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Experimental separation of genetic and demographic factors on extinction risk in wild populations

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Abstract. When populations reach small size, an extinction risk vortex may arise from genetic (inbreeding depression, genetic drift) and ecological (demographic stochasticity, Allee effects, environmental fluctuation) processes. The relative contribution of these processes to extinction in wild populations is unknown, but important for conserving endangered species. In experimental field populations of a harvested kelp (*Postelsia palmaeformis*), in which we independently varied initial genetic diversity (completely inbred, control, outbred) and population size, ecological processes dominated the risk of extinction, whereas the contribution of genetic diversity was slight. Our results match theoretical predictions that demographic processes will generally doom small populations to extinction before genetic effects act strongly, prioritize detailed ecological analysis over descriptions of genetic structure in assessing conservation of at-risk species, and highlight the need for field experiments manipulating both demographics and genetic structure on long-term extinction risk.

Key words: *Allee effect; demographic stochasticity; extinction; genetic diversity; inbreeding;* Postelsia palmaeformis; *sea palm.*

Introduction

Species extinction is occurring at an unprecedented pace because of human activities on the environment (Lawton and May 1995, Worm et al. 2006), and the causes and consequences of extinction are a general societal concern. A fundamental question is determining what factors influence the chances of extinction. Despite the importance of answering this question, we have virtually no detailed empirical information on the mechanisms and dynamics of extinction in nature because extinction events are infrequent and usually involve rare organisms, which are difficult to study. Nevertheless, understanding the processes by which extinction occurs is critical to implementing appropriate conservation strategies, and for understanding patterns and dynamics of local ecological communities.

Small population size is thought to increase extinction risk through several mechanisms, beyond the obvious fact that fewer individuals must die or fail to reproduce for extinction to occur. First, small population size can increase the risk of extinction as a result of demographic stochasticity (Lande 1988, Lande et al. 2003, Jeppsson and Forslund 2012). Because deaths, births, and mate finding are discrete events, populations might decline due to chance events, even if average survival and birth rates would produce positive population growth rates. Second, small population size may lead to Allee effects (Lande 1988, Groom 1998), where positive density dependence causes small populations to decline at ever-accelerating rates. At low abundance, individuals might be unsuccessful in finding mates with which to breed, group defenses against predators might become less effective, fertilization efficiencies might decline (Levitan et al. 1992), or harsh physical conditions might exert stronger effects with fewer neighbors to ameliorate them (by trapping water, moderating temperatures, or disrupting wind or water shear; e.g., Schiel and Choat 1980). Finally, small population size can introduce genetic features that reduce population growth rates. Because small populations have few genetically different individuals, offspring are highly interrelated after several generations, potentially causing inbreeding depression. High offspring relatedness results in higher homozygosity, revealing rare deleterious recessive mutations that lower population growth rates, and removing any heterozygote advantages (Charlesworth and Charlesworth 1987). Additionally, through genetic drift, bene-

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ficial alleles can be lost and deleterious alleles revealed by chance at small population sizes, which also can lower population growth rates (Lande 1994).

