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A review of extinction in experimental populations

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Summary

1. Population extinction is a fundamental ecological process. Recent experimental work has begun to test the large body of theory that predicts how demographic, genetic and environmental factors influence extinction risk. We review empirical studies of extinction conducted under controlled laboratory conditions. Our synthesis highlights four findings.

First, extinction theory largely considers individual, isolated populations. However, species interactions frequently altered or even reversed the influence of environmental factors on population extinction as compared to single-species conditions, highlighting the need to integrate community ecology into population theory.

2. While most single-species studies qualitatively agree with theoretical predictions, studies are needed that quantitatively compare observed and predicted extinction rates. A quantitative understanding of extinction processes is needed to further advance theory and to predict population extinction resulting from human activities.

3. Many stresses leading to population extinction can be assuaged by migration between subpopulations. However, too much migration increases synchrony between subpopulations and thus increases extinction risk. Research is needed to determine how to strike a balance that maximizes the benefit of migration.

4. Results from laboratory experiments often conflict with field studies. Understanding these inconsistencies is crucial for extending extinction theory to natural populations.

Key-words: demographic stochasticity, environmental stochasticity, extinction, genetic bottleneck, population dynamics

Introduction

Extinction is a fundamental ecological process and the ultimate fate of all populations. A reliable, quantitative theory of extinction is increasingly important for anticipating the effects of the growing global imprint of humanity (Kerr & Currie 1995). A thorough understanding of processes that influence extinction risk and associated principles that govern population dynamics may inform multiple applied ecological problems, including the conservation of threatened and endangered species, reserve design and development, habitat restoration, management of harvested species, and biological control. Improved understanding of extinction also benefits our basic understanding of metapopulation and metacommunity dynamics, large-scale biogeographical patterns, and fine-scale distribution of species over small spatial scales.

Plausible explanations of population extinction abound (e.g. Lawton 1997; Earn, Levin & Rohani 2000; Lande, Engen & Sæther 2003; Frankham 2005). However, confirming links between observed extinctions and theory is complicated by the complexity of natural systems, where populations exist within a network of interacting species, are distributed patchily over a heterogeneous space, and are subject to a range of environmental conditions. In field studies, isolating populations from such confounding factors is often impossible or logistically prohibitive (but see Berggren 2001; Drayton & Primack 1999; Grevstad 1999 for examples of successful field studies). Laboratory tests of extinction theory present a feasible alternative by providing a simplified, tractable system where exogenous factors can be eliminated, and targeted mechanisms can be controlled, replicated and manipulated (Lawton 1995; Daehler & Strong 1996; Benton *et al*. 2007). These simplified systems trade a degree of realism (Carpenter 1996) for the ability to identify building blocks of population theory that can subsequently be applied to more natural conditions. Indeed, we argue that microcosm experiments are a crucial proving ground for ecological theory: what cannot be demonstrated in the lab can hardly be expected to hold in nature.

Laboratory systems are particularly useful for experimental studies of population extinction given the undesirability of eliciting the extinction of natural populations and the unparalleled capability that laboratory systems provide for

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Table 1. Theoretical factors influencing extinction risk, and studies that have tested these theories in controlled laboratory settings

