypes (Pareto β) and linkage disequilibrium between loci ($\bar{r}_d$) to characterize the distributions of genotypic diversity 1) in haploid genetic pool (HGP) and 2) in diploid genetic pool (DGP), and 3) over the entire population by grouping HGP and DGP. For computing population genetic indices in the entire population, we calculated allele frequencies using their true counts over a total of $(2 - \mathrm{ph}) \times N$ alleles in the entire population. Therefore, we counted 1 allele for a haploid individual, 1 allele for a diploid heterozygote, and 2 alleles for a diploid homozygote, then divided by the total number of count of alleles: $f(a)_{\mathrm{entire}} = \frac{n_a^{\mathrm{haploids}} + n_a^{\mathrm{diploids}}}{(2-\mathrm{ph})\times N}$ with $f(a)_{\mathrm{entire}}$, $f(a)_{\mathrm{diploids}}$, and $f(a)_{\mathrm{haploids}}$ being the frequency of allele a in the entire population and in the haploid and the diploid parts of the population, respectively; where n_a^{haploids} and n_a^{diploids} are the respective number of allele a counted in the haploid and diploid parts of the population. We did not apply any correction as the one proposed by Bessho and Otto (2017, 2020) that aims at computing "evolutionary relevant average allele frequencies" but rather analyzed our simulation output as one would do with a dataset from the field with the aim to bridge theoretical predictions with future observations. Allele frequencies in our work correspond to the "haploid-diploid Wright–Fisher model with local regulation and asexual looping" case in Bessho and Otto (2020).

In the DGP, we explored the combined effects of haplodiplontic life cycle and partial clonality on heterozygosity by computing the observed mean and variance of the inbreeding coefficient, mean(F_{IS}) and var(F_{IS}), respectively.

Genotypic Richness (*R*)

The R index of clonal diversity (Dorken and Eckert 2001) was defined as follows:

$$R = \frac{(G-1)}{(N-1)}$$

where G is the number of distinct genotypes (i.e., genets) and N is the number of genotyped samples.

Size Distribution of Multilocus Repeated Genotypes (Pareto β)

The parameter Pareto β describes the slope of the power-law inverse cumulative distribution of the size of lineages (Arnaud-Haond et al. 2007):

$$N \geq X = \alpha X - \beta$$

where $N \geq X$ is the number of sampled ramets belonging to genets containing X or more ramets in the sample of the population studied, and the parameters α and β are fitted by regression analysis.

Linkage Disequilibrium Between Loci ($\bar{r}_d$)

We computed linkage disequilibrium using $\bar{r}_d$ (Agapow and Burt 2001). The mean correlation coefficient ($\bar{r}$) of genetic distance (d) between unordered alleles at n loci ranged from 0 to 1. This metric has the advantage of limiting the dependency of the correlation coefficient on the number of alleles and loci and is well suited to studies of partially clonal populations.

$$\bar{r}_d = \frac{V_D - \sum_{j=1}^{j=n} \mathrm{var}_j}{2\sum_{j=1}^{j=n}\sum_{k>j}^{k=n}\sqrt{\mathrm{var}_j.\mathrm{var}_k}}$$

with $V_D = \frac{\sum_{a,b\neq a}^{v} D_{a,b}^2 - \frac{\left(\sum_{v} D_{a,b}\right)^2}{v}}{v}$ and $\mathrm{var}_j = \frac{\sum d^2 - \frac{\left(\sum_{v} d\right)^2}{v}}{v}$

where D is the number of loci at which 2 individuals, a and b, differ (i.e., genetic distance between 2 individuals over all their loci), d is the number of different alleles between 2 individuals at locus j (for diploids, d can be 0, 1, or 2; for haploids, d can be 0 or 1), and v is the number of unique possible pairs of individuals a and b where $b \neq a$ within a population.

The Inbreeding Coefficient (F_{IS})

The Wright's (1921, 1969) inbreeding coefficient F_{IS} accounts for intraindividual genetic variation as a departure from Hardy–Weinberg assumptions of the genotyped populations. We computed one F_{IS} value per diploid population and per locus l as $F_{IS_l} = \frac{Q_{w,l} - Q_{b,l}}{1 - Q_{b,l}}$, where $Q_{w,l}$ is the population probability that 2 homologous alleles within an diploid individual are identical and $Q_{b,l}$ is the population probability that 2 homologous alleles between different diploid individuals are identical. We, then, computed mean and variance of the empirical F_{IS} distribution obtained from the 10 000 independent F_{IS} values per scenario (100 independent loci × 100 replicated simulations).

Simulations, analyses, and plots were performed using Python3.8 with Numpy1.17.3, Scipy1.3.0, Sympy1.4, Matplotlib3.1.0, and Seaborn0.10.1 packages.

Results

Both alone and combined, haplodiplontic life cycles and clonality changed the range of population genetic indices. Ten thousand generations seemed to be enough to reach equilibrium for all indices at all explored proportions of haploids (ph) and in diplontic (D) and haplontic (H) populations, at all rates of clonality (Supplementary Figures S1–S4), except for fully clonal populations and fully sexual populations considering linkage disequilibrium. Fully clonal diplontic populations are known to require infinite number of generations to reach steady-state distributions of population genetic indices in finite mutating populations (Reichel et al. 2016).

Genetic Differentiation Between Temporal Genetic Pools

After 10 000 generations from a homogeneous ancestral population, fully sexual populations showed F_{st} between 2 successive generations in each ploidy pool ranging from 0.23 to 0.64 (Supplementary Figure S5). As a function of the proportion of haploids, the pattern followed a concave-upward parabola curve that reached its minimum value for proportion of haploids around 0.6. In fully sexual populations, F_{st} between haploids and diploids at one generation ranged between 0.17 and 0.38 slightly increasing with increasing proportion of haploids.

In fully clonal populations, F_{st} between 2 successive generations in each ploidy pool ranged from 2×10^{-6} to 2×10^{-3}. F_{st} between haploids and diploids at one generation in fully clonal populations varied over a larger range, between 0.07 and 0.4, depending on the proportion of haploids. As function of the proportion of haploids, the pattern followed a concave-downward parabola curve that reached its maximal value for proportion of haploids around 0.6. The variance of these distributions increased with increasing proportions of haploids.

