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Cloning and Selfing Affect Population Genetic Variation in Simulations of Outcrossing, Sexual Sea Stars

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Abstract. Many sea stars are well known for facultative or obligate asexual reproduction in both the adult and larval life-cycle stages. Some species and lineages are also capable of facultative or obligate hermaphroditic reproduction with self-fertilization. However, models of population genetic variation and empirical analyses of genetic data typically assume only sexual reproduction and outcrossing. A recent reanalysis of previously published empirical data (microsatellite genotypes) from two studies of one of the most well-known sea star species (the crown-of-thorns sea star; *Acanthaster* sp.) concluded that cloning and self-fertilization in that species are rare and contribute little to patterns of population genetic variation. Here we reconsider that conclusion by simulating the contribution of cloning and selfing to genetic variation in a series of models of sea star demography. Simulated variation in two simple models (analogous to previous analyses of empirical data) was consistent with high rates of cloning or selfing or both. More realistic scenarios that characterize population flux in sea stars of ecological significance, including outbreaks of crown-of-thorns sea stars that devastate coral reefs, invasions by *Asterias amurensis*, and epizootics of sea star wasting disease that kill *Pisaster ochraceus*, also showed significant but smaller effects of cloning and selfing on variation within subpopulations and differentiation between subpopulations. Future models or analyses of genetic variation in similar study systems might benefit from simulation modeling to characterize possible contributions of cloning or selfing to genetic variation in population samples or to understand the limits on inferring the effects of cloning or selfing in nature.

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Abbreviations: BC, British Columbia; CA, California; COTS, crown-of-thorns sea star (*Acanthaster* sp.); F_{ST}, genetic difference between subpopulations; *H*, heterozygosity; OR, Oregon; SSWD, sea star wasting disease; WF, Wright-Fisher.

Online enhancements: appendices.

Introduction

Sea stars or starfish (Asteroidea) include some of the most ecologically important animals in the oceans and occupy a foundational position in the ecological literature (Paine, 1966, 1969; Lawrence, 2013; Lafferty and Suchanek, 2016). Well-known examples include outbreaking populations of the crown-of-thorns sea star (COTS; *Acanthaster* sp.) (Haszprunar and Spies, 2014; Haszprunar *et al.*, 2017) that prey on corals and devastate coral reefs around the Indo-Pacific (Birkeland, 1982; Birkeland and Lucas, 1990; Uthicke *et al.*, 2009, 2018; De'ath *et al.*, 2012; Pratchett *et al.*, 2014; Babcock *et al.*, 2016; Haywood *et al.*, 2019; Pratchett *et al.*, 2021). Invasions of soft-sediment habitats in Australia by the northern Pacific sea star *Asterias amurensis* lead to massive populations that devastate local bivalve prey and bivalve fisheries (Ward and Andrew, 1995; Byrne *et al.*, 1997, 2013; Goggin, 1998; Parry and Cohen, 2001; Lawrence, 2013; Richardson *et al.*, 2016). In the northeastern Pacific, epizootic occurrences of sea star wasting disease (SSWD) cause mass mortality of many sea stars, including *Pisaster ochraceus*, and trigger widespread disruption of ecological interactions due to reduced predation (Bates *et al.*, 2009; Hewson *et al.*, 2014; Eisenlord *et al.*, 2016; Menge *et al.*, 2016; Montecino-Latorre *et al.*, 2016; Miner *et al.*, 2018; Moritsch and Raimondi, 2018; Schiebelhut *et al.*, 2018; Harvell *et al.*, 2019; Kay *et al.*, 2019; Ruiz-Ramos *et al.*, 2020).

The boom-and-bust nature of demographic variation in these and other echinoderm species is expected to have significant effects on population genetic variation (Uthicke *et al.*, 2009). Models and quantitative analyses of such genetic variation typically assume that populations consist of demes or subpopulations (with gonochoric outcrossing individuals that mate at random with each other) linked to each other by gene flow and shared population history. Important life-history traits that shape population genetic variation in such systems include body size and fecundity, maturation time and longevity, seasonality of gonad development, fertilization ecology, parental brood care, dispersal potential, and the mode and duration of larval development (Marko, 2004; Bradbury *et al.*, 2008; Cowen and Sponaugle, 2009; Hellberg, 2009; Marko *et al.*, 2010; Marko and Hart, 2011; Doll *et al.*, 2021; Ebert, 2021).

Most population genetic analyses of sea stars do not explicitly take into account the contributions of non-outcrossing modes of reproduction common in sea stars. Occasional or facultative fission and other forms of cloning by splitting of the adult body into two or more parts (each with one or more arms) are widespread and well known among some sea star species (Achituv and Sher, 1991; Alves *et al.*, 2002; Skold *et al.*, 2002; Rubilar *et al.*, 2005; Haramoto *et al.*, 2007; Barker and Scheibling, 2008; Sterling and Shuster, 2011). In some species and lineages in which fission and cloning by adults is the dominant mode of reproduction (*e.g.*, Clements *et al.*, 2019), population genetic variation appears to be strongly affected by cloning (Garcia-Cisneros *et al.*, 2018); those results suggest that the effects of facultative fission or cloning should be detectable in other species.

More recently recognized (and possibly more significant) modes of asexual reproduction include budding, fission, or other forms of cloning by planktonic sea star larvae (Bosch *et al.*, 1989; Bosch, 1992). These modes of cloning have been well documented in laboratory cultures and in collections of wild-caught larvae from the plankton (Jaeckle, 1994; Lacalli, 2000; Vickery and McClintock, 2000; Eaves and Palmer, 2003; Knott *et al.*, 2003; Janies *et al.*, 2019; Collin *et al.*, 2020; see the review by Allen *et al.*, 2018), including both wild (see photos in Suzuki *et al.*, 2016) and cultured (Allen *et al.*, 2019) larvae of COTS. Because larvae are much more abundant than adults, but smaller and less likely to be caught in the act of fission, this more stealthy mode of asexual reproduction could have much more substantial effects on population genetic variation than fission by adult sea stars.

Similarly, the occasional or facultative occurrence of self-fertile hermaphrodites is also taxonomically widespread among echinoderm species that are nominally gonochoric (Moore, 1932, 1935; Komatsu and Oguro, 1972; Komatsu *et al.*,1979; Yamaguchi and Lucas, 1984; Lawrence, 1987; Byrne *et al.*, 2003), including some species of *Asterias* and *Acanthaster* that are of considerable ecological interest (Retzius, 1911; Morris, 2002; Guerra *et al.*, 2020). Hermaphrodites in such species have been implicitly assumed to be relatively rare, but their frequency is generally not known. In species and lineages in which all individuals are hermaphrodites and self-fertilization is expected to be widespread, population genetic variation appears to be dominated by the effects of selfing (*e.g.*, Puritz *et al.*, 2012). Like the effects of fission and cloning, these effects of hermaphroditism and selfing on population genetic differences between species suggest that the effects of facultative selfing by hermaphrodites could also affect population genetic variation within nominally gonochoric outcrossing species (Jarne and Auld, 2006).

These well-known effects of cloning and selfing on genetic variation in comparisons between sea star species with and without these alternative modes of reproduction are consistent with theoretical predictions (Balloux *et al.*, 2003) and simulations (Stoeckel *et al.*, 2021) of cloning or selfing effects on genetic variation (Halkett *et al.*, 2005; Arnaud-Haond *et al.*, 2007; Reichel *et al.*, 2016; Stoeckel *et al.*, 2021). However, the population genetic effects of facultative cloning and selfing within gonochoric sexual species of echinoderms and other marine invertebrates with nominally sexual outcrossing reproduction (*e.g.*, Uthicke *et al.*, 1998) are less well known (Reichel *et al.*, 2016). It may be especially important to estimate the rate of clonal reproduction in partially clonal groups of ecological significance, such as invasive species (*e.g.*, Ryan *et al.*, 2021) or endangered foundational species (*e.g.*, Reynes *et al.*, 2021); but the theory predicts that such estimates will be accurate only with repeated temporal sampling of populations (*e.g.*, Ali *et al.*, 2016; Becheler *et al.*, 2017). Because frequent and widespread sampling of populations over time is not feasible for many populations of rare, endangered, or inaccessible species, there seems to be an ongoing need for the implementation and development of both simulation-based and sample-based population genetic approaches to estimate the contributions of cloning and selfing to demographic variation.