Prediction	Supported	Unsupported
Demographic factors are predicted to be important primarily at small population sizes. They are also predicted to be important in both single populations and metapopulations, and may influence (meta)population extinction in the following ways.		
1. Small populations are more likely to go extinct because of demographic stochasticity (Lande *et al*. 2003).		(Burkey 1997; Belovsky *et al*. 1999)
2. The magnitude of population density fluctuations resulting from demographic stochasticity should scale inversely with the square root of population numbers (May 1973).	(Desharnais *et al*. 2006)	
3. Moderate levels of migration between populations should decrease extinction by providing a 'rescue effect' (Allen *et al*. 1993).	(Forney & Gilpin 1989; Holyoak & Lawler 1996a*; Amezcua & Holyoak 2000*; Drake *et al*. 2005; Dey & Joshi 2006)	
4. High levels of migration between populations should increase extinction risk by synchronizing population fluctuations, increasing the chance that coupled populations will decline simultaneously (Earn *et al*. 2000).	(Burkey 1997*; Holyoak & Lawler 1996b*; Dey & Joshi 2006; Molofsky & Ferdy 2005)	
Decreased genetic variation is hypothesized to increase the probability of population extinction. This may occur through various causes.		
5. Founder effects and/or population bottlenecks should increase extinction risk due to decreased genetic variance (decreased effective population size, N_e, from inbreeding) (Frankham 2005).	(Radwan 2003; Reed *et al*. 2003)	
6. Reduced genetic variation may increase extinction risk by limiting the ability to adapt to stressful environments.	(Frankham *et al*. 1999; Bijlsma *et al*. 2000; Reed & Bryant 2000; Reed *et al*. 2002)	
7. Migration should reduce extinction risk by decreasing the genetic consequences of small population size (Mills & Allendorf 1996; Spieth 1974; Vucetich & Waite 2000).	(Waite *et al*. 2005)	
Environmental factors are predicted to influence population extinction via environmental quality and environmental variation, as follows.		
8. Habitats with greater food supply should support larger populations and thus have lower extinction risk (Bayliss & Choquenot 2003).	(Philippi *et al*. 1987; Belovsky *et al*. 1999; Bancroft & Turchin 2003)	(Holyoak 2000*; Luckinbill 1973*, 1974*)
9. Larger habitats should support larger populations and therefore lead to lower extinction risk (Klok & De Roos 1998; Hanski 1999).	(Burkey 1997*; Forney & Gilpin 1989; Warren 1996; *)	(Luckinbill 1974*)
10. Increasing food variation should lead to greater probability of extinction and should decrease the time to extinction (Tuljapurkar & Orzack 1980; Davis *et al*. 2003; Lande *et al*. 2003).	(Belovsky *et al*. 1999; Benton *et al*. 2001, 2002; Drake & Lodge 2004)	(Grover 1988*; 1991*)
11. Environmental stressor such as chemicals may decrease time to extinction.	(Shirley & Sibly 2001; Beketov & Liess 2006)	
12. Autocorrelation in environmental variability (red noise) should increase population extinction (Lawton 1997).**	(Pike *et al*. 2004)	
13. Spatial variation in environmental quality should decouple dynamics of subpopulations and thereby decrease the risk of population extinction (Allen *et al*. 1993; Earn *et al*. 2000).	(Gonzalez & Holt 2002)	(Godoy & Costa 2005)
In addition, factors within these broad categories are predicted to interact. For example, inbreeding is predicted to increase the influence of environmental stress.		

*Experiments conducted in community context.

**While there is some support for this simple hypothesis, the relationship between environmental autocorrelation and population dynamics is likely complex (Heino *et al*. 2000), depending on the time scale of autocorrelation (Orland & Lawler 2004) and its synchrony with population generation time (Luckinbill & Fenton 1978).

replication of the extinction process. As a result, a number of studies have examined extinction theory using model laboratory systems (Table 1). These studies have tested a wide variety of causal mechanisms using a variety of model species. They have yielded new insights into several factors that influence extinction risk, including the stabilizing influence of spatial and temporal heterogeneity, the demographic and genetic benefits of migration, and the influence of interacting species on population processes. A synthetic review of these studies and the insights they provide will facilitate continued progress in the study of population extinction and is therefore timely.

Here we review laboratory tests of extinction theory, summarize their findings, and make recommendations for next steps. Our review includes papers found by searching all databases within the Web of Science for all available dates

(1956–June 2007) using the following searches: (1) experimental AND population AND extinction; (2) extinction AND *Drosophila*; (3) extinction AND *Tribolium*; (4) extinction AND house fly; (5) extinction AND *Daphnia*; (6) extinction AND *Artemia*; (7) extinction AND Collembolan; (8) extinction AND beetle; (9) extinction AND protozoa; (10) extinction AND laboratory experiment; (11) extinction AND greenhouse experiment; (12) competition AND extinction AND experiment; (13) predation AND extinction AND experiment. These searches yielded a total of 1016 papers. We also examined pertinent studies published before 1956, and therefore not listed in the Web of Science, that were cited in these papers. In total, 53 studies fit our search criteria: laboratory experiments in which either (a) population extinction occurred, or (b) population extinction did not occur (nor were they necessarily designed to study extinction), but factors postulated to directly influence extinction risk were experimentally examined. These studies are listed in Table 2.

Empirical tests of extinction theory

Factors leading to population extinction can be separated into three broad categories.