The importance of the different mechanisms by which small populations may affect extinction risk has been controversial. Attention has increasingly focused on the effects of genetic factors (Franklin 1980, Barrett and Kohn 1991, Lacy 1997, Frankham 2005, Bouzat 2010, Miller et al. 2012), in part because of well-documented effects of inbreeding in captive populations, including those of conservation interest in zoos (Charlesworth and Charlesworth 1987, Lacy 1997, O'Brien 1994, Griffen and Drake 2008), and because rapid improvements in DNA analysis has facilitated assessment of genetic structure in small populations. Some biologists have questioned this emphasis, and suggested that nongenetic factors such as demographic stochasticity and environmental factors that cause small population size have overriding importance in natural populations (Simberloff 1988, Caughley 1994, Caro and Laurenson 1994). Furthermore, theory indicates that, at the point populations are sufficiently small for inbreeding to become important, demographic and environmental stochasticity are likely to cause extinction regardless of inbreeding depression (Lande 1988, Schaffer and Samson 1985, Menges 1991). Other models linking genetic and demographic processes, however, indicate that these may interact to increase extinction risk of small populations (Mills and Smouse 1994, Tanaka 1997, Jaquiéry et al. 2009). Despite the importance of understanding the relative roles of demography and genetic processes for developing sound conservation decisions, experimental tests of these processes in nature are still sparse. Correlations between population size, heterozygosity, life history features, and local extinction have been observed (Pimm et al. 1988, Saccheri et al. 1998, Sæther et al. 2005, Dournier and Cheptou 2012), and experiments provide evidence that some of the mechanisms associated with small population size affect short-term components of population fitness (Jiménez et al. 1994, Newman and Pilson 1997, Madsen et al. 1999, Nieminen et al. 2001). However, we lack experimental information on the relative importance of different processes on long-term population dynamics and persistence.

Here we report an experiment that independently manipulated genetic composition and population size in free-living populations of a species of conservation concern, the harvested kelp *Postelsia palmaeformis* (the sea palm; see Plate 1), and document the relative role of genetic and demographic factors in determining extinction risk.

Materials and Methods

To test the roles of genetic and demographic factors on extinction risk, we independently manipulated the genetic diversity and population size of free-living populations of the sea palm, which lives along wave-exposed rocky shores of the northeastern Pacific. The sea palm is ideal for this experimental study because it has relatively insular populations (Dayton 1973, Paine 1988, Kusumo et al. 2006, Barner et al. 2011) that sometimes go extinct (Paine 1979, 1988). Furthermore, prior observations of populations on the verge of extinction have frequently detected stunted individuals (R. T. Paine, *personal communication*), leading us to suspect a priori that genetic factors might contribute significantly to extinction risk. The macroscopic sporophyte of the sea palm grows during the relatively calm summer months, drips spores during the late summer onto the rock below when exposed in air at low tide, and dies during the winter when large waves rip it from the rock, along with sessile species that have encroached during the summer (Dayton 1973, Paine 1979). The resulting enemy-free clearings are ideal for survival of the next generation of plants, hence limited spore dispersal (<1 m; Dayton 1973, Paine 1988, Kusumo et al. 2006) is apparently adaptive. Dislodged adults do not die immediately, but can float to more distant areas and deposit spores to start new colonies. Such events are extremely rare, however, because they require a fertile plant to be washed onto a cleared area of rock at the proper tidal height and high wave exposure regime, and then to deposit spores before it is washed away by another wave as little as 10 seconds later. Experimenters can substantially relax these colonization constraints by creating clearings at suitable sites on the shore and enclosing plants in attached wire baskets that keep fertile plants in these clearings for up to a month (Paine 1988). Using these methods, we established experimental, free-living populations of sea palms in previously vacant sites in appropriate habitat (see Plate 1). We varied the genetic composition of these populations by starting populations randomly assigned to a genetic founder treatment of either a single individual (completely inbred), six individuals from the same source population, or six individuals taken from six different regional populations (outbred).

We established experimental plots on Tatoosh Island, Washington, USA (48°24′ N, 124°44′ W), in three general areas that lacked extant sea palm populations, but exhibited conditions associated with successful sea palm populations elsewhere around the island (strong wave exposure; see Paine 1979): the Glacier (west side of the island), the Finger (northwest side of the island), and Simon's Landing (southeast side of the island; see map in Paine 1988). The latter two are known to have had natural populations in the past. The Glacier site was sufficiently large to hold 2–3 replicates at once in its north, central, and south areas. At these sites, we established circular clearings in the middle intertidal zone (dominated by the mussel *Mytilus californianus*) of approximately 0.5 m in diameter using metal scrapers. The plots were then treated with NaOH to eliminate any possibility of pre-colonization by residual spores.