Recently, Uthicke *et al.* (2021) used classic population genetic analytical methods to search for evidence of cloning or selfing in two previously published population genetic datasets from studies of COTS (Yasuda *et al.*, 2009; Harrison *et al.*, 2017). (Although Haszprunar *et al.* [2017] proposed the name *Acanthaster* cf. *solaris* for the Pacific species of COTS, and that name has been used by Uthicke *et al.* [2021] and others [*e.g.*, Guerra *et al.*, 2020] there is renewed uncertainty about the taxonomy of that species and the origin and identity of the type specimens [see Haszprunar and Spies, 2014]. Here we refer to the Pacific species of COTS as *Acanthaster* sp. pending a future taxonomic revision.) Uthicke *et al.* (2021) noted that larval cloning has been observed in this species in the lab and that hermaphrodites have been found in the wild; but they argue that their results were not consistent with the expected effects of facultative cloning and selfing in nature, including reduced heterozygosity for individual loci across multiple individuals and subpopulations and the occurrence of pairs of clones (individuals with the same multilocus genotype). Uthicke *et al.* (2021) concluded that cloning and selfing

did not influence the pattern of genetic variation within and between individuals in those two studies and that these alternative modes of reproduction are probably rare in nature.

Here we reconsider those conclusions. Instead of applying analytical methods to empirical data to infer possible contributions of cloning or selfing, we used individual-based simulations to model population genetic variation with and without cloning or selfing. We modeled two datasets like those analyzed by Uthicke *et al.* (2021) consisting of small numbers of loci for many individuals in a simplified population structure that reflects the assumptions of typical population genetic analytical methods. We also modeled three realistic demographic scenarios for sea stars, including population outbreaks (*e.g.*, COTS on coral reefs), founder effects (*e.g.*, colonization and invasion of Australia by *A. amurensis*), and bottlenecks (*e.g.*, mortality of *P. ochraceus* caused by SSWD epizootics). In all of those models and scenarios, we readily detected the expected effects of cloning and selfing on population genetic variation, including genetic variation within subpopulations (mean heterozygosity, lower with selfing), genetic differences between subpopulations (F_{ST}, lower with cloning), and frequencies of identical multilocus genotypes (clone pairs, higher with cloning). Some of the simulation results suggested that previous empirical studies (like those reanalyzed by Uthicke *et al.*, 2021) may have unexpectedly captured some of the effects of unobserved facultative cloning and selfing in nature and that these processes might be more common (and have more important effects on population genetic variation) than we and others have previously assumed. We hope that our results might motivate other researchers to consider the possible effects of hermaphroditism, selfing, fission, and larval cloning in future surveys of population genetic structure, adaptive molecular evolution, and other population genetic analyses in these and other sea star species. The addition of simulations with and without these alternative modes of reproduction could help to identify expected contributions of cloning or selfing to observed genetic variation in population samples or help to define the limits on inference of those effects under different population models.

Materials and Methods

We used SLiM version 3.4 (Haller and Messer, 2019) to create Wright-Fisher (WF) population models with discrete generations, random mating within subpopulations, hermaphroditic individuals, equal male and female allocation, and fixed subpopulation sizes (N). SLiM uses scripts in the Eidos language (Haller, 2016) to simulate the evolution of genetic variation forward in time. We wrote Eidos scripts to simulate genetic variation in two simple population models that differ in subpopulation structure and incorporate the assumptions underlying the data analyses in two previous empirical studies of microsatellite variation in COTS. For each model, we wrote nine similar scripts that combine three different values of two parameters representing rates of self-fertilization or rates of asexual reproduction (cloning). We used those two models to ask how the addition of clonal reproduction by adults or larvae, self-fertilization by hermaphroditic adults, or the combination of those two processes might have influenced the magnitude and distribution of genetic variation observed in previous empirical studies of COTS.

We also wrote scripts to simulate more realistic models of sea star populations under one of three demographic scenarios, including (i) explosive population growth during COTS outbreaks on coral reefs in eastern Australia, (ii) founder effects associated with *Asterias amurensis* invasions of estuaries in Tasmania or Victoria, and (iii) population bottlenecks in *Pisaster ochraceus* due to mortality during epizootics of SSWD in the northeastern Pacific. For each of those models, we wrote four similar scripts that combined two different values for the cloning and selfing parameters. We used those models to ask how cloning and selfing would be expected to influence population genetic variation under each of the three demographic and ecological scenarios. Details of script development and testing and explanations of the typical structure of WF models in SLiM 3.4, including some ways in which the mechanics of cloning and selfing within SLiM differ from the biology of cloning and selfing in sea stars, are given in Appendix A1 (available online).

Two models of microsatellite variation in Acanthaster *sp.*

The goal of these two models was to simulate genetic variation in a WF population that has a population structure similar to the population model assumptions underlying the two data analyses presented by Uthicke *et al.* (2021). First, we developed a model with a structure that corresponds to the analysis of COTS genotype data presented by Harrison *et al.* (2017; Fig. 1). The empirical data came from populations sampled at a relatively small spatial scale off of northeastern Australia and showed little evidence of population substructure. As a result, Uthicke *et al.* (2021) analyzed and presented data on genetic variation for the single population sample (totaling 2719 individual sea stars from 13 reef locations), with the implicit assumption that those individuals are all members of one panmictic deme (we refer to this as the Panmictic model). Second, we developed a similar model of the COTS genotype data presented by Yasuda *et al.* (2009). Those empirical data came from populations of *Acanthaster* sp. sampled on a broader spatial scale, with apparent subpopulation structure in 4 oceanographic regions; Uthicke *et al.* (2021) analyzed and presented data on genetic variation separately for those 4 subpopulations (totaling 995 individuals from 17 locations in 4 oceanographic regions). In order to reflect that data structure, our model consisted of four subpopulations or demes connected by gene flow (we refer to this as the Subdivided model). We chose realistic mutation rates (*e.g.*, Drake *et al.*, 1998; Ellegren, 2000; Kayser *et al.*, 2000; Baer *et al.*, 2007), migration rates (*e.g.*,

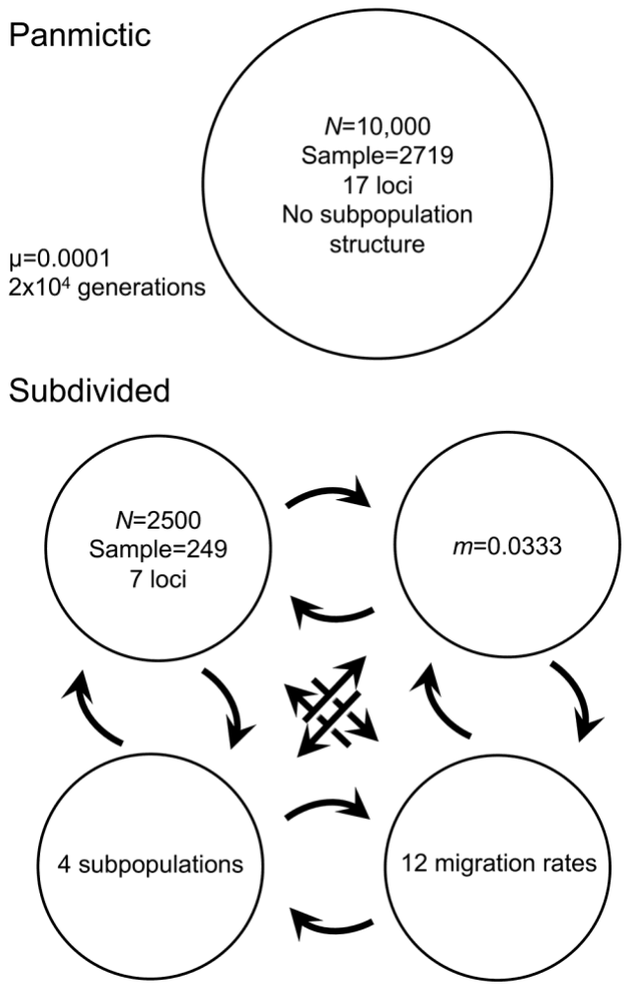

Figure 1. Graphical representation of the Panmictic model and the Subdivided model of population genetic variation in crown-of-thorns sea stars (COTS; *Acanthaster* sp.). Each circle represents a deme. In the Panmictic model, all individuals were assumed to mate randomly with each other in a single deme. In the Subdivided model, the total population consisted of four subpopulations that represent regional COTS population groups connected to each other by symmetrical gene flow in an island model; each of the 12 immigration rates (*m*; arrows) was 0.0333, so that total immigration rate into each subpopulation was 0.1. Both models assume old population histories of 20,000 generations before a random sample of individuals was drawn in the last generation.