Demographic stochasticity: variation in population growth resulting from variation in individual growth, reproduction, or lifespan.

Genetic effects: inbreeding depression and the loss of genetic variation.

Environmental stress: effects that result from physiological costly, toxic, or variable environments.

Multiple theoretical predictions exist for each of these factors (Table 1). While most extinction models are for single species, real populations are always embedded within communities comprised of interacting species. Drawing on Sih *et al.* (1985) and Møller (2005) who demonstrate the importance of species interactions in structuring populations and communities, we submit that in many cases, interspecific interactions will alter or reverse the influence of demographic, genetic and environmental factors, compared with single-species theory. In our review we therefore draw attention to these contrasts.

DEMOGRAPHIC EFFECTS

The standard theory holds that, because variation among individuals has the greatest effect at small population size, demographic effects become stronger as population size decreases (Lande *et al.* 2003). Primarily in the context of biological control and conservation (Drayton & Primack 1999), demographic effects have been studied with respect to four overlapping categories: population variability, initial population size, migration, and population synchrony.

Population variability

Demographic stochasticity can cause fluctuations in population density, the magnitude of which are predicted to scale inversely with the square root of population numbers (May 1973). Thus, the larger the population, the less susceptible it is to fluctuations that result in extinction. Consistent with this prediction, Desharnais *et al.* (2006) examined *Tribolium castaneum* in small and large habitats and found that larger habitats supported larger populations that were more similar in size to deterministic model predictions than populations in small habitats, suggesting a decrease in the amount of demographic stochasticity (i.e. decreased variability) in larger populations.

If large populations are less susceptible to variability due to demographic stochasticity, these populations should in turn be less prone to extinction. Three studies conducted under controlled conditions have found population persistence to scale inversely with population variability. Using bruchid beetles (*Callosobruchus maculates*), Vucetich *et al.* (2000) simulated four levels of environmental variability by removing beans on which beetles had oviposited, and found that populations with greater variability had a greater risk of extinction. Similarly, Forney & Gilpin (1989) found that variability in population size and extinction risk were both lower in *Drosophila pseudoobscura* because of migration between subhabitats than for *D. hydei* that did not migrate. Finally, competition between *Daphnia magna* and *D. longispina* increased population variability, resulting in population extinction (Bengtsson & Milbrink 1995).

Initial population size

Two laboratory studies have tested the prediction that small populations are at greater risk of extinction from demographic effects and should go extinct more rapidly than large populations. Specifically, these studies examined the influence of initial population size on time to extinction. Using *Artemia franciscana*, Belovsky *et al.* (1999) did not detect an effect of initial population size on time to extinction, even when populations differed in their initial sizes by a factor of 10, from 2 to 20. The authors reasoned that, due to a lack of other limiting factors, small populations far below carrying capacity were able to increase quickly, thus avoiding extinction from demographic stochasticity.

Similarly, Burkey (1997) did not detect a relationship between initial population size and time to extinction of a top predator (large ciliates) that fed on smaller bacteriovore ciliates. In trials with three different intermediate predator ciliates, no significant correlation between initial density of the top consumer (*Euplotes aediculatus*, ranging from 4 to 91 individuals) and time to extinction of this consumer was detected. A marginally non-significant correlation between time to extinction and initial density of *Euplotes* ($r = 0{\cdot}339$, $P = 0{\cdot}07$) might be due to information lost to termination of the experiment before extinction (data censoring).

Populations may be temporarily small following a population bottleneck (as in these experiments), or they may be kept small through environmental or other factors. The ability of populations in these experiments to rapidly increase when small may have limited the capacity of experiments to detect

Table 2. Study systems used to examine hypothesized factors contributing to population extinction