We collected founder plants from six different source populations in the Cape Flattery region of the Washington coast (described in Kusumo et al. 2006). These included three sites from the main islets of Tatoosh (Fingernail, Northwest Point, Rainbow Rock), isolated offshore rocks to the west of Tatoosh (West Rocks), and two sites from the mainland (East Cape Flattery and Slant Rock). Plants were collected and introduced to experimental plots in late July, when they had begun to release spores. The upper one-third of the plants (fronds, which contain the reproductive sori, plus upper stipe) were placed in 10×15 cm packages made of 2.5-cm mesh metal chicken wire with a piece of 0.5-cm plastic mesh lodged in the upper roof to reduce the chances of plants slipping out the top. Each package was strapped to five stainless-steel eye screws installed on the rock using heavy-duty cable ties (see Plate 1). These packages remained for approximately one month at each site, which allowed an extended period for plants to drip their spores on to the underlying rock. Eventually the packages rusted away, leaving a bare surface for plant establishment.

Plants were placed in packages in one of three founder treatments: a single plant (inbred), six plants from the same source population ("control"), or six plants from each of the six regional source populations ("outbred"). These founder numbers fell well within the typical clump size distribution of detached floating adults washed up on a regularly monitored stretch of beach on the island (Appendix A: Fig. A1). In May of the following year, established populations were thinned to either large (50 plants) or small (20 plants) size, and their population dynamics then followed at monthly intervals from April to September in each year for up to 12 years, or until extinction occurred (see Plate 1). In some cases, initial populations did not become established at sufficient sizes to use in the experiment. In these cases, the establishment process was repeated for missing treatments until a complete replicate was obtained. We established a total of eight replicates of each of the six treatments. Each replicate was located at the same site at the same general time period and used the same source population in all but the outbred treatments. Although our experiment did not allow us to identify general differences among source populations arising from genetic differentiation (Kusumo et al. 2006) from variation in the environmental conditions experienced by each replicate set of plots, our genetic analyses indicated that all source populations were equally represented (Barner et al. 2011). Because of the large area required to carry out the experiments, plots were reused to gain additional replication once sufficient plots became available following extinctions to allow installation of a complete set of treatments at a site at the same time. The earliest replicates were installed in 1999 (2000 experimental starting date), and the latest replicate in 2006. Treatments were assigned randomly to prevent plot effects from confounding treatment effects. Staggering replicates may have introduced temporal variation into the experiment, which could potentially reduce statistical power, but the blocked design allowed us to account statistically for these effects. The staggered design could also be viewed as beneficial in that the results are general across a wider set of temporally varying environmental conditions.

We quantified genetic diversity among the treatments in the first generation using microsatellite markers using methods reported in prior papers (Kusumo et al. 2006, Barner et al. 2011). Analyses used nine microsatellite markers with 3 to 13 alleles per locus. We collected ~1 g of tissue from the basal meristem of each founding individual. Polymerase chain reaction (PCR) analysis was carried out at the DNA Core Sequencing Facility at the University of Chicago (Illinois, USA) and at the Field Museum Pritzker Laboratory for Molecular Systematics and Evolution (Chicago, Illinois, USA). Genotypes were assigned to individuals using hand-scored GeneMapper chromatograms. Some populations went extinct prior to tissue collection for genetic analysis, so could not be included in the analysis. The analysis showed that the experimental treatment generated clear differences in genetic diversity as assessed by microsatellites (Appendix A: Fig. A1).

We statistically tested for effects of population size, genetic composition, and their interaction using survival analysis with a Cox proportional-hazards model on each experimental treatment, blocked by experimental replicate. We also applied a Cox proportional-hazards model with time-dependent genetic and population size effects, but the time dependence term was not significant ($P = 0.65$), and did not change the results; therefore, we focused on time-independent models. We also examined mean time to extinction using a blocked two-way factorial ANOVA on log-transformed data, assuming that populations that have yet to go extinct will be extinct at our next census date (April 2013). Because census periods contained gaps over the winter, when the populations are in the microscopic gametophyte state, and because not all populations are extinct, the survival analysis is more robust, but we report the ANOVA results for completeness. We further examined the functional relationship of population size on extinction risk by regressing starting (April) population size in one year against the extinction status of the population in the following year using logistic regression. We further explored the effects of founder genetics by testing whether or not a population established after initial deployment of experimental plants, using a χ^2 test of independence. This comparison may not necessarily reflect genetic effects, however, because approximately six times more reproductive tissue was present in founders in control and outbred treatments compared to inbred treatments. We also calculated per capita reproduction in founder plants prior to thinning to final population sizes, and tested for genetic effects using a one-way ANOVA on $\ln(x + 0.1)$-transformed data.