In partially clonal populations, F_{st} between 2 successive generations in each ploidy pool and F_{st} between haploids and diploids at one generation were all under 6.5×10^{-4} and 9×10^{-3}, respectively.

Effects of Haploid–Diploid Partially Clonal Life Cycle on Genetic Drift and Effective Population Sizes

Effective population sizes measured from the standardized variance in allele frequency including both the haploid and diploid genetic pools varied under the joint action of the proportion of haploids in populations and their rate of clonal reproduction (Figure 3 and Supplementary Figures S6 and S7). Increasing the rate of clonality increased effective population sizes and its variance, regardless of proportion of haploids. The effective population sizes followed a concave-downward shape with increasing proportions of haploids for rates of clonality less than $c <\sim 1 - \frac{\alpha}{N}$ with α a positive constant. The maximum of this concave-downward relationship was reached at lower proportion of haploids and resulted in higher effective population sizes as rates of clonality increased. Effective population sizes nearly linearly decreased with increasing proportion of haploids for rates of clonality higher than $c >\sim 1 - \frac{\alpha}{N}$.

Influence of the separate and combined effect of partial clonality and haplodiplonty on genotypic and genetic diversities

Repeated Genotypes and Genotypic Diversity

When genotyping the entire population, genotypic richness (R) in both the haploid and diploid genetic pools presented the same global patterns as obtained in diplontic and haplontic populations (Supplementary Figure S8). It followed a nonlinear trend as a function of rate of clonality, previously well approximated by the relationship $R = \sqrt{1 - c2}$ in diplontic taxa (Stoeckel et al. 2021). In haplodiplontic populations with skewed proportions of haploids versus diploids, fully clonal (c = 1) populations showed higher genotypic richness than observed in populations with very rare events of sex (c = 0.999), as expected in very clonal, diplontic populations with dominant genetic drift (Balloux et al. 2003; Reichel et al. 2016). The distribution of genotypic richness varied widely with the

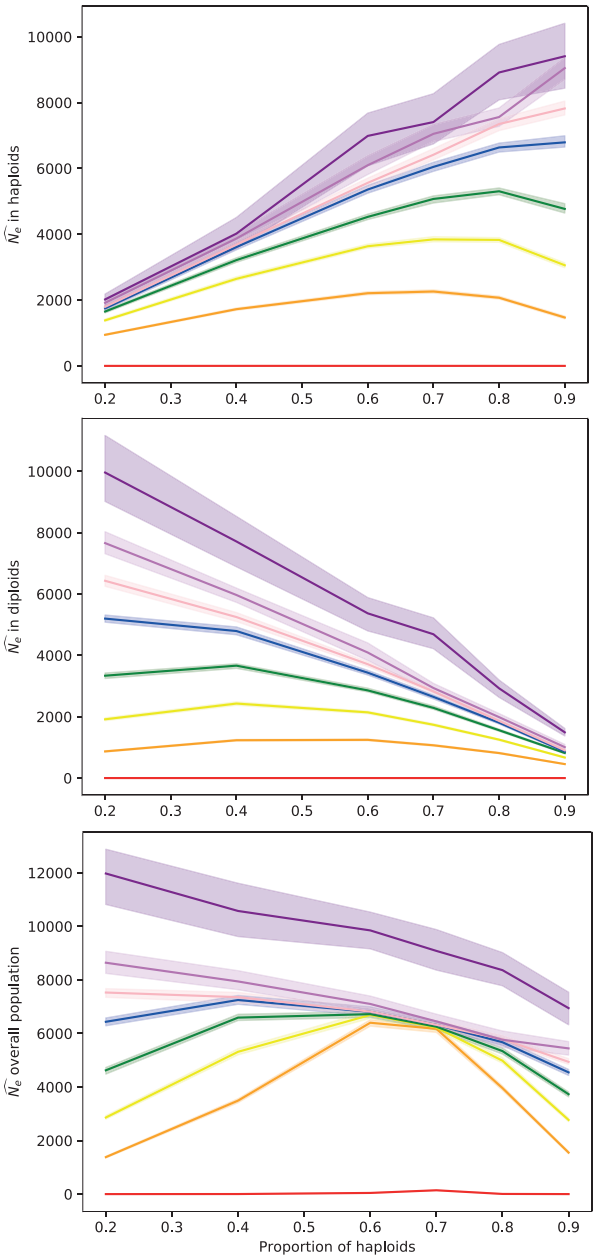

Figure 3. Effective population sizes measured from the standardized variance in allele frequency over 2 successive generations in haploids (top plot), in diploids (second plot), and in the entire population (including both haploid and diploid genetic pools, bottom plot) as function of proportion of haploids in populations and rates of clonality. Population size of 10 000 individuals and mutation rate of 10^{-4}. Solid lines plot the average values, and semitransparent envelops the confidence interval obtained by bootstrapping on the 100 measures. Colors account for rates of clonality: red for $c = 0$ (only sexual), orange $c = 0.2$, yellow $c = 0.4$, green $c = 0.6$, blue $c = 0.8$, pink $c = 0.9$, orchid $c = 0.99$, and purple $c = 1$ (only clonal).

proportion of haploids and the relative strength of genetic drift and mutation force (Supplementary Figures S9 and S10).

Pareto β also followed a nonlinear trend as a function of the rate of clonality, similar to the pattern previously observed in diplontic taxa (Stoeckel et al. 2021), in both the haploid and diploid genetic pools. Regardless of the proportion of haploids, the maximum Pareto β always occurred in fully sexual populations and its value depended on the population size while its lower bound is fixed at 0, corresponding to populations with one large, very dominant even fixed genet (Figure 4). Estimates of Pareto β showed a higher range of distributions in more sexual populations, which increased more at skewed proportion of haploids versus diploids. Interestingly, Pareto β nicely identified artefactual coupling of identical genotypes that occurred in fully sexual populations, despite the 100 loci simulated, resulting in some lower Pareto β values than observed in partially clonal populations. The few identical genotypes found in exclusively sexual populations were caused by genetic drift that skewed allelic frequencies at most loci and likely resulted in the repeated occurrence of few haplotypes by random association. Pareto β over the entire haplodiplontic populations increased as the proportion of haploids increased, but always remained below values observed in haplontic and diplontic populations. During the sexual cycle, new genotypes were created during the segregation phase (i.e., when diploids produced haploids via meiosis). Fusion (haploid to diploid) did not change the haplotypic diversity because it only consisted of a combination of already existing haplotypes present in the haploid pool. Genetic drift in mostly sexual populations acted to fix haplotypes, whereas clonality limited the effect of genetic drift. Under limiting conditions (i.e., low proportion of haploids), more haplotypes were fixed in sexual populations than in partially clonal populations, resulting in higher proportions of repeated multilocus genotypes in haplodiplontic sexual populations with low proportion of haploids than in more clonal populations with the same proportion of haploids.