Reference	Study system	Experimental factor (no. levels)	No. reps[a]	Impact
Amezcua & Holyoak 2000	Predator: *Actinosphaerium nucleofilum* Prey: *Tetrahymena pyriformis* (protozoans)	Prey migration (2)	3–6	↓T,↑S
Bancroft & Turchin 2003	*Oryzaephilus surinamensis* (grain beetle)	Habitat fragmentation (3)		↓D
		Decrease food amount (2)	????	↓D,↓P
Beketov & Liess 2006	*Artemia* sp. (brine shrimp)	Predation (2)	32	↓D
		Toxic chemical (4)		↓D
		Interaction effect		↓P
Bengtsson & Milbrink 1995	Competitors: *Daphnia magna* and *D. longispina*	Interspecific competition (2)	122	↑V,↓D,↓P
		Size selective predation (3)	4–20	–
Belovsky *et al*. 1999	*Artemia franciscana* (brine shrimp)	Initial population size (7)		–
		Increasing food amount (12)		↑P
		Variation in food supply (3)		↓P
Benton *et al*. 2002	*Sancassania berlesei* (soil mites)	Variation in food supply (4)	5	↑V,↓D
Benton *et al*. 2001	*Sancassania berlesei* (soil mites)	Synchrony in food variation (6)	5	↑S
Bijlsma *et al*. 2000	*Drosophila melanogaster* (fruit flies)	Inbreeding (11)	10–30	↓P
		Environmental stress (3)		↓P
Bull *et al*. 2007	Parasitoid: *Anisopteromalus calandrae* (wasp)	Variation in food supply (2)	????	↓T
	Competitors: *Callosobruchus maculatus* and *C. chinesnsis* (beetles)	Apparent competition (2)		↓T
Burkey 1997	Three trophic levels using several species of protozoans	Decrease patch size (4)	5–288	↓T
		Habitat fragmentation (3)		↑T
		Migration (2)		–
		Initial population size (C[b])		–
Desharnais *et al*. 2006	*Tribolium castaneum* (flour beetle)	Increase habitat size (2)	3	↓M
Dey & Joshi 2006	*Drosophila melanogaster* (fruit flies)	Migration rate (3)	????	↑S
Drake & Lodge 2004	*Daphnia magna* (freshwater zooplankton)	Variation in food supply (3)	92–96	↓P
Drake *et al*. 2005	*Daphnia magna* (freshwater zooplankton)	Propogule pressure (11)	7–14	↑P
Drayton & Primack 1999	*alliaria petiolata* (garlic mustard plant)	Decrease population size (2)	56–61	↓P
Ebert, Lipsitch & Mangin 2000	Host: *Daphnia magna* (freshwater zooplankton) Parasites: several species of bacteria, microsporidians, and fungus	Parasite presence (6)	5	↓P
Ellner *et al*. 2001	Predator: *Phytoseiulus persimulis*	Metapopulation (2)	????	–
	Prey: *Tetranychus urticae* (mites)	Habitat structure (2)		↑P
Frankham 1995	*Drosophila malanogaster*, *D. virilis* (fruit flies), *Mus* musculus (mouse)[c]	Inbreeding (5–8)	–[c]	↓P
Frankham *et al*. 1999	*Drosophila melanogaster* (fruit flies)	Inbreeding combined with environmental stress (2)	5	↓P
Fryxell *et al*. 2006	*Tetrahymena thermophila* (protozoan)	Harvesting strategy	????	↓P
Fryxell *et al*. 2005	*Tetrahymena thermophila* (protozoan)	Presence of harvesting reserve	8	↑P
Godoy & Costa 2005	*Tribolium castaneum* (flour beetles)	Difference between source and sink habitats (4)	12	–
Gonzalez & Holt 2002	*Paramecium tetraurelia* (protozoan)	Source-sink habitats with autocorrelated temperature (6)	3	↑D
Goodnight & Craig 1996	Competitors: *Tribolium castaneum* and *Tribolium confusum* (flour beetles)	Natural selection (3)	10	–
Grover 1991	*Scenedesmus quadricauda* and *Chlorella* sp. (microalgae)	Variation in nutrient additions	2	↑T
Grover 1988	*Synedra* sp. and *Fragilaria crotonensis* (microalgae)	Variation in nutrient additions	2	↑P
Holyoak 2000	Predator: *Didinium nasutum*	Increase prey food level (3)	9	↑D[b],↓P
	Prey: *Colpidium striatum* (protozoan)	Habitat fragmentation w/migration (2)		↑P
Holyoak & Lawler 1996a	Predator: *Didinium nasutum* Prey: *Colpidium striatum* (protozoans)	Metapopulations (2)	3	↑P
Holyoak & Lawler 1996b	Predator: *Didinium nasutum* Prey: *Colpidium striatum* (protozoans)	Migration rate (3)	3	↓↑P[d]
Ives *et al*. 2004	*Aureobasidium pullulans* (fungus)	Migration (10)	1	↑D,↑P
Leslie *et al*. 1968	Competitors: *Tribolium castaneum* and *Tribolium confusum* (flour beetles)	Competition	40	↓P
Long *et al*. 2007	Competitors: *Colpidium striatum* and *Tetrahymena thermophila* (protozoans)	Immigration (3)	3	↑P,↑D
		Variation in temperature (6)		↓P,↓D