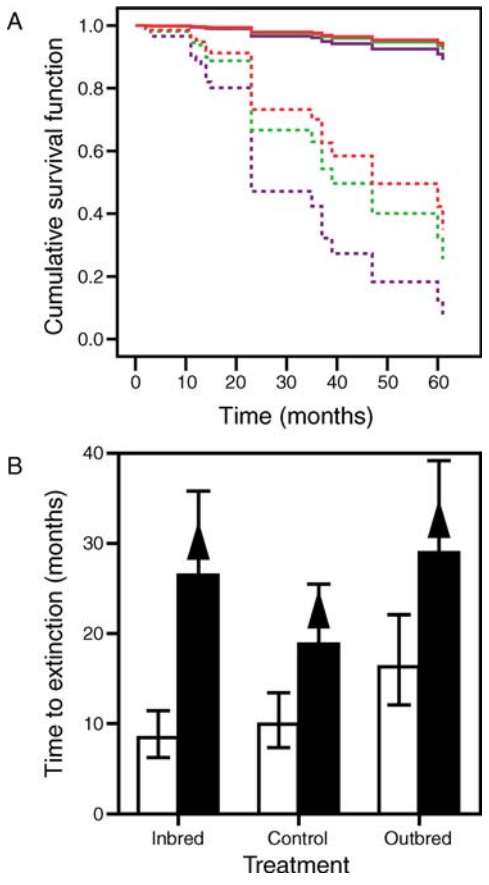

FIG. 1. Extinction risk of a harvested kelp (sea palm, *Postelsia palmaeformis*) as a function of initial experimental treatments ($n = 8$) crossing founder genetic diversity (purple indicates inbred populations; green, control; and red, outbred populations) with starting population size (dashed lines show low abundance, and solid lines show high abundance). (A) Cumulative population failure (extinction) rate through time estimated from a Cox proportional-hazards model of survival. Population size affected extinction risk ($P < 0.001$); genetic treatment did not ($P = 0.38$). (B) Observed time to extinction, showing back-transformed means ± SE of log-transformed data. Black bars show high abundance, and white bars show low abundance. Arrowheads at mean values indicate treatments where some populations had yet to go extinct, making conservative estimates. All extant populations started at large size. Initial population size affected time to extinction (ANOVA, $P = 0.004$); genetic treatment did not ($P = 0.23$).

We assessed the contribution of different demographic processes to extinction risk through analysis of seasonal survival and fecundity rates of each population in each year. We estimated average sporophyte survival rate during the summer growing season (s) by comparing censuses of population size in the late summer (N_{ls}; August–September) to population size in spring (N_{sp}; May) of the same year (t): $s = N_{ls,t}/N_{sp,t}$. We estimated average per capita recruitment (λ) by comparing population size in the spring to the population size at the end of summer in the prior year: $\lambda = N_{sp,t+1}/N_{ls,t}$. Note that recruitment includes individual fecundity, and subsequent winter survival and reproduction of offspring (spores, gametophytes, and young sporophytes).