The distributions of Pareto β in each ploidy pool remained stable in values and ranges with rates of clonality regardless of the relative strength of genetic drift versus mutation force. However, the distributions were still sensitive to the proportion of haploids when computed over the entire population, whatever the relative strength of genetic drift and mutation force (Supplementary Figures S11 and S12).

Linkage Disequilibrium

In haplontic and diplontic populations (H and D), any level of sexual reproduction wiped out significant linkage disequilibrium in the haploid pool, the diploid pool, and altogether (Figure 6) excepted when genetic drift dominated mutation (i.e., small populations; Supplementary Figures S13 and S14). Only fully clonal populations presented significant linkage disequilibrium in HGP and DGP. Those results are in accordance with results obtained by Navascués et al. (2010) for diplontic taxa: linkage disequilibrium is only expected in partially clonal populations when genetic drift dominates the dynamics of genetic diversity. In haplodiplontic populations, linkage disequilibrium over the entire population (HGP and DGP grouped together) occurred either at very high clonal rates (whatever the proportion of haploids) or in completely sexual populations (Figure 5). Linkage disequilibrium in this last case, obtained by grouping HGP and DGP in fully sexual populations, resulted from the fact that we are there grouping together 2 divergent genetic pools (i.e., a Wahlund effect). Interestingly, values of linkage disequilibrium remained very stable and similar to the diplontic range of values across the proportion of haploids. In other words, the haplodiplontic life cycles would need very skewed proportion of haploids to impact linkage disequilibrium. Those trends were maintained whatever the relative strength of genetic drift versus mutation force (Supplementary Figures S13 and S14). Increasing mutation force decreased linkage disequilibrium. Notice that when genetic drift dominates mutation forces ($\mu < 1/N$), haplodiplontic fully sexual populations showed

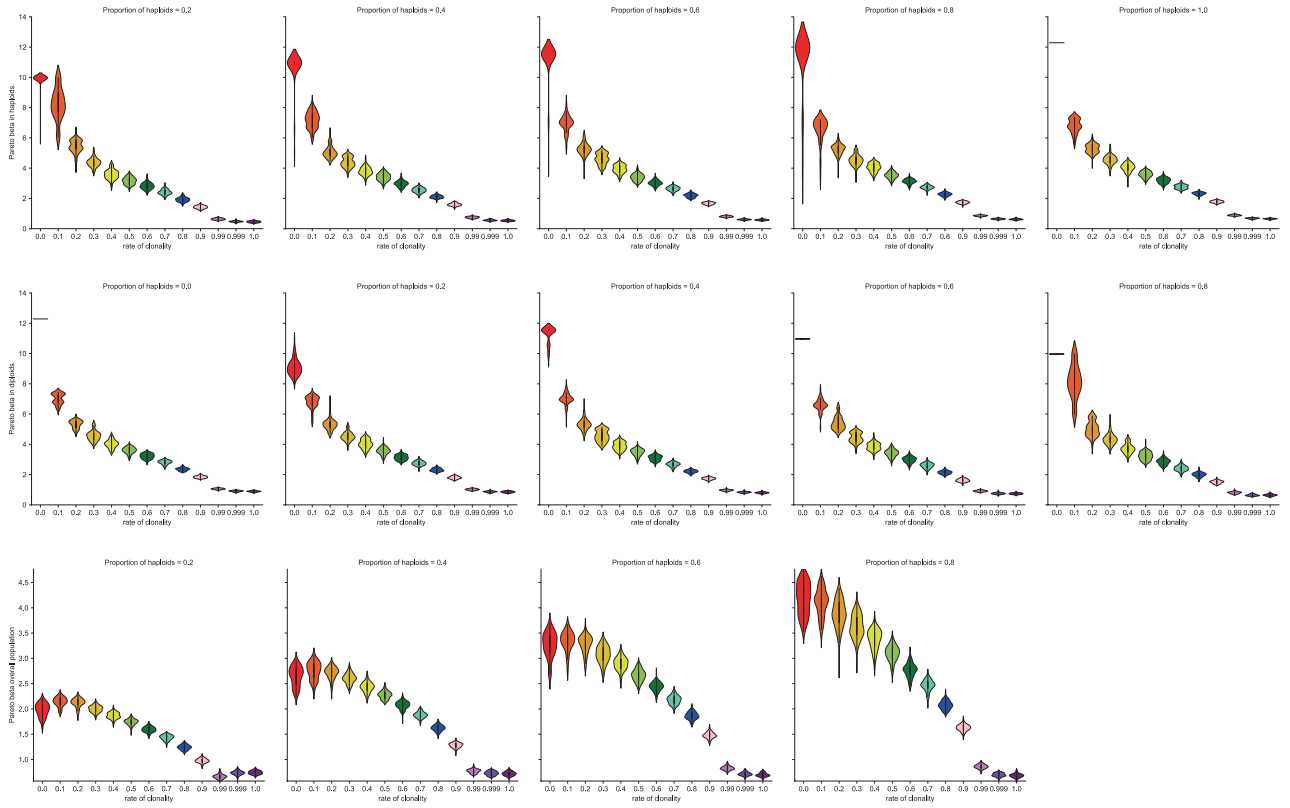

Figure 4. Violin plot of the ranges of genotypic diversity computed as Pareto beta (β) as function of proportion of haploids and of rates of clonality. Distributions of Pareto beta in haploids (first row of plots), in diploids (second row), and over the entire population (third row). Colors account for rates of clonality: red for $c = 0$ (only sexual), orange-red $c = 0.1$, orange $c = 0.2$, gold $c = 0.3$, yellow $c = 0.4$, yellow-green $c = 0.5$, green $c = 0.6$, aquamarine $c = 0.7$, blue $c = 0.8$, pink $c = 0.9$, orchid $c = 0.99$, violet $c = 0.999$, and purple $c = 1$ (only clonal). Population size of 10 000 individuals and mutation rate of 10^{-4}, violin plots are computed on 100 replicates.

higher linkage disequilibrium at low proportion of haploids than in only clonal populations.