Table 2. *Continued*

Reference	Study system	Experimental factor (no. levels)	No. reps[a]	Impact
Luckinbill 1974	Predator: *Didinium nasutum* (protozoan) Prey: *Paramecium aurelia* (protozoan)	Decrease patch size (2) Increase prey food level (2)	1–10	↓T ↓T
Luckinbill 1973	Predator: *Didinium nasutum* (protozoan) Prey: *Paramecium aurelia* (protozoan)	Increase prey food level (2) Presence of movement retardant (2)	????	↓T ↑T
Luckinbill & Fenton 1978	*Colpidium campylum* and *Paramecium primaurelia* (protozoans)	Increasing frequency of environmental variation (3)	4	↓P
Molofsky & Ferdy 2005	*Cardamine pensylvanica* (annual plant)	Migration rate (4)	4	↓↑T[e]
Mora *et al*. 2007	*Brachionus plicatilis* (rotifer)	Environmental warming Overexploitation Habitat fragmentation	????	↓D ↓D ↓D
Morin 1999	*Colpidium striatum* and *Blepharisma americanum* (protozoans)	Prey availability (2) Intraguild predation (2)	4	↓P
Orland 2003	*Colpidium striatum* (protozoan)	Density perturbation (7) Food renewal frequency (2)	3	↓↑V[f] ↓↑V[f]
Orland & Lawler 2004	*Colpidium striatum* (protozoan)	Increasing frequency of resource fluctuation (6)	3	↓V,↑D
Park 1954	Competitors: *Tribolium castaneum* and *Tribolium confusum* (flour beetles)	Temperature, humidity	28	–[g]
Petchey 2000	*Colpidium* or *Paramecium (* protozoans)	Temperature autocorrelation (2)	10	↑V
Philippi *et al*. 1987	*Drosophila* spp. (fruit flies)	Reduced food abundance (2)	5	↓P
Pike *et al*. 2004	*Folsomia candida* (springtails)	Autocorrelated culling (6)	10	↓T
Radwan 2003	*Rhizoglyphus robini* (bulb mite)	Inbreeding (2)	22–88	↓P
Reed & Bryant 2000	*Musca domestica* (house flies)	Decrease population size (4)	3–6	↓P
Reed *et al*. 2003	*Drsophila melanogaster* (fruit flies)	Inbreeding rate (3)	80–160	↓P
Reed *et al*. 2002	*Drosophila melanogaster* (fruit flies)	Inbreeding rate (8) Environmental stress (4)	128	↓P ↓P
Shirley & Sibly 2001	*Drosophila melanogaster* (fruitflies)	Migration (2) Evolution caused by pollution (2)	5 5	↑P ↓P
Vucetich *et al*. 2000	*Callosobruchus maculates* (beetle)	Increase food level (10) Increase population mortality (4)	12	↑T ↓T
Wade 1980	Competitors: *Tribolium confusum* and *Tribolium castaneum* (flour beetles)	Migration rate (3) Competition (2)	18	↑V ↓↑T[h]
Waite *et al*. 2005	*Callosobruchus maculates* (bean beetles)	Migration (4)	20	↑P
Warren 1996	Several species of protozoans	Habitat destruction (3) Migration (2)	4	↓P ↑P

[a]per treatment, [b]refers to top predator, [c]data obtained from previously published studies, [d]extinction was lowest at intermediate migration rates, [e]extinction times were longest at intermediate levels of migration,[f] was bimodal effect, [g]competitive dominant depended on environmental conditions, [h]depends on the growth rate of the species involved the competitive relationship. D = density, T = extinction time, M = demographic stochasticity, *P* = persistence (i.e. the qualitative absence of extinction), S = population synchrony, V = population variability, '–' indicates no observed effect.

the effect of demographic stochasticity on extinction risk. Extinction risk from demographic stochasticity may be more apparent in species that are less likely to increase rapidly following population bottlenecks (*K*-selected species) or that are constrained to low population sizes for long periods of time.