We ascribe demographic stochasticity to variability in population growth arising strictly from sampling effects (Lande et al. 2003). Therefore, we averaged the seasonal survival of each population in each year and took this (0.408 ± 0.337 [mean ± SD], $n = 122$) to characterize the expected survival rate of any individual in the absence of environmental variability in the broadest sense (including both abundance-dependent and abundance-independent variation in demographic rates). In the absence of environmental variability, individual survival outcomes should follow a binomial distribution, as there are two possible outcomes for each individual: alive or dead. Similarly, we calculated expected recruitment (λ) by averaging per capita recruitment across each extant late-summer population in each year (2.728 ± 6.225, $n = 95$). In the absence of broad-sense environmental variability, we assume per capita recruitment is Poisson distributed with a mean of λ. Under this distribution, the probability that an individual leaves no recruits is

$$\lambda_0 = \lambda^0 \times \exp(-\lambda)/0! = \exp(-\lambda).$$

The probability of an individual leaving offspring in the following year is the probability that it survives the summer growing season (s) times the probability that it leaves one or more recruits ($1 - \lambda_0$), given that it survived, so the probability that an individual does not contribute to the population in the following year is ($1 - s \times (1 - \lambda_0)$). The probability of extinction under demographic stochasticity is the product of the probability that an individual leaves no offspring across all individuals, or

$$p(\text{extinction})_{ds} = [1 - s \times (1 - \lambda_0)]^N. \quad (1)$$

Contributions of Allee effects were investigated by testing for relationships between starting abundance in a particular year ($N_{sp,t}$) and average survival rate (s), or late-summer population size ($N_{ls,t}$) and spring recruitment ($N_{sp,t+1}$) in a population using least-squares nonlinear regression and comparison of nested models. Incorporating abundance-dependent mean vital rates yields an extinction risk estimate that includes both demographic stochasticity and Allee effects:

$$p(\text{extinction})_{ds+ae} = (1 - S(N) \times [1 - \lambda_0(N)])^N. \quad (2)$$

Hence, the difference between Eqs. 2 and 1 estimates the net contribution of Allee effects. Without genetic effects, the difference between the extinction risk function estimated from the data and Eq. 2 represents effects on extinction risk of annual variation in vital rates generated by environmental stochasticity.

RESULTS AND DISCUSSION

Extinction risk was strongly associated with population size in our experiments, as assessed by both survival analysis ($P < 0.001$; Fig. 1A; Appendix B: Table B1) and

analysis of time to extinction ($P = 0.003$; Fig. 1B). On average, time to extinction was about twice as long in high-population size treatments as in low-population size treatments. The effects of abundance on annual extinction risk were also apparent in subsequent years (logistic regression, $P < 0.001$), with a rapid decrease in extinction risk between spring populations of 10 to 100 individuals (Fig. 2A). Including initial population size in the regression analysis provided no additional explanatory ability ($P = 0.34$).

Genetic effects, and their interaction with abundance, were not statistically detectable either in the survival analysis ($P = 0.53$ and $P = 0.82$, respectively; Fig. 1A; Appendix B: Table B2) or in the analysis of time to extinction ($P = 0.28$ and $P = 0.59$, respectively; Fig. 1B). The Cox proportional-hazards model (Fig. 1A) hinted that survival probability might be lowest for inbred populations and highest for outbred populations. Genetic treatment did not affect the probability of establishing a population (chi-square test, $P = 0.15$), although the probability of establishment with genetic diversity increased weakly (Appendix B: Fig. B1). Per capita recruitment success of founders was also unrelated to genetic treatment (ANOVA, $P > 0.95$; Appendix B: Fig. B1).

Our data showed evidence of positive density dependence (Allee effects). Average survivorship in a plot increased with population size in a decelerating manner ($P = 0.002$, $n = 122$; Appendix C: Fig. C1, Table C1), which could be described by the logistic function:

$$s(N) = \exp[-1.369 + 0.268\ln(N_{\mathrm{sp},t})]/$$
$$(1 + \exp[-1.369 + 0.268\ln(N_{\mathrm{sp},t})]).$$

Including genetic treatment provided no significant improvement in fit ($P = 0.39$). Spring recruitment (Appendix C: Fig. C2) showed evidence both of negative density dependence at high abundance ($P < 0.0001$) and Allee effects at low abundance ($P < 0.0001$). Although models with parameters specific to genetic treatment provided a better fit to the recruitment data ($P = 0.01$; Appendix C: Table C1), these effects were not strong enough to translate into differences in annual extinction risk among genetic treatments ($P = 0.50$; Fig. 2A).