F_{IS} Distributions in the Diploid Compartment

Increasing the proportions of haploids tended to decrease mean F_{IS} values toward more negative values and increase the proportion of negative F_{IS} (Figure 6). It also increased the variances among loci, slightly in partially clonals and more strongly in fully clonal populations. Small population sizes of haploids increased genetic drift, which decreased the number of possible haplotypes in the whole population and increased the probability of homozygotes in diploid stage by fusion while small population sizes of diploids increased the probability of heterozygotes.

In haplodiplontic populations, the variance of F_{IS} among loci as function of rates of clonality followed a u-shaped curve, with lower variance of F_{IS} among loci obtained for low rates of clonality (c around 0.1–0.3) rather than for fully sexual populations in diplontics. Increasing the proportion of haploids in population tended to accentuate this u-shaped relationship and to increase the variance of F_{IS} at all rates of clonality. Interestingly, these trends were maintained whatever the relative strength of genetic drift versus mutation force, except for fully clonal populations (Supplementary Figures S15–S18).

Sampling Populations

If genotypic and genetic indices showed identifiable variations with rates of clonality and proportion of haploids when knowing the genotypes of all individuals in population, estimating their true values from samples is much more problematic. As demonstrated in diplontic populations (Stoeckel et al. 2021; Arnaud-Haond et al. 2020), most indices based on genotypic diversity, and thus the occurrence of repeated genotypes in population, were sensitive to the sample density (i.e., the sample size relative to the population size). Sampling with realistic sample sizes (several tens to hundreds samples) consistently failed to provide a picture of true genotypic diversity in the whole population. Subsampling also weakened identifiable signals linking rates of clonality and the proportion of haploids to estimated genotypic diversity.

R estimates from samples in partially clonal populations were strongly biased toward 1, leading to overestimation of the importance of sexuality in populations (Supplementary Figure S19). A correct and unbiased estimate of R could only be achieved by genotyping the entire population, which makes this parameter useless to interpret in common molecular ecology studies and to estimate rates of clonality.

Interestingly, however, estimations of Pareto β with sample sizes of 150 individuals in HGP and DGP and over the entire population were similar to sampling the entire population (Supplementary Figures S20). For rates of clonality higher than $c = 0.4$, sampling only 30 individuals was sufficient to obtain unbiased measures of Pareto β whatever the proportion of haploids.

As similarly demonstrated in diplontic populations (Stoeckel et al. 2021; Arnaud-Haond et al. 2020), indices based on genetic diversity were robustly estimated from low sample sizes. Random