Vogler *et al.*, 2012), and other parameters so that we observed a range of values for population genetic variables that were similar to the empirically observed population genetic variables reported by Uthicke *et al.* (2021) and Yasuda *et al.* (2009).

At the end of each simulation instance, we took a random sample of 2719 individuals (equivalent to the total population sample analyzed by Uthicke *et al.*, 2021) or a random sample of 249 individuals from each subpopulation (a total of 996, similar to the total of 995 individuals analyzed by Yasuda *et al.*, 2009), calculated mean heterozygosity *H* (the proportion of heterozygous loci averaged across all loci and individuals), and counted the total number of clone pairs (pairs of identical diploid genotypes). We used the count of clone pairs to characterize each simulation result because Uthicke *et al.* (2021) emphasized that the seemingly rare occurrence of pairs of identical diploid genomes (what they called "repeated MLG," or multilocus genotypes) was evidence of limited cloning in nature. We used mean heterozygosity because Uthicke *et al.* (2021) emphasized the expected contributions of cloning to heterozygote excess or of selfing to heterozygote deficits (relative to a population genetic model under equilibrium conditions). We compared results for 20 instances of each of 9 scripts (with different combinations of three values of the cloning rate parameters and three values of the selfing rate parameters) to the single instance reported by Harrison *et al.* (2017) or by Yasuda *et al.* (2009) and summarized by Uthicke *et al.* (2021). We also used those results to select a combination of values of the cloning rate and selfing rate parameters that gave a mean result ($n = 20$ instances) similar to the point estimates of mean heterozygosity reported in Harrison *et al.* (2017) or Yasuda *et al.* (2009) and the total number of clone pairs reported by Uthicke *et al.* (2021) for each of those empirical studies.

Models of genomic variation in three scenarios: outbreaks, founder effects, bottlenecks

The scripts for these three demographic models use many of the same script elements that we used in the Panmictic and Subdivided models, but they have a different purpose. Rather than being used to interpret patterns of genetic variation observed in analyses of previously published empirical results (for small numbers of rapidly evolving microsatellite-like loci) and the possible contributions of cloning and selfing to those previously observed results, we used these three models to predict possible associations between population genetic variation (for large numbers of loci with slower rates of evolution) and sea star demographic scenarios that are of wide interest to ecologists and conservation biologists.

Outbreaks. We modeled five subpopulations of $N = 5000$ individuals each, with stepping-stone gene flow between adjacent subpopulation pairs, corresponding to the 5 regions of the Great Barrier Reef (Lizard Island, Cooktown, Cairns, Townsville, and Swains) in which *Acanthaster* sp. individuals were sampled by Harrison *et al.* (2017; Fig. 2). We used a smaller subpopulation size compared to the Panmictic model because the last step in the simulation (counting clone pairs) involves an all-against-all comparison of mutations at all loci in both genomes of all individuals and because the computation time increases with the square of the total population size ($N = 25{,}000$). See Appendix A1 (available online) for a description of the modeled genomes.

We chose model parameters, especially the location and timing of the initiation of oubreaks, to approximate the demography of COTS outbreaks in nature (*e.g.*, Lucas, 1984; Babcock

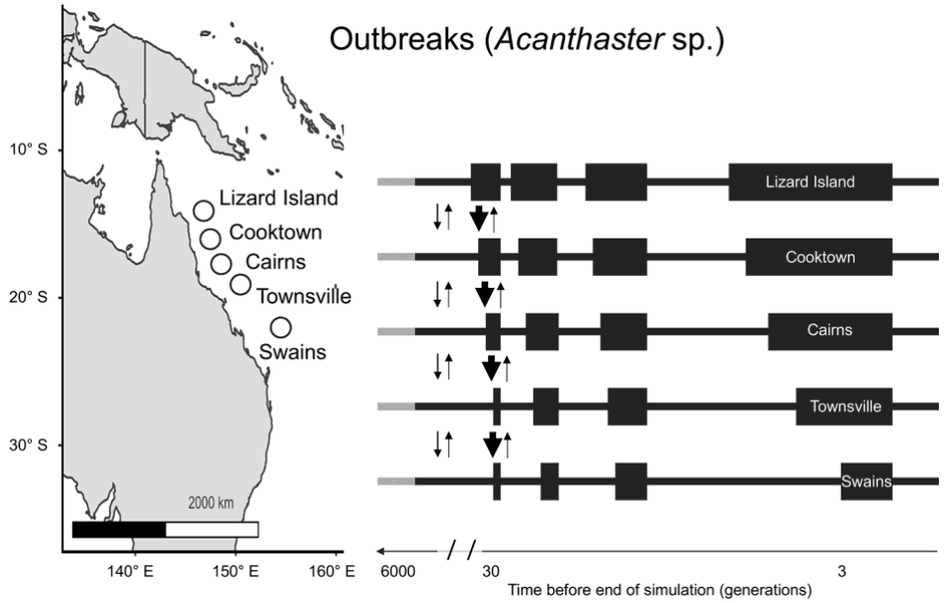

Figure 2. Graphical representation of the Outbreaks model of population genetic variation in crown-of-thorns sea stars (COTS; *Acanthaster* sp.). Each horizontal bar represents one of five subpopulations from northeastern Australia at left; the width of each bar represents the subpopulation age (note the logarithmic timescale); the gray portion of each bar shows the burn-in period (before cloning and selfing parameters are set); the height of each bar represents subpopulation size ($N = 5000$; $N = 50{,}000$ during outbreaks). Vertical arrows show asymmetrical gene flow (arrow thickness) between subpopulation pairs ($m = 0.001$ from north to south in most generations; $m = 0.0001$ from south to north in all generations; $m = 0.5$ for one generation in each subpopulation pair in each outbreak). For clarity, not all changes in migration rates (m) for all population pairs in all four outbreaks are shown.

and Mundy, 1992; Fabricius *et al.*, 2010; Caballes and Pratchett, 2014; Pratchett *et al.*, 2014, 2017; Harrison *et al.*, 2017), similar to the population model called Scenario 4 analyzed by Harrison *et al.* (2017, fig. 2d). See the model descriptions in Appendix A1 (available online) and the scripts in Appendix A2 (available online) for details. We encourage interested readers to download our model scripts, run them in the graphical user interface application called SLiMgui (Haller and Messer, 2019), and use the Population Visualization tool for an interactive view of the model dynamics.

Like the Panmictic and Subdivided models, we chose values for some of the parameters (especially the outbreak population sizes, $N = 50{,}000$) as a compromise between ecological realism (to reflect the extraordinary increase in local numbers of COTS during outbreaks) and model tractability, especially the pairwise comparison of all loci in all genomes to count pairs of clones. We have not thoroughly explored the consequences of different choices for these parameter values. At the end of each instance of each script, we calculated three population genetic variables for the whole population: mean heterozygosity H (all loci in all individuals in all five subpopulations); F_{ST} between the two most distant subpopulations (all individuals in Lizard Island and Swains); and the number of clone pairs (between all individuals in all five subpopulations).

Founder effects. We modeled 3 subpopulations of $N = 10{,}000$ each that represent a source subpopulation in the native range of *Asterias amurensis* in the northwestern Pacific and 2 subpopulations in the southwestern Pacific that invaded and successfully colonized estuarine habitats of Australia associated with the Derwent River estuary in Tasmania and Port Phillip Bay in Victoria (Fig. 3). We chose model parameters that reflected previous inferences about the origin, timing, and population biology of that colonization *via* ballast-water transfer of planktonic larvae (Ward and Andrew, 1995; Andrew, 1998; Goggin, 1998; Murphy and Evans, 1998; Richardson *et al.*, 2016). We designed our model to reflect that plausible population history, but we have not explored other plausible histories or patterns and how they would interact with the effects of cloning or selfing on simulated genetic variation. We used the same model genome, genomic element types, mutation types, mutation rate, recombination rates, and cloning or selfing rates as in the Outbreaks model (see Appendix A2 (available online)), but with no gene flow between subpopulations following either of the colonization events. We used an initial size $N = 2$ for each founder event, which represented a pair of spawning adults that generated the larvae to initiate each colonization, followed by exponential growth in population size up to $N = 10{,}000$. This is equivalent to starting the founder event with two introduced adult individuals that subsequently reproduced sexually to initiate the first generation of each invasive subpopulation. Transport of adults or juveniles is also considered to be a possible scenario for introduction of