Detailed analysis and modeling of demographics across years revealed shifts in the relative contributions of different ecological processes at small population size (Fig. 2B). Demographic stochasticity had proportionally large contributions at extremely small (<5 individuals) populations sizes, largely because *Postelsia* exhibited relatively high annual per capita reproductive potential. Allee effects became more important at modest population sizes (5–10 individuals), perhaps through the mutual amelioration of desiccation during low tides. Extinction risk at larger population sizes was primarily determined by environmental variability driving annual variation in demographic rates. Genetic treatment had minimal effects on these contributions (Fig. 2B).

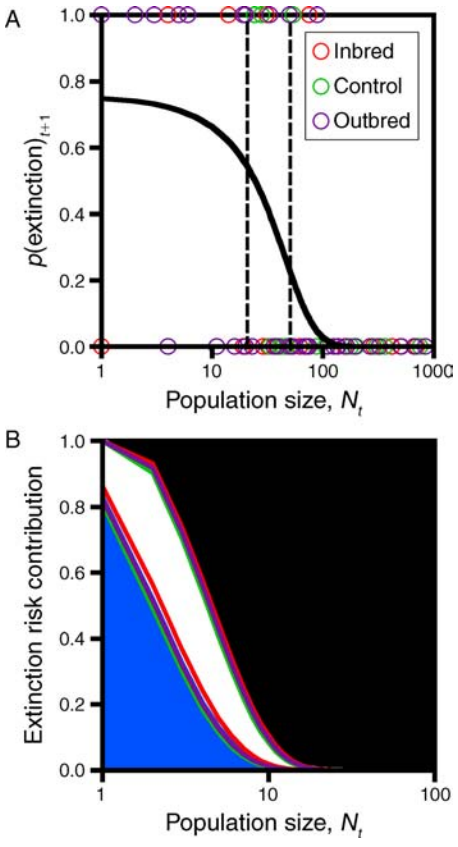

FIG. 2. Patterns of extinction risk in experimental, free-living sea palm populations as a function of population size in the prior year. Note that the *x*-axes are on a log-scale. (A) Probability of overall extinction, showing a sharp drop between 10 and 100 individuals. The solid black line shows the function of best fit from logistic regression. The dashed lines show the initial population size treatments in the experiment. (B) Estimated relative contributions of three different ecological processes to extinction risk over the range of population size exhibiting appreciable extinction risk. Demographic stochasticity is shown with blue, Allee effect with white, and environmental stochasticity with black. Colored lines show different genetic treatments; colors are as in panel (A).

Our results demonstrate that extinction risk in sea palms expands rapidly at small population sizes. Interestingly, this elevated risk occurs in the vicinity of 50–100 suggested by prior "rules of thumb," indicating that these indeed may be useful where detailed demographic information is not available. Taken together, our results also corroborate theoretical predictions (Schaffer and Samson 1985, Lande 1988, Menges 1991) that demographic processes predominate in shaping elevated extinction risk at small population size.

The minor effects of genetic treatment on extinction risk were unexpected for several reasons. First, prior observations of less vigorous individuals in populations nearing extinction suggested genetic effects at small populations. However, adverse environmental conditions, exacerbated by Allee effects, can also cause poor