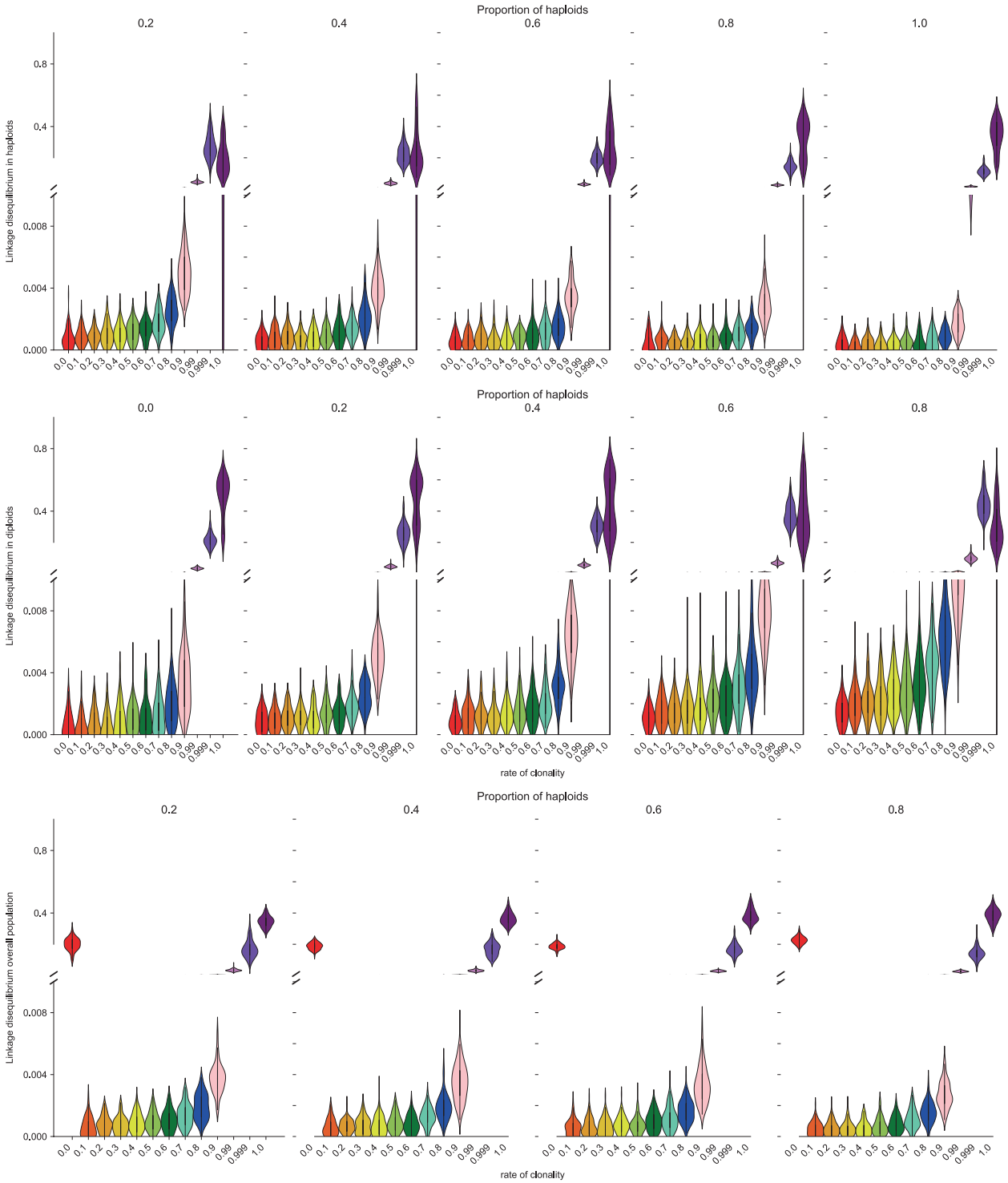

Figure 5. Violin plot of the ranges of linkage disequilibrium computed as $\bar{r}_d$ as function of proportion of haploids and of rates of clonality. Distributions of linkage disequilibrium in haploids (first row of plots), in diploids (second row) and over the entire population (third row). Colors account for rates of clonality: red for $c = 0$ (only sexual), orange-red $c = 0.1$, orange $c = 0.2$, gold $c = 0.3$, yellow $c = 0.4$, yellow-green $c = 0.5$, green $c = 0.6$, aquamarine $c = 0.7$, blue $c = 0.8$, pink $c = 0.9$, orchid $c = 0.99$, violet $c = 0.999$, and purple $c = 1$ (only clonal). Population size of 10 000 individuals and mutation rate of 10^{-4}; violin plots are computed on 100 replicates.

sampling of 30–50 individuals per ploidy pool was enough to nicely estimate average values of genetic indices (F_{IS} and linkage disequilibrium as $\bar{r}_d$) in populations whatever the proportion of haploids and rates of clonality (Supplementary Figures S21 and S22). Reliably estimating their variances among loci, however, required sampling a minimum of 150 individuals. Interestingly, as found in diplontic populations (Stoeckel et al. 2021), the variance of F_{IS} among loci was more discriminant to assess the extent of clonality when using low

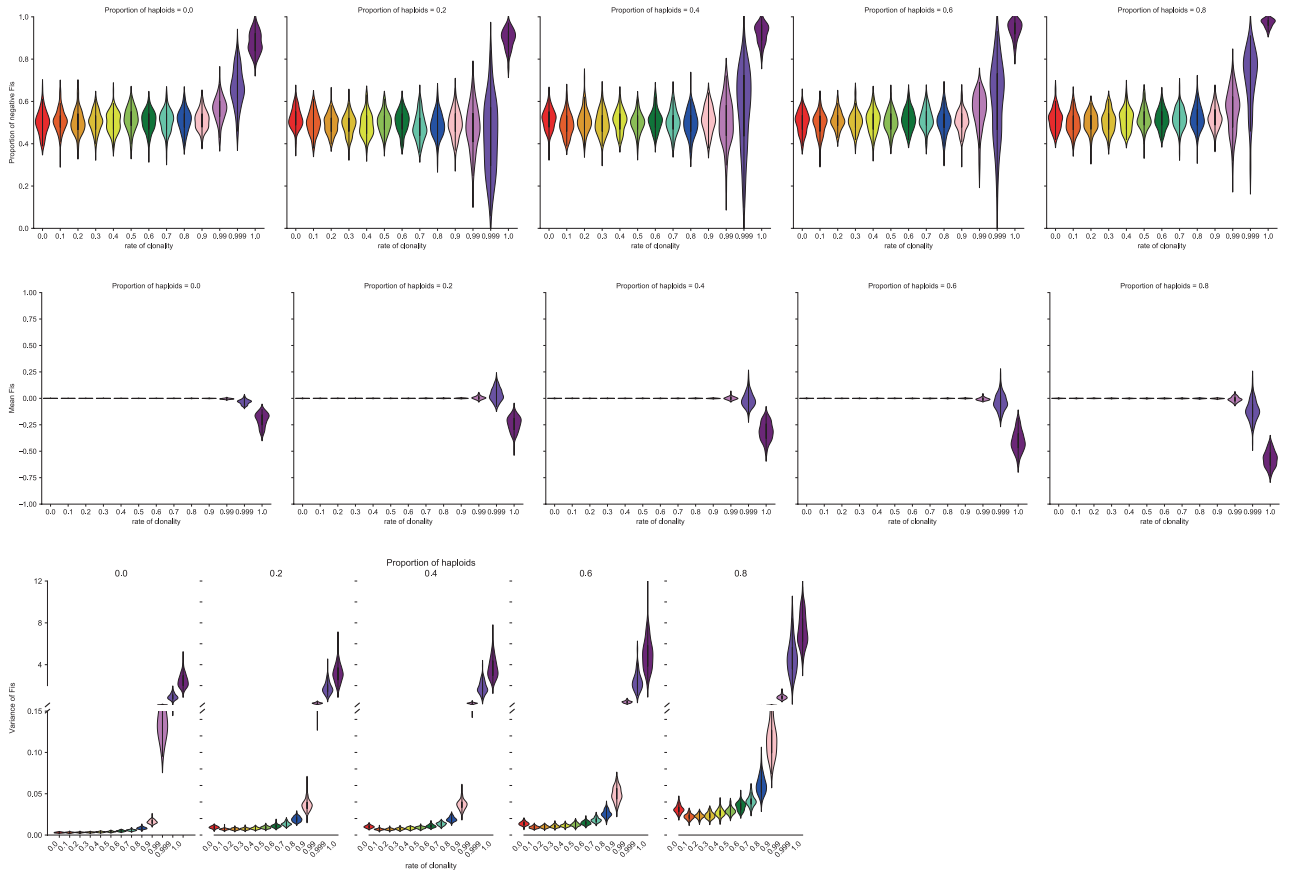

Figure 6. Violin plot of the proportion of loci with negative F_{IS} (first row of plots), proportion of average F_{IS} (second row of plots), and variance of F_{IS} across loci (third row of plots) as function of proportion of haploids and of rates of clonality. Colors account for rates of clonality: red for $c = 0$ (only sexual), orange-red $c = 0.1$, orange $c = 0.2$, gold $c = 0.3$, yellow $c = 0.4$, yellow-green $c = 0.5$, green $c = 0.6$, aquamarine $c = 0.7$, blue $c = 0.8$, pink $c = 0.9$, orchid $c = 0.99$, violet $c = 0.999$, and purple $c = 1$ (only clonal). Population size of 10 000 individuals and mutation rate of 10^{-4}, violin plots are computed on 100 replicates.

sample size than the entire population. Contrary to diplontic populations, at low samples sizes, variance of F_{IS} among loci followed a concave-upward parabolic curve with increasing rates of clonality. Maximum variance of F_{IS} among loci was even higher in fully sexual populations than in fully clonals for unbalanced proportions of haploids (ph ≤ 0.4 and ph ≥ 0.8). Finally, despite the robustness of their estimates to sampling, indices based on genetic diversity poorly segregated with rates of clonality and proportion of haploids.