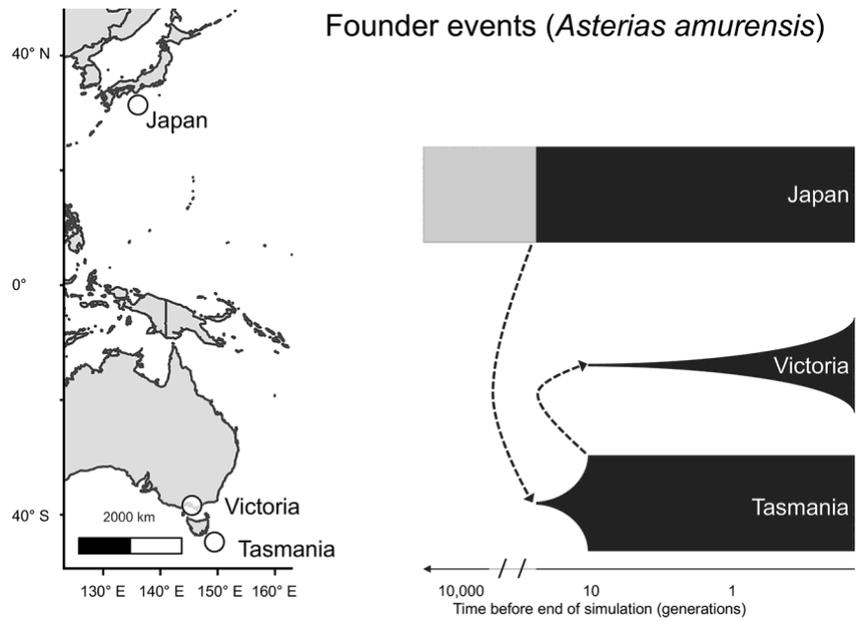

Figure 3. Graphical representation of the Founder effects model of population genetic variation in *Asterias amurensis*. Each horizontal bar represents one of three subpopulations from Japan, southern Australia, or Tasmania at left; the width of each bar represents the subpopulation age (note the logarithmic timescale); the gray portion of each bar shows the burn-in period (before cloning and selfing parameters are set); the height of each bar represents subpopulation size (maximum $N = 10{,}000$, minimum $N = 2$ at each founder event). Dashed arrows show two founder events (invasion of the Derwent River estuary in Tasmania, then invasion of Port Phillip Bay in Victoria); curved edges show exponential population growth after each subpopulation split. There is no gene flow in this model.

A. amurensis to Tasmania. We have not explored the consequences of larger initial subpopulation sizes at colonization (representing a larger number of spawning adults that contributed gametes to the pool of planktonic larvae in the ballast-water introduction).

Similar to the model of COTS outbreaks, we developed that model as a compromise between realism and tractability. In particular, the population sizes are several orders of magnitude smaller than census populations sizes in the Derwent River estuary (see Goggin, 1998). We have not explored the possible importance of modeling millions (rather than thousands) of individuals. At the end of each instance of each script, we calculated H (all loci in all individuals in the two invasive subpopulations in Tasmania and Victoria), F_{ST} between the invasive subpopulations, and the number of clone pairs in each of the invasive subpopulations. Unlike the model of COTS outbreaks (where we included all subpopulations in these calculations), we focused only on population genetic variables for the two invasive subpopulations, and we ignored the Japanese source subpopulation because we meant to focus only on the potential contribution of cloning or selfing to genetic variation associated with the colonization and invasion events.

Bottlenecks. This model combined elements of both the Outbreaks model (in *Acanthaster*) and the Founder effects model (in *Asterias*), including population splitting, exponential population growth, stepping-stone gene flow, and spatial variation in model parameter values between some subpopulations. We modeled three subpopulations of *Pisaster ochraceus* of $N = 10{,}000$ each that represent sea stars from different parts of the broad geographical range of this species in the northeastern Pacific that have different population histories and have experienced different intensities of mortality from SSWD (Eisenlord *et al.*, 2016; Menge *et al.*, 2016; Miner *et al.*, 2018; see also Hodin *et al.*, 2021; Fig. 4). These include older subpopulations in California (CA) and Oregon (OR) and a younger subpopulation in British Columbia (BC) that was probably established by a range expansion following the end of the last Pleiostocene glaciation (Marko *et al.*, 2010). Populations of *P. ochraceus* and other species in California have experienced more severe mortality from SSWD in comparison to ecologically significant but quantitatively less severe mortality in some populations to the north (OR, BC) (Miner *et al.*, 2018). We used the same model genome, genomic element types, mutation types, mutation rate, recombination rates, and cloning or selfing rates as in the Outbreaks model and the Founder effects model. We simulated three cycles of mortality and population bottlenecks (representing the effects of three SSWD epizootics). Those three cycles could represent the combined effects of two cryptic or only partially documented disease events starting in the late twentieth century, followed by a third well-documented epizootic that resulted in the ecologically disastrous mortalities of *P. ochraceus* and

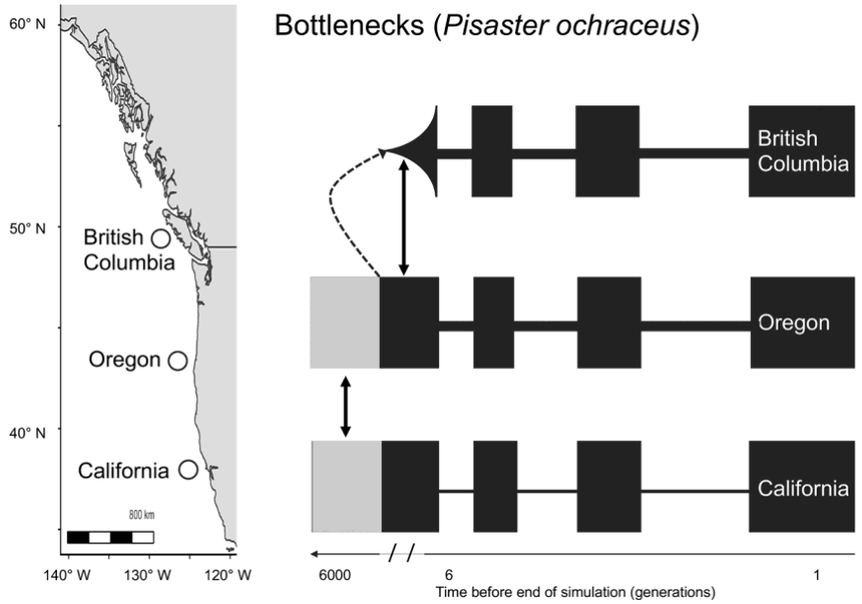

Figure 4. Graphical representation of the Bottlenecks model of population genetic variation in *Pisaster ochraceus*. Each horizontal bar represents one of three subpopulations from the northeastern Pacific at left; the width of each bar represents the subpopulation age (note the logarithmic timescale); the gray portion of each bar shows the burn-in period (before cloning and selfing parameters are set); the height of each bar represents subpopulation size (maximum $N = 10{,}000$, minimum $N = 10$ in California after sea star wasting disease). The dashed arrow shows the subpopulation split (establishment of the British Columbia subpopulation at the end of the last Pleisotocene glaciation); curved edges show exponential population growth after the subpopulation split. Double-headed arrows show symmetrical gene flow between subpopulation pairs ($m = 0.001$ throughout the simulation).

other species starting in 2013 (Miner *et al.*, 2018). Alternatively, this model could represent the effects of a first epizootic starting in 2013 plus the predicted effects of two subsequent SSWD events that may be reasonably expected to recur in association with future cycles of ocean warming and increases in pathogen abundance or infectivity (Eisenlord *et al.*, 2016). At the end of each instance of each script, we calculated H (all loci in all individuals in all three subpopulations), F_{ST} between adjacent subpopulations that differ in the intensity of SSWD mortality (CA, OR) and between subpopulations that differ in population history (OR, BC), and the number of clone pairs (between all individuals in all subpopulations).

Analysis of population genetic variables

We found large differences in population genetic variables between our simulations of cloning and selfing effects on microsatellite variation (in the Panmictic model and the Subdivided model) relative to published patterns of microsatellite variation in COTS (Yasuda *et al.*, 2009; Harrison *et al.*, 2017). We present graphical or text summaries of those results without a quantitative analysis.