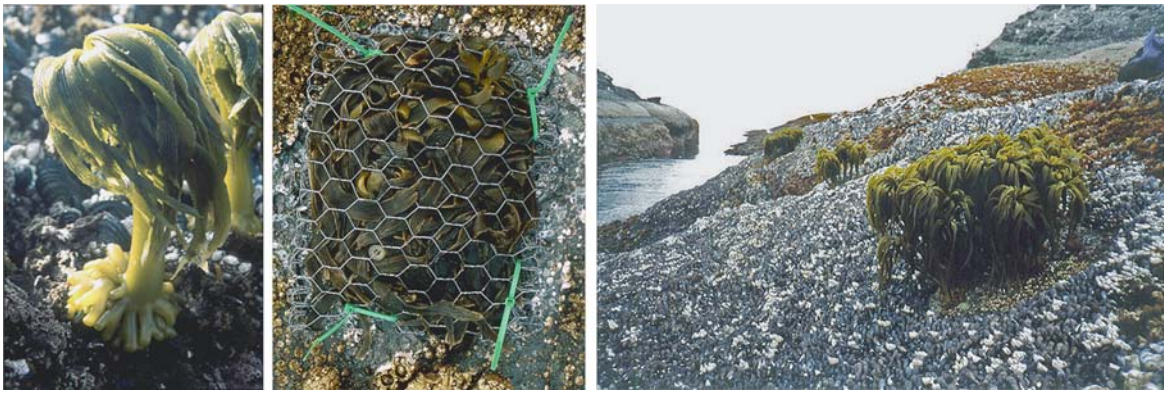

PLATE 1. Photos of experimental system: (left) close-up of a sea palm; (center) method for establishing new sea palm populations; and (right) established sea palm populations of large and small size showing the clear delineation of experimental populations. Photo credits: (left) J. T. Wootton; (center and right) C. A. Pfister.

phenotypic performance. Second, high inbreeding coefficients are assumed to imply elevated extinction risk arising from genetic processes (Keller and Waller 2002, Spielman et al. 2004), and sea palms exhibit significant F_{is} indices (the interindividual fixation index within subpopulations; Kusumo et al. 2006), which can be interpreted as indicating inbreeding. The detrimental paradigm of inbreeding effects (Bouzat 2010) may not consistently apply in natural populations, however, for both population genetic reasons (e.g., purging of deleterious alleles; Tallmon et al. 2004, Bouzat 2010) and ecological reasons (e.g., environmental variability, integration of multiple life history components). Although we found some evidence of genetic treatment on recruitment success (Appendix C: Fig. C2), this effect does not result in altered extinction risk because post-recruitment survival exhibits opposite tendencies (Appendix C: Fig. C1).

Although our results are consistent with general theory (Lande 1988), is there generality of our findings beyond this particular species? Several features of sea palm life history are shared across many species. First, the metapopulation structure of sea palms, capable of generating periodic bottlenecks, is likely present in many species. Second, a haplo-diploid life cycle combined with selfing, which has little short-term fitness effect in this species (Barner et al. 2011), might increase the efficiency of revealing deleterious alleles to natural selection. Efficiently revealing deleterious alleles could result in higher extinction risk, but purging those alleles might reduce extinction risk. Also, purging cannot eliminate inbreeding effects from heterozygote advantage. Furthermore, recent modeling of extinction risk under a variety of demographic and genetic scenarios found no strong relationship between time to extinction and selfing (Jaquiéry et al. 2009). Thus, sea palm life cycle attributes could result in either relative insensitivity or sensitivity to extinction risk from genetic factors, depending on the underlying genetic processes. Finally, self-fertilization should minimize the relative risk of extinction from demographic effects by increasing reproductive assurance at low population size (Barner et al. 2011). Therefore, the attributes of sea palms that facilitated our experiments do not necessarily predispose this species to strong demographic effects relative to genetic effects. Further experiments independently manipulating demographic and genetic characteristics in free-living populations will reveal the features of species that change the balance of demographic vs. genetic factors on the extinction process.

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Supplemental Material

Appendix A

Founder treatment genetic diversity and comparison to natural dispersing clump sizes (*Ecological Archives* E094-196-A1).

Appendix B

Analysis details of treatment effects on population establishment, survival, and time to extinction (*Ecological Archives* E094-196-A2).

Appendix C

Relationship of annual survival and recruitment to population size and genetic treatment (*Ecological Archives* E094-196-A3).