Discussion

Few studies have explored the population genetics of experimental and field haplodiplontic species despite the common occurrence of haplodiplonty across eukaryotes (Valero et al. 1992; Mable and Otto 1998). This is likely due to the lack of formal methodology and recommendations that would enable linking ranges of population genetic indices with ecological and evolutionary features, like reproductive modes and characteristics of haplodiplontic life cycle (but see, Krueger-Hadfield and Hoban 2016). Our results showed that haplodiplontic life cycles interact with partial clonality to jointly affect the influence of genetic drift and the resulting range of genetic and genotypic diversity, with potential consequences for the ecology and evolution of such organisms. We demonstrate that we cannot directly use knowledge developed for diplontic species to transpose it to haplodiplontic species when implementing sampling strategies and interpreting population genetic indices. Future empirical studies should thus follow specific recommendations developed here below for haplodiplontic species. Hereafter, we discuss the implications of our results for understanding genotypic and genetic diversity in partially clonal, haplodiplontic populations and provide some recommendations for sampling and genotyping in these taxa.

The specificities of haplodiplontic life cycle on genetic diversity and the importance of hysteresis in maintaining cohesion between haplontic and diplontic compartments.

Across generations, the haplodiplontic life cycle allows for the temporal evolution of 2 genetic pools with different ploidies depending on the reproductive mode (Supplementary Figure S23). The existence at one time of 2 genetic pools hierarchically structures the coalescence between individuals over generations when considering the entire population.

In the haplodiplontic life cycle, clonality allows lineages to remain in the same ploidy compartment over generations, limiting the influence of compartments on each other's genetic evolution. This slows down the mixture and thus the homogenization between the 2 genetic pools with different ploidies at one generation. In fully clonal populations, we even expect that the population diverges into 2 genetic compartments with a stable ploidy over time, evolving independently, until the accumulation of genetic divergence that would result into the formation of 2 species, 1 haploid and 1 diploid.

Sexuality, however, results in the reversal of genetic ancestries between ploidy compartments over generations. Exclusive sexuality in the haplodiplontic life cycle without mechanisms favoring hysteresis (i.e., dependence of the states of a genetic pool on its historical, previous states), such as overlapping generations or dormancy, also allows for the independent evolution of 2 genetic pools that cyclically interchange their ploidy along generations without mixing their ancestries until they accumulate enough genetic divergence to form 2 new haplodiplontic, temporally unphased, species. The recent advances in DNA sequence-based species identification techniques have led to the increasing recognition of this kind of temporal divergences of closely related fungi species that occupy the same niche (e.g., complexes of sibling parasite species infecting the same host plant, and even the same organ), challenging the competitive exclusion principle (Fitt et al. 2006; Hamelin et al. 2016). In natural populations of sexual, haplodiplontic macroalgae, we see no evidence of genetic differentiation among HGP and DGP probably due to overlapping generations as the species studied thus far are perennial (Engel et al. 2004; Krueger-Hadfield et al. 2013). But multiple haplodiplontic populations of microorganisms, such as unicellular algae, rhizaria, and opisthokonts, may also mainly reproduce by sexuality without such hysteresis mechanisms. We lack knowledge on reproductive modes and life cycle of such microorganisms in nature. Direct observations are either nearly impossible or so costly that the only perspective to understand their biology would be to rely on indirect inferences based on genotyping. Here, we showed that this type of life cycle with fully sexual reproduction explicitly favors the sympatric evolution of temporal, diverging genetic pools from a single finite population.

Interestingly, partial clonality introduces a temporal continuity between those potentially diverging genetic pools by keeping ancestral diversity in the same temporal ploidy pool over few generations and by allowing mixing between genetic pools with different ploidies along generations. Partial clonality renders genetic evolution of haplodiplontic taxa more similar to simpler and better studied diplontic or haplontic life cycles in terms of the distributions of population genetic indices. This homogenizing effect is even more visible with skewed proportion of haploids. It is still currently unclear what the role of partial clonality is in natural populations, though many macroalgae with haploid-biased populations (e.g., *Chondrus crispus* Krueger-Hadfield et al. 2013) or mosses with a dominant haploid stage (e.g., Patiño et al. 2013) could serve as excellent empirical models to test the predictions we have uncovered here.

More than ~1/*N* clonal or sexual events in strictly sexual and clonal populations, respectively, were sufficient to remove any genetic differentiation between their temporal genetic pools. This effect may explain why most, if not all, haplodiplontic species are partially clonal. It is, however, not intuitive that very few clonal reproductive events gave the same genetic diversity results as many more clonal events. Partial clonality affects the evolutionary trajectories of genotype frequencies over a few generations, but as soon as generations accumulate, the probability that all lineages of clonal genotypes have segregated and recombined increases to reach 1 asymptotically (Reichel et al. 2016). Such dynamics imply that for understanding the evolution of partial clonality versus full sexuality or full clonality, we should consider events occurring at short evolutionary time scales, such as demographic events such as bottlenecks, invasions of new regions, and para- and peripatric evolutionary events. Empirical studies of the partially clonal macroalgal genus *Agarophyton* may shed light on some of these processes as species in this genus have repeatedly invaded soft sediment habitats through various anthropogenic means, leading to large variations of their reproductive system in different populations (Guillemin et al. 2008; Krueger-Hadfield et al. 2016; Becheler et al. 2020). Comparing sexual populations to the clonal ones will elucidate the effects of bottlenecks as well as evolutionary events that occur in sympatry or parapatry.