We found more subtle population genetic variation between our simulations of cloning and selfing effects on mean heterozygosity H or on F_{ST} between subpopulation pairs in the Outbreaks, Founder effects, and Bottlenecks models. For those three model results, we used two-way ANOVA in Rstudio (R Core Team, 2018) to identify significant effects of cloning, selfing, or their interaction; and we used those results to ask whether mean outcomes from replicate instances of different scripts (with different parameter value combinations) were consistent with the null hypothesis that those mean outcomes are drawn from the same single distribution of values. Because SLiM simulations are deterministic, any parameter value difference (or other difference in the model structure) will affect the simulation outcomes in a systematic way; in particular, a model difference such as the zero or non-zero value of the cloning or selfing rate parameters must generate a statistically significant difference in mean outcomes if results from a sufficiently large number of instances of the two scripts are compared. An alternative interpretation of our ANOVA results is to ask whether the mean outcomes from a reasonably large number of instances (100 for all scripts of all models) were consistent with the expected differences that should arise, given the parameter value differences between scripts (with or without cloning or selfing) and given the stochastic differences between instances of the same script with the same combination of parameter values but a different random number of seeds.

In all three of those models, we found large differences in the counts of clone pairs (especially associated with differences between scripts in the cloning rate parameter). Like

our simulations of microsatellite variation (in the Panmictic and Subdivided models), we present summaries of those differences but without quantitative analysis of their statistical significance.

Results

Two models of empirical population genetic variation in Acanthaster *sp.*

In these simple models, we used a broad range of parameter values for both the cloning and selfing rates (from 0 to 0.3) and observed a wide range of results for both mean heterozygosity ($0.6 < H < 0.9$) and number of clone pairs (up to 59). In the Panmictic model (Fig. 5), low cloning or selfing rates (0 and 0.05, respectively) generated relatively high heterozygosities and few clone pairs. The highest selfing rate (0.3) greatly reduced heterozygosity, and the highest cloning rate (0.3) generated many more observed clone pairs, as expected. In the Subdivided model (Fig. 6), we observed many fewer clone pairs overall but a similar overall effect of selfing and cloning on genetic variation. See Appendix A2 (available online) for code and individual results.

In general, the simulations suggested that for the model structure and parameter values used, cloning or selfing rates greater than zero were needed to generate mean heterozygosity values and counts of clone pairs similar to those observed in empirical data. In the Panmictic model, we were readily able to find parameter values (cloning = 0.17, selfing = 0.21) for which results from multiple instances were closely clustered around the mean heterozygosity ($H = 0.67$) and counts of clone pairs (12) in the empirical results of Harrison *et al.* (2017; Fig. 7). In the Subdivided model, a cloning rate of 0.33 (with no selfing) generated a cluster of instances with population genetic variation similar to the empirical results ($H = 0.79$, 6 clone pairs) of Yasuda *et al.* (2009).

We emphasize that these simulation results should not be interpreted as estimates of the rate of cloning or selfing by COTS in nature, which are expected to be a challenge to estimate by using sampling schemes that are typical of empirical studies (Stoeckel *et al.*, 2021). The results from our simulations depend

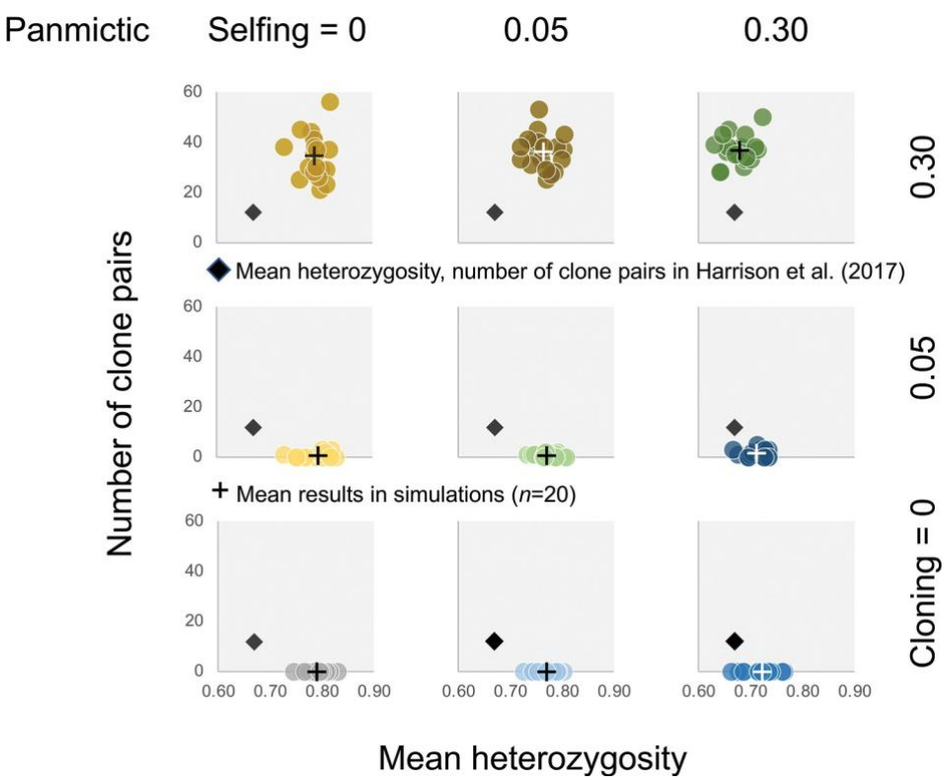

Figure 5. Mean heterozygosity (H) averaged across loci and all individuals in the population, and number of clone pairs, in the Panmictic model of population genetic variation in crown-of-thorns sea stars (*Acanthaster* sp.). Each symbol represents the result from one instance of a script with a unique random number seed and one of nine combinations (indicated by colors) of three selfing rates (0, 0.05, 0.3) and three cloning rates (0, 0.05, 0.3). In each data graphic, the plus sign indicates the mean value of H and the mean count of clone pairs for 20 instances of the script; the black diamond indicates the value of H and number of clone pairs reported by Uthicke *et al.* (2021) based on reanalysis of empirical microsatellite data from *Acanthaster* sp. in Harrison *et al.* (2017).

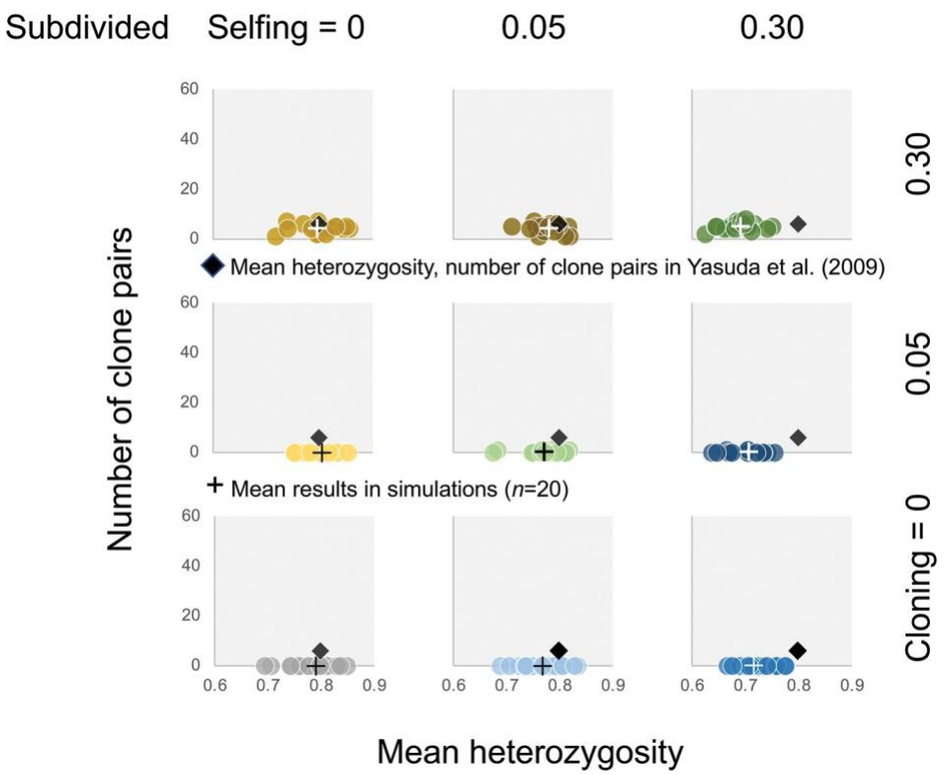

Figure 6. Mean heterozygosity (H) averaged across loci and all individuals in the population, and number of clone pairs, in the Subdivided model of population genetic variation in crown-of-thorns sea stars (*Acanthaster* sp.). Each symbol represents the result from one instance of a script with a unique random number seed and one of nine combinations (indicated by colors) of three selfing rates (0, 0.05, 0.3) and three cloning rates (0, 0.05, 0.3). In each data graphic, the plus sign indicates the mean value of H and the mean count of clone pairs for 20 instances of the script; the black diamond indicates the value of H and number of clone pairs reported by Uthicke *et al.* (2021) based on reanalysis of empirical microsatellite data from *Acanthaster* sp. in Yasuda *et al.* (2009).

on other parameter values and model structural features, in particular, on the mutation rate. Many other model results could be simulated using different model structures and other combinations of parameter values (and this is the approach used in a wide variety of approximate Bayesian computation methods for inferring population demographic parameter values from empirical data). Instead, our results should be interpreted more simply, as indicators that the empirical results from previous analyses of small numbers of microsatellite loci may be consistent with a wide range of non-zero values for selfing and cloning rates by COTS in nature, including some relatively high rates of cloning or selfing or both (Fig. 7).