Migration

Countering the influence of small population size is movement into and/or among populations. Ives *et al*. (2004) have shown that when negative density dependence is weak, migration can increase population size if immigration rate is positive when populations are growing and negative when they are declining, even when migration leads to no net additions or removals from a population. This is because migration acts to average population densities between patches. This averaging reduces variation in population size through time and therefore increases the population growth rate (which averages geometrically because population growth rates are serially multiplicative). Experiments in which mortality and migration rates between populations in paired habitats were manually imposed showed that average population densities of the fungus *Aureobasidium pullulans* increased with higher migration rates, even without addition of fungal cells from outside the paired habitats.

Two studies have experimentally demonstrated rescue effects, in which immigration maintains non-self-sustaining local populations. *Daphnia magna* populations exposed to different combinations of inoculum number and introduction frequency (the combination of which yielded 11 different levels of immigration rate, defined as inoculum number ×

introduction frequency), persisted longer at higher immigration rates (Drake, Baggenstos & Lodge 2005). Further, inoculum size and introduction frequency had no impact on population persistence by themselves, but it was only their combined impact as immigration rate that was positively correlated with population persistence. Dey & Joshi (2006) also found that extinctions of *Drosophila melanogaster* only occurred within subpopulations that were not connected by migration.

By rescuing small populations from extinction, migration is predicted to increase the overall persistence time of interconnected populations (i.e. metapopulations). Forney & Gilpin (1989) compared population persistence of two species of *Drosophila* within single large habitats, and habitats that were subdivided into two smaller habitats with or without passageways allowing movement. While extinction rates of both species were lowest in the single large habitat, there were fewer extinctions in subdivided, connected habitats relative to unconnected controls for the species that readily migrated (*D. pseudoobscura*) than for the species that did not migrate (*D. hydei*) (Forney & Gilpin 1989). In several instances, a rescue effect was observed where extinct *D. pseudoobscura* subpopulations were repopulated by migration from the other subpopulation.

Similar effects occur in multispecies systems. In an experiment where protist predators *Actinosphaerium nucleofilum* could not migrate between subhabitats, but the ciliated protozoan prey *Tetrahymena pyriformis* could migrate, Amezcua & Holyoak (2000) found that prey migration from 'source' habitats where prey were invulnerable to predation could rescue prey from extinction in 'sink' habitats where predation was a threat. In a similar experiment with protist predators *Didinium nasutum* feeding on protist prey *Colpidium striatum*, Holyoak & Lawler (1996a) found that predator and prey populations in subhabitats connected by migration persisted longer than those in similar sized isolated habitats and connected subpopulations showed asynchronous fluctuations; extinct subhabitats were frequently rescued by immigration.

Natural migration is often accompanied by significant dispersal mortality. The beneficial effects of migration may therefore be overestimated in these and other laboratory studies that do not account for this threat. This is particularly true of studies in which investigators manually transfer individuals among populations to simulate migration or where populations are simply separated by a partial partition (as in Forney & Gilpin 1989), because individuals that leave one population are assured of joining another population and the chance of unsuccessful migration is absent. Alternatively, this artefact is absent from lab studies such as that by Molofsky & Ferdy (2005) discussed below, where errant migration is allowed.

Population synchrony

While migration between subpopulations can increase metapopulation persistence, theory predicts that if migration rates are too high, population dynamics will be synchronized across subpopulations, thus increasing the chance of simultaneous reductions to small size where demographic stochasticity can operate (Earn *et al*. 2000). Despite extensive focus on the relative effects of migration and environmental autocorrelation in establishing spatial synchrony (reviewed in Bjørnstad, Ims & Lambin 1999) and the difficulty of disentangling these factors in natural systems, empirical data under controlled conditions to test this theory are limited.

In a study with an annual plant, *Cardamine pensylvanica*, greater connections among subpopulations increased metapopulation persistence (Molofsky & Ferdy 2005). The authors manipulated the degree of migration via seed dispersal by varying the distance between subpopulations. Populations persisted longer when connected than when isolated, and persistence declined with average distance between populations because fewer seeds successfully migrated between populations (i.e. migrant mortality was higher). Where populations were sufficiently close to be effectively continuous, extinction levels were similar to those found in metapopulations with very low levels of connectance. The previously discussed study by Dey & Joshi (2006) using *Drosophila melanogaster* examined high, low, or no migration and similarly found that high migration rates synchronized subpopulation fluctuations, while low migration rates did not. However, no extinctions were reported at either level of migration.