Our results also promote the interest for future researches on fully clonal and fully sexual haplodiplontic populations to search, identify, and study the ecological prevalence and evolutionary dynamics of biological and environmental features that would introduce hysteresis in their genetic pools, preventing the divergence between their genetic pools. They may also complete the view of partial clonality as a stabilizing reproductive mode, homogenizing genetic diversity over generations (Reichel et al. 2016; Rouger et al. 2016) and between different temporal genetic pools that may evolve from the life cycle, as demonstrated here, or from other environmental and biological features. Finally, future genetic studies interested in the ecology and taxonomy of fully sexual haplodiplontic species without hysteresis mechanisms, such as unicellular taxa and microorganisms, should explore the possible existence of complexes of sibling species occupying the same niches over time.

The Synergistic Impacts of Haplodiplontic Life Cycle and Partial Clonality on Genotypic and Genetic Diversity

When all individuals in a population were genotyped, the distributions of genotypic indices in separated HGP and DGP were nicely segregated as function of rates of clonality if higher than $c \geq 0.2$, with a limited influence of proportion of haploids, as long as not extremely skewed (i.e., for $0.1 < ph < 0.9$). When computed on the entire population by grouping HGP and DGP together, distributions of genotypic indices were synergistically influenced by both rates of clonality and proportion of haploids. However, at all levels of the population, genotypic indices proved strongly sensitive to sampling. Pareto β appeared to be the more robust to sampling while genotypic richness (*R*) was more sensitive.

In contrast and as observed in diplontic species (Stoeckel et al. 2021), distributions of genetic indices in separated HGP and DGP, and on the entire population by grouping HGP and DGP together, poorly segregated as function of rates of clonality. Distributions began to be identifiable only with high rates of clonality ($c \geq 0.6$). They were also influenced by the proportion of haploids, especially when the ratio haploid versus diploid was extremely skewed. Such influence came from the genetic drift imposed by the genetic pool with the lower size. The population size of the HGP had more influence on distributions of genotypic and genetic indices. Indeed, it determined the number of possible haplotypes over the entire population, determining linkage disequilibrium between genes and limiting genotypic diversity even for the larger population size of diploids.

Recommendations to Infer Proportion of Haploids and Rates of Clonality in Population Using Genotyping

Population genetics provides robust methods and cheap ways to quantitatively infer biological traits and features linked to evolutionary forces, especially for species for which direct observations and measures cannot be performed (Ellegren and Galtier 2016; see the pioneering work of Marshall and Brown 1974 on rates of clonality). Previous work on diplontic species have demonstrated

that roughly inferring rates of clonality within field and experimental populations can be fulfilled using interpretations of affordable, one-shot genotyping (De Meeûs et al. 2006; Stoeckel et al. 2021). Here, we showed that reproductive mode and the haplodiplontic life cycle interacted to shape distributions of genotypic and genetic indices that differ from distributions previously known and commonly interpreted in diplontic species. Therefore, studying genetic diversity in haplodiplontic populations needs specific recommendations.

Our results argued that genotypes in haplodiplontic populations should be processed separately at HGP and DGP levels, as well as at the entire population level by grouping HGP and DGP together, to finally obtain 3 values per population per index. To date, haplodiplontic data sets have been typically split into HGP (grouping individuals with 1 allele at all loci) and DGP (grouping individuals with 2 alleles at least one of their loci), and then processed in parallel to obtain 2 values of each index for each population (e.g., Engel et al. 2004; Krueger-Hadfield et al. 2013). Downstream processing of population genetic data in programs, such as GenAlEx (Peakall and Smouse 2006, 2012), requires separate diploid and haploid input files. Programs dedicated to the analyses of partially clonal populations, such as Rclone (Bailleul et al. 2016), to date, only allow for diploid populations even if some of those methods and software can be used with some caution in haplodiplontic taxa (Krueger-Hadfield et al. 2013; Montecinos et al. 2017). Sampling enough individuals of each of the ploidy pools can be a very challenging task (see also Krueger-Hadfield and Hoban 2016), especially in isomorphic taxa where distinguishing haploids from diploids is sometimes impossible on the field. Krueger-Hadfield et al. (2013) used a combined approach of ploidy determination based on reproductive structures, a chemical test, and microsatellite loci in *C. crispus*. They found that ploidy determination was rarely wrong with reproductive material using either the chemical test or microsatellite genotyping and were able to determine that some diploids were fixed homozygotes at their microsatellite loci. They highlighted the importance of having several approaches and combining sources of information to determining ploidy, but not all haplodiplontic taxa have multiple methods to determine ploidy (e.g., see Krueger-Hadfield et al. 2016 in *Agarophyton vermiculophyllum*).

Determining the sample size needed to make accurate inferences is another challenge. Krueger-Hadfield and Hoban (2016) provided some of the first guidelines for estimating power under different sampling strategies of haplodiplontic populations to estimate population genetic metrics. Estimation of power is not a common practice in molecular ecology and ignoring it can lead to biased results (Hoban 2014). In general, they recommend that at least twice the number of haploids were sampled in a population as diploids (i.e., 150 haploids and 80 diploids had a power estimate of 0.96 for detecting significant genetic differentiation among populations). Those recommendations were made on the intuition that the proportion of each ploidy stage affects the genetic trajectories and the range of population genetics indices. Our results here demonstrated that the proportions of each ploidy stage may have strong effects on comparisons and interpretations of genetic and genomic indices between populations with different proportion of haploids and between sample dates. The proportion of haploids in haplodiplontic populations is particularly important as 1) it determines the number of putative recombining haplotypes that can occur in this system and 2) because genetic drift is twice as active in the haploid when compared with the diploid stage, which makes evolutionary trajectories more stochastic. Even if the sample size required depends on populations and targeted inferences, we showed here that, sample size of 30 random individuals in each genetic pool was sufficient for all the genetic indices we studied here, whereas 150 individuals would be required for assessing robust genotypic diversity using Pareto β.