Models of genomic variation in three scenarios: outbreaks, founder effects, bottlenecks

Like the two simple models of few loci, our more realistic models of demographic variation also showed that non-zero values of the cloning and selfing parameters affected mean heterozygosity (H, with a larger effect of selfing compared to cloning), subpopulation differences in allele frequencies (F_{ST}, also with a larger effect of selfing), and the number of clone pairs (with a much greater effect of cloning). Appendix A2 (available online) includes the code for each model, results from individual instances of each script, and a two-way ANOVA used to test hypotheses about differences between means from scripts with and without cloning or selfing.

Outbreaks. Selfing reduced mean heterozygosity by about 15% in total in this model (Fig. 8) from $H \sim 0.018$ (mean across all 100 instances) to $H \sim 0.016$; cloning also decreased heterozygosity overall, but the selfing effect was larger than the effect of cloning (Fig. 8). In the two-way ANOVA, both of those main effects on heterozygosity were highly significant ($P \ll 0.001$), but the interaction between them was not significant for this number of instances of the model ($P = 0.121$).

Effects of cloning or selfing on F_{ST} (between the Lizard Island and Swains subpopulations) were much smaller. Selfing increased F_{ST} from 0.0029 to 0.0030 without cloning (Fig. 8) and by a similar proportion with cloning; for this large number of instances (100), that small of an effect of selfing on F_{ST} was highly significant in the two-way ANOVA ($P \ll 0.001$). Cloning led to slight reductions in F_{ST} both with and without selfing, but this small effect was not significant ($P = 0.16$).

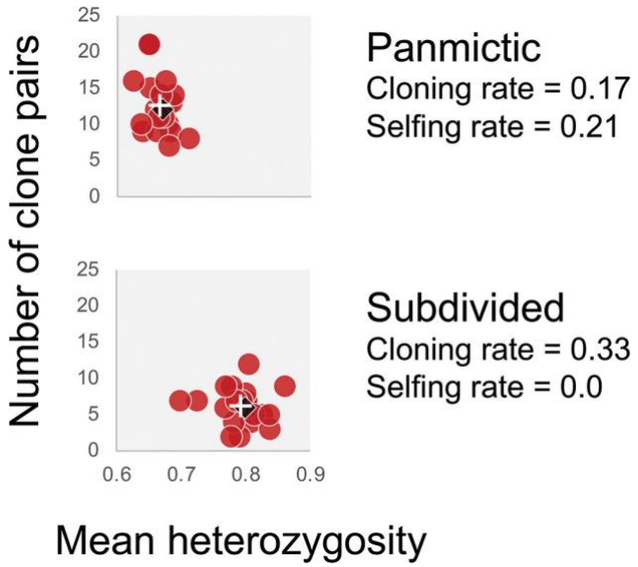

Figure 7. Mean heterozygosity (H) averaged across loci and all individuals in the population, and number of clone pairs, in the Panmictic model and in the Subdivided model population genetic variation in crown-of-thorns sea stars (*Acanthaster* sp.). Each symbol represents the result from one instance of a script with a unique random number seed and a selected combination of the cloning and selfing rate parameters chosen so that the mean value of H and the mean count of clone pairs for 20 instances of the script (plus sign) were very similar to the value of H and the number of clone pairs (black diamond) reported by Uthicke *et al.* (2021) based on reanalysis of empirical microsatellite data from *Acanthaster* sp. in Harrison *et al.* (2017) or in Yasuda *et al.* (2009).

We observed no clone pairs in any of the 200 instances of either script without cloning (with or without selfing; Table 1). By contrast, we always observed at least 1 clone pair in all 200 instances of both scripts with cloning, and the mean number of clone pairs was similar with (7.7) and without (8.1) selfing. Those consistent results suggested that empirical discoveries of individuals with identical genomes should probably be ascribed to clonal reproduction and that in this realistic scenario one should not expect to discover pairs of individuals with identical genomes, except when clonal reproduction is occurring.

However, like our simpler models, the results of the Outbreaks model do not point to a specific rate of cloning that could be inferred from a count of clone pairs in empirical data. Although the Outbreaks model was designed to mimic COTS demographic variation, and the counts of clone pairs from the Outbreaks model (7–8 clone pairs) were similar to the empirical counts of clone pairs in the multilocus COTS genotype data of Harrison *et al.* (2017; 12 clone pairs; see Uthicke *et al.*, 2021), the comparison between the model and the empirical results is not direct because the total sample of individuals is much larger in our simulations ($N = 25{,}000$, or $\sim 3.1 \times 10^8$ pairwise comparisons; Table 1) than the sample of individuals in the empirical data of Harrison *et al.* (2017; $N = 2719$, or $\sim 3.6 \times 10^6$ pairwise comparisons).

Founder effects. Similar to the Outbreaks model, we found highly significant effects of selfing on both H and F_{ST} (both $P \ll 0.001$). The magnitude of the selfing effect on heterozygosity was also similar to the Outbreaks model (about 10% decrease; Fig. 9). The effect of selfing on F_{ST} was larger in the presence of cloning (increased from $F_{ST} \sim 0.020$ to $F_{ST} \sim 0.022$) than in the absence of cloning (from 0.020 to 0.021; Fig. 9), but that interaction effect was marginally not significant ($P = 0.056$). By contrast, we found no main effects of cloning on heterozygosity or population differentiation in this model.

Like the Outbreaks model, we found no clone pairs in simulations where the cloning and selfing rates were both 0 (Table 1), but we found a few (up to 4) clone pairs in some (7) instances of the model with selfing = 0.1. These are identical pairs of genotypes that arose due to selfing, following the effects of one or both founder events. Like the Outbreaks model, we always found some clone pairs (up to 48) in all 200 instances of both scripts with cloning. Most of those clone pairs occurred in the Tasmania subpopulation (which was older, was larger in most generations, and had more opportunities to accumulate identical genotypes).

Bottlenecks. The effects of cloning and selfing on heterozygosity in this model were similar to the Outbreaks model. Selfing decreased H by about 10% both with and without cloning (Fig. 10); the effects of cloning were much smaller (Fig. 10), but both effects were highly significant in the two-way ANOVA ($P \ll 0.001$).

We separately analyzed the effects of cloning and selfing on pairwise population differentiation (between CA and OR and between OR and BC) in order to distinguish the possible contribution of differences in mortality rate (CA, OR) and differences in population history (OR, BC). However, we found very similar effects of cloning and selfing on both of those pairwise population differences. Selfing increased population differentiation between the CA and OR subpopulations from $F_{ST} \sim 0.012$ to $F_{ST} \sim 0.013$ both with and without cloning (Fig. 10); F_{ST} was slightly lower with cloning, but the effect was much smaller (about a 1% decrease) compared to the effect of selfing (about a 10% increase). The selfing effect was highly significant ($P \ll 0.001$), whereas the cloning effect was only marginally significant ($P = 0.044$).

Differentiation between the OR and BC subpopulations showed a similar pattern, with slightly weaker effects overall. Selfing increased differentiation by about 3% from $F_{ST} \sim 0.0069$ to $F_{ST} \sim 0.0071$ both with and without cloning, and the main effect of selfing was highly significant ($P \ll 0.001$). Cloning decreased differentiation by a smaller proportion (about 1%), and that effect was marginally not significant ($P = 0.062$).