Inferring clonality, and more generally, reproductive modes encompass 3 different objectives that need to be distinguished.

First Objective

If either clonal or sexual reproductive modes are suspected to be highly dominant in population but one suspects few events of the other reproductive mode, our results allow us to deliver several recommendations. The following should be expected if clonality dominates: 1) Pareto β values under 2, 2) significant linkage disequilibrium measured in the HGP and DGP separately, 3) heterozygote excesses measured as negative mean F_{IS}, and 4) high variance of variance of F_{IS} in the DGP, including some positive F_{IS} values. On the other hand, detecting the occurrence of a few clonal events in mainly sexual population will be trickier because we cannot rely on genetic indices as they are poorly identifiable in this case. We are, thus, left with counting on the observation of few repeated genotypes in the HGP and DGP, making sure that repeated genotypes are not due to either 1) low resolutive marker set, using P_{sex} and probability of identity (as recommended and used in Stoeckel et al. 2006; Arnaud-Haond et al. 2007; Villate et al. 2010) or 2) genetic differentiation between temporal genetic pools as expected in haplodiplontic populations without hysteresis mechanism. With these 2 points verified, we recommend sampling at least 150 random individuals per population and scaling the measured Pareto β values found in each ploidy pool (i.e., HGP and DGP), on the distributions that are provided here.

Second Objective

We want to rank different populations along a continuum of sexual to clonal. In this case, Pareto β would be the best index as long as the same sampling scheme and effort are applied (i.e., exceeding more than 150 individuals sampled per population). Genetic indices would be only helpful for efficiently ranking populations with clonal reproduction exceeding $c > 0.6$. If some populations were suspected to be fully sexual, it would be important to verify that at least one mechanism of hysteresis favors genetic homogenization of their 2 temporal genetic pools. This can be achieved indirectly by measuring F_{st} between haploid and diploid genetic pools sampled at the same time. With some differentiation, we expect more linkage disequilibrium in fully sexual populations than in partially clonal ones. We also expect that Pareto β values computed on fully sexual populations would be smaller than in partially clonal populations.

Third Objective

We want to quantitatively infer approximate rates of clonality. As for diplontic species (Balloux et al. 2003; De Meeûs et al. 2006; Stoeckel et al. 2021), high rates of clonality would be easily qualified and quantified, preferentially using genetic indices that are robust to sampling and whose distributions are very identifiable. Yet, and as in the case of diplontic species (Stoeckel et al. 2021), quantifying low rates of clonality remains a challenging, and even unreachable task. Reaching this goal would require dedicated methods exploiting all small variations in these indices. As such, our simulation results provide a nice training base for a future Bayesian or machine-learning approaches, even if machine-learning approaches are still poorly developed in the area of

evolutionary biology (Sheehan and Song 2016). Our results also advocate for using other forms of exploitable, identifiable genetic signals other than those only based on one-shot sampling measures, such as methods based on genetic transitions as proposed for diplontic populations (Becheler et al. 2017).

If the goal is to estimate or rank multiple populations by their proportion of haploids, our results showed that this can only be achieved with at least a rough prior on the rate of clonality in the studied populations. This can be achieved using genotyping itself. First, the Pareto β should be computed in HGP and DGP separately to estimate an approximate rate of clonality. For better quantification, researchers need to compare those values to the one obtained from a similar fully sexual population of the same population size. To obtain this estimated measure, it is possible to simulate a similar number of individuals by randomly drawing the measured allele frequencies and computing the distribution of Pareto β from those samples. Then, we can estimate the proportion of haploids from the difference of the Pareto β value measured on the whole sample, including HGP and DGP, to the Pareto β in the separate HGP and DGP using our results. The higher this difference, lower the proportion of haploids in the studied populations.

Perspectives

Considering their massive effects on genetic and genotypic diversity, inferring the respective size of haploid and diploid pools in haplodiplontic populations and the reproductive mode of populations of haplodiplontic taxa is crucial to understand their ecology and evolution, and for developing strategies with which to manage such populations. Here, we found that the recommendations made for diplontic populations (i.e., to rely on F_{IS}, specifically on the variance of F_{IS} along the genome to estimate rates of clonality, De Meeûs et al. 2006; Stoeckel et al. 2014, 2021), does not make sense for haplodiplontic taxa. This is due to the noise caused by the haploid stage on F_{IS} distributions. Pareto β seems to be the best genotypic index to use to assess both the rates of clonality and the proportion of haploids in sampled populations, whereas it was rarely, if ever used before. We also provided the first empirical prediction of effective population sizes in haplodiplontic life cycle coupled with partially clonal reproduction. These estimates aided our understanding of the joint effects of those 2 traits on the dynamics of neutral genetic diversity. The link we did here between effective population size and expected distributions of genotypic and genetic indices may also help bridge future theoretical developments as proposed by Bessho and Otto (2020) with field and experimental studies. In addition, our simulations constitute a first training base for future inference methods and a rational justification to explore dedicated inference methods based on the dynamics of evolutionary trajectories rather than on analyses using one-shot genotyping, as already successfully done for diplontic species (Becheler et al. 2017).

Despite the complexity of our results, our simulations were a simplified view of how genetic diversity may evolve with finite, mutating, partially clonal, haplodiplontic organisms. Other traits, such as dispersal mode, varying rates of clonality in haploid and diploid life stages or type of sexual reproduction (i.e., selfing vs. outcrossing, see discussion in Krueger-Hadfield 2020), may fundamentally change the results here. Thus, it is necessary to accumulate more population genetic data on various haplodiplontic species from which meta-analyses can be triggered to elucidate trends on how those important biological features shape the evolution of genetic diversity, how it drives the evolutionary trajectories of populations at short and longer terms, and ultimately shed new light, respectively, on how haplodiplontic, haplontic, and diplontic life cycles and on how reproductive modes may evolve.

Supplementary Material

Supplementary material is available at *Journal of Heredity* online.

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Conflict of Interest

The authors declare that they have no financial conflicts of interest based on the content of this article.

Author Contributions

S.S. and S.A.K.H. conceived the project; S.S. formalized scripts and series, and performed simulations; S.S. and S.A.H. interpreted data; and S.S., S.A.H., and S.A.K.H. wrote the manuscript.

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