We found larger numbers of clone pairs in this model compared to all others and greater variation between scripts (with and without cloning or selfing). We found no clone pairs in any of the 100 instances without cloning and without selfing (Table 1). Many instances of the model with selfing alone also included zero clone pairs; but other instances often included some clone pairs (mean = 13), and clones were common in

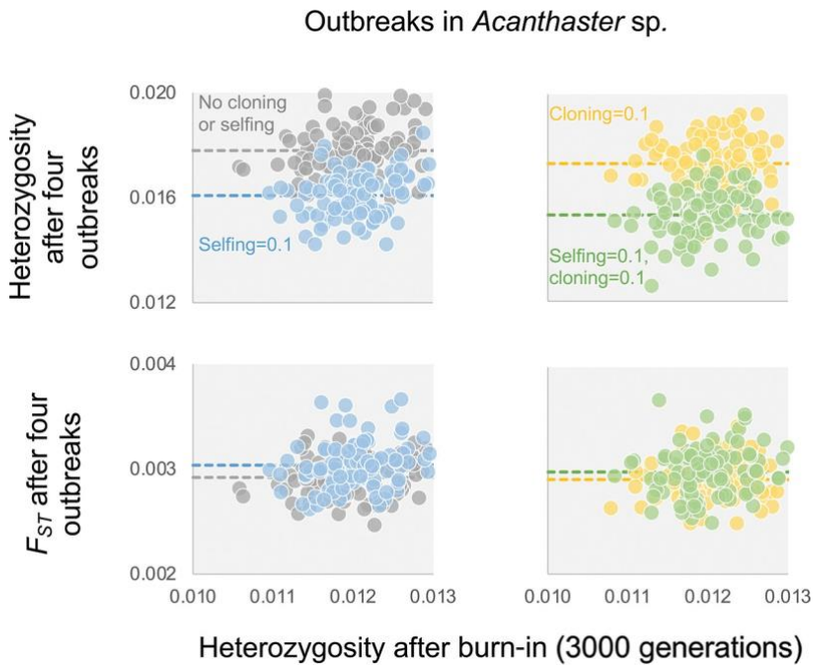

Figure 8. Mean heterozygosity (H) or genetic differences between subpopulations (F_{ST}) between the Lizard Island and Swains subpopulations in the Outbreaks model of population genetic variation in crown-of-thorns sea stars (*Acanthaster* sp.). Each symbol represents the result from one instance of a script with a unique random number seed and one of four combinations (indicated by colors) of selfing rates (0, 0.1) and cloning rates (0, 0.1). Each value is plotted against H after the burn-in period of the simulation (a measure of stochastic variation between identical instances of the model). Horizontal dashed lines show mean values of the dependent variable at the end of the simulation for 100 instances of each script.

a few instances (up to 711). Like the Founder effects model, these clone pairs in the Bottlenecks model can be ascribed to the effects of selfing in small populations after one or more of the SSWD outbreaks at the end of the simulation.

We found the largest number of clone pairs in the Bottlenecks model with cloning (>37,000 pairs; Table 1). This large effect could be attributed to accumulation of and mating between clones over a series of severe bottleneck events closely spaced in time. Although the total number of clone pairs seems very large, it represents a small proportion (~0.008%) of the total number of pairwise genotype comparisons (~4.4 × 10^8) among all 30,000 individuals at the end of each simulation.

Discussion

Although cloning and hermaphroditism are taxonomically widespread among sea stars, analyses of the population genetic effects of such traits have been limited to species in which fission by adults is the dominant mode of reproduction (Garcia-Cisneros *et al.*, 2018) or to species with obligate hermaphroditic mating systems and self-fertilization (Puritz *et al.*, 2012). Uthicke *et al.* (2021) argued that the population genetic effects of facultative cloning (by larvae) and selfing in COTS were not detectable in previously published empirical data and dismissed apparent evidence of clones as sampling errors. Our simulations of those analyses suggest instead that those previously published empirical results were consistent with non-zero rates of cloning or selfing or both. Simulations of three realistic demographic scenarios suggest that both cloning and selfing can shape the population genetic patterns resulting from ecologically important processes, including explosive population growth, founder events, or mortality from epizootics.

The five relatively simple models that we used do not directly show that cloning or selfing is occurring in nature among nominally gonochoric sea stars with sexual reproduction. They

Table 1

Counts of clone pairs in the Outbreaks, Founder effects, and Bottlenecks models

	Cloning = 0		Cloning = 0.1	
Model	Selfing = 0	Selfing = 0.1	Selfing = 0	Selfing = 0.1
Outbreaks	0	0	8.1	7.7
Founder effects	0	0.11	31	32
Bottlenecks	0	13	37,805	37,459

All counts are means from 100 instances of the same script; 0 indicates that all 100 instances included no clone pairs.

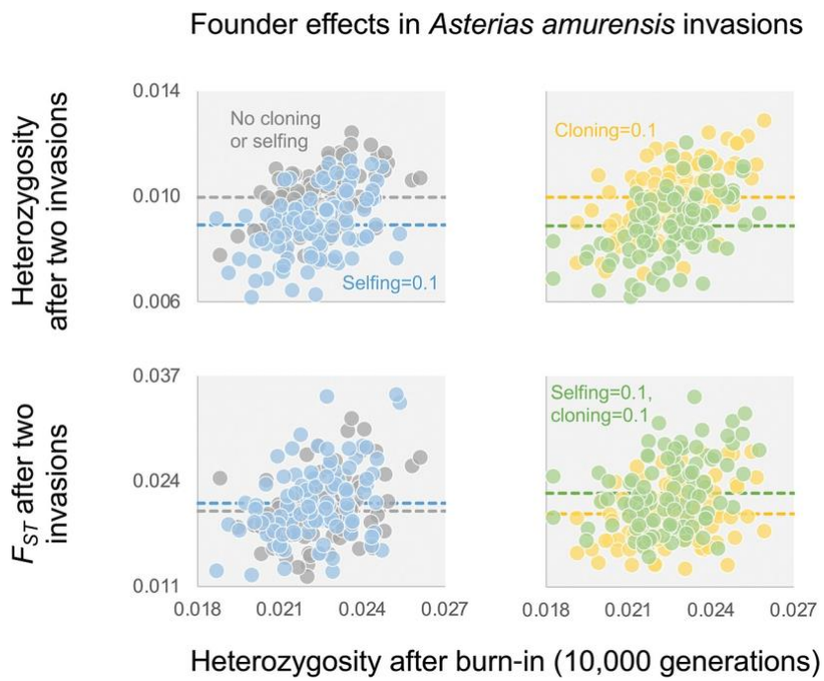

Figure 9. Mean heterozygosity (H) or genetic differences between subpopulations (F_{ST}) between the two invasive subpopulations (Tasmania, Victoria in the Founder effects model of population genetic variation in *Asterias amurensis*. Each symbol represents the result from one instance of a script with a unique random number of seeds and one of four combinations (indicated by colors) of selfing rates (0, 0.1) and cloning rates (0, 0.1). Each value is plotted against H after the burn-in period of the simulation (a measure of stochastic variation between identical instances of the model). Horizontal dashed lines show mean values of the dependent variable at the end of the simulation for 100 instances of each script.

do, however, suggest that it may be difficult to rule out cloning or selfing effects in a single instance based on empirical data from a population sample. Our results suggest, in particular, that the discovery of multiple pairs of identical multilocus genotypes in population samples is likely to indicate cloning or selfing in nature (Figs. 5, 6; Table 1).

We used simple models that do not include overlapping generations, frequency-dependent or context-dependent selection, explicit spatial structure, epistasis, quantitative trait variation, and other features of genes, organisms, and populations that can be added to forward simulations in SLiM (Haller and Messer, 2019; Riggs *et al.*, 2021). An advantage of this simplicity is that our simulation results may be generally applicable to understanding the population genetic consequences of cloning and selfing by sea stars. This simulation approach has not previously been used to understand population genetic variation in asteroids. However, our results are consistent with the expected effects of cloning and selfing on heterozygosity within subpopulations (lower with selfing) and allele frequency differences between subpopulations (lower with cloning), based on the extensive literature from studies of mixed mating systems in plants. Temporal, spatial, and ecological variation in rates of selfing (or outcrossing) and asexual propagation in these systems are known to have important effects on genetic variation within subpopulations as well as divergence between subpopulations and species (Clauss and Mitchell-Olds, 2006; Mable and Adam, 2007; Rasmussen and Kollmann, 2008; Arnaud-Haond *et al.*, 2020).

We found few small differences between cloning or selfing effects on population genetic variables associated with massive gene flow (Outbreaks), some gene flow (Bottlenecks), or no gene flow (Founder effects). For example, selfing reduced heterozygosity by a similar proportion in the Outbreaks model (Fig. 8) and in the Bottlenecks model (Fig. 10), with considerably different magnitudes and symmetries of gene flow. Similarly, we found little difference in cloning or selfing effects on H and F_{ST} between models of large populations that experience transient massive increase in size (Outbreaks) or models with transient severe decrease in size (Founder effects, Bottlenecks). This suggests that our simulation results and our conclusions should not be very sensitive to our choices of model structure and parameter values. We found just one large difference between models: the strong effect of cloning on the occurrence of clone pairs in the Bottlenecks model. That difference may be related to the series of severe bottleneck events over a few generations in that model, but we have not explored the sensitivity of that result to changes in the timing or severity of SSWD.

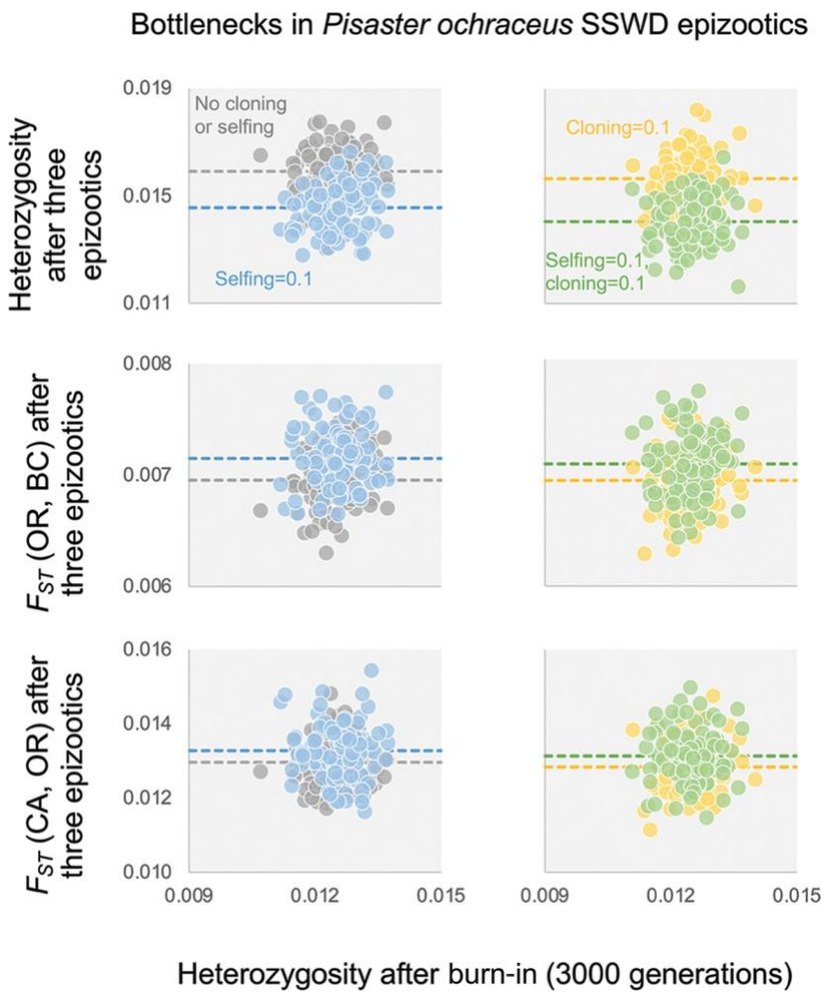

Figure 10. Mean heterozygosity (H), genetic differences between subpopulations (F_{ST}) between the Oregon (OR) and British Columbia (BC) subpopulations, or F_{ST} between the California (CA) and OR subpopulations in the Bottlenecks model of population genetic variation in *Pisaster ochraceus*. Each symbol represents the result from one instance of a script with a unique random number seed and one of four combinations (indicated by colors) of selfing rates (0, 0.1) and cloning rates (0, 0.1). Each value is plotted against H after the burn-in period of the simulation (a measure of stochastic variation between identical instances of the model). Horizontal dashed lines show mean values of the dependent variable at the end of the simulation for 100 instances of each script. SSWD, sea star wasting disease.

Our results suggest that empirical population genetic patterns found in sea star population samples might be shaped in part by unrecognized cloning or selfing in nominally gonochoric species. How could empirical studies (including our own studies of sea star phylogeography and adaptive molecular evolution) take this potential effect into account? It may be useful to do so in comparative analyses that contrast populations or species with different life-history traits or in different ecological conditions. If adaptive differences between species or ecological differences between populations include differences in rates or opportunities for cloning or selfing to occur, then some differences in population genetic variables between species or populations could reflect those cloning or selfing effects, rather than other more traditional considerations in comparative phylogeography (*e.g.*, population size, gene flow, metapopulation history). Our results suggest that unrecognized cloning or selfing in some species or populations could bias such comparative results in unpredictable and interesting ways.

Accounting for facultative cloning or selfing in nature could be especially important in scenarios where these additional modes of reproduction might reflect adaptive phenotypic plasticity (*e.g.*, Sterling and Shuster, 2011). For example, larval cloning may be induced by risk of predation (Vaughn and Strathmann, 2008; Vaughn, 2009). We previously hypothesized that hermaphroditism in COTS might be adaptive as a tactic for reproductive assurance when adult sea stars live at low population density, with few nearby mates for successful

fertilization (Guerra *et al.*, 2020). Analysis of the COTS genome has emphasized the diversity and adaptations of the chemosensory abilities of COTS adults (Hall *et al.*, 2017), including pheromone signaling from predators that may promote defensive behavior (*e.g.*, Deaker *et al.*, 2021) or pheromone signaling from conspecifics that may contribute to the aggregation of individuals and destructive grazing of corals (reviewed in Motti *et al.*, 2018). Such behavior could also promote the formation of spawning aggregations. By contrast, the absence of pheromone cues from conspecifics could indicate the scarcity of nearby mates and act as a cue for the development of hermaphroditic gonads and self-fertilization as an adaptive solution to mitigate the risk of fertilization failure. Additional experiments and field surveys of the incidence of cloning and hermaphroditism under risky conditions are needed to further explore that potential for adaptive plasticity in mating system traits.

Unfortunately, our results do not point to a general solution to the need to account for the contributions of cloning and selfing to genetic variation in gonochoric sexual sea stars. One approach (like that used by Uthicke *et al.*, 2021) emphasizes counting pairs of identical multilocus genotypes and discounting the role of cloning on the basis of rare clone pairs in population samples. In some of our results, a non-zero cloning rate generated only rare clone pairs (Figs. 5, 6; Table 1), so searching for identical multilocus genotypes may sometimes fail to distinguish populations with and without cloning. In other results, a non-zero selfing rate also produced occasional identical genotype pairs (Table 1), but at rates that would be difficult to detect in small population samples and difficult to distinguish from the effects of cloning. In general, the effects of selfing on heterozygosity and population differentiation resemble the effects of other forms of inbreeding and would be difficult to recognize in empirical data from population samples.

A more positive view is that the effects of moderately high cloning and selfing rates (0.1) on H and F_{ST} were small in our simulations when considered as the mean across many instances of a single model and script. Differences were typically ~10%–15% (or less) between mean values from results with and without cloning or selfing. The largest differences we observed between any pair of instances for two different scripts of the same model for either population genetic variable were about threefold differences in F_{ST} between some instances of the Founder effects model with selfing ($F_{ST} \sim 0.035$) and without selfing ($F_{ST} \sim 0.012$) (Fig. 9). For comparative population genetic studies where selfing or cloning effects are suspected, and where the scale of differences in population genetic diversity or differentiation are like those we observed, a simulation approach like this could be used to model the expected contribution of cloning or selfing. Simulation results could be used to generate a frequency distribution of expected population genetic parameter values for different simulated rates of cloning or selfing, and the empirical observations could be compared to that distribution. That approach could help empiricists to rule out possible contributions of unobserved cloning or selfing to genetic variation in population samples or help to quantify the possible cryptic effects of cloning and selfing. In other comparative studies where the scale of differences in population genetic diversity or differentiation are much larger than those we observed, a simulation approach could be used to show that biologically reasonable rates of cloning or selfing could not account for the observed patterns of variation in population samples; this approach could also be used to rule out an important contribution of cloning or selfing to genetic variation in nature.

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