Similar effects can be expected when predator–prey dynamics are included. Holyoak & Lawler (1996b) varied the degree of migration between subhabitats where *Didinium nasutum* fed on *Copidium striatum* (protozoans). They found that variation between subhabitats decreased with increasing migration and that extinction was lowest at intermediate levels of migration. Finally, Burkey's (1997) study referred to above, also examined the influence of migration between subhabitats and found that populations in habitats that were linked by dispersal went extinct significantly sooner than those in similarly sized, isolated habitats. Burkey hypothesized that subpopulation synchronization led to lower persistence times in linked habitats, but did not demonstrate that subpopulations were in fact synchronized.

Studies discussed to this point have examined demographic stochasticity within isolated populations. However, populations are generally not isolated, and interactions within a community context may also reduce population sizes to levels where demographic stochasticity becomes important. As a single example, Leslie *et al*. (1968) found that *Tribolium castaneum* generally was competitively superior to *T. confusum*. However, in 4 of 40 competition trials, adult *T. castaneum* were reduced to fewer than 10 individuals, from which they never recovered and finally went extinct.

In conclusion, available laboratory evidence provides mixed support for demographic effects predicted by theory. The effect of demographic stochasticity on population fluctuations, and thus on extinction, appears to scale as predicted by theory. Also consistent with theory, moderate migration can decrease extinction risk, and high levels of migration can synchronize population dynamics between subhabitats. However, while population extinction does correlate with population synchrony, evidence is lacking to determine

whether synchronization of population dynamics is the cause of observed extinctions. Finally, available evidence does not support the predicted correlation between extinction time and initial population size.

GENETIC EFFECTS

It has been argued that most species are driven to extinction by environmental or demographic factors before genetic factors have time to act (Lande 1988). However, copious evidence demonstrates that inbreeding depression and the loss of genetic variation decrease fitness when populations become small through reduction of large populations to a few individuals (bottleneck) or initial introductions of a few individuals (founder events) (James 1970). Reduced fitness in turn can increase extinction risk (reviewed in Frankham 2005). We separate our review of genetic effects into two categories: inbreeding depression and migration.

Inbreeding depression

Reed *et al*. (2003) examined extinction in *Drosophila melanogaster* populations with full-sib mating vs. effective population sizes (N_e) of 10 or 20 individuals. Across all three treatment levels, population extinction rates consistently increased with inbreeding coefficient, and were highest in treatments with full-sib mating. Further, all three treatment levels had lower fitness than outbred control populations, as measured by the number of adult progeny produced by five male-female pairings from each extant population at generation 60.

The amount of inbreeding that can occur before population survival is reduced has important implications for managing small populations. Frankham (1995) argued that monotonically decreasing survival with increased inbreeding was a spurious result derived not from inbreeding, but from the accumulated impacts of environmental factors. He reanalysed five previously published data sets from laboratory studies on *Drosophila* spp. to argue that the effects of inbreeding had a threshold effect on extinction risk, with no effect at low levels of inbreeding, but rapidly increasing extinction risk at intermediate (inbreeding coefficient of 0·3) to high levels of inbreeding.

Inbreeding depression can act through multiple pathways to influence extinction risk. Radwan (2003) found that inbreeding depression in bulb mites *Rhizoglyphus robini* subjected to continuous sib-mating caused extinction of genetic lines through increased sterility and preadult mortality. Effects of inbreeding may also manifest as decreased ability to adapt to stressful environments. For example, Reed & Bryant (2000) examined extinction in housefly *Musca domestica* populations initiated with 5, 50, 500 or 1500 individuals. After 68 generations, five of six populations started with 50 individuals were extinct, with high correlation between time to extinction and average fecundity ($R^2 = 0{\cdot}90$, $P < 0{\cdot}05$), while all populations in other treatments persisted. After 28 generations the response to environmental stress was estimated by comparing larval viability when exposed to thermal or dietary stress. Under stress, larvae from populations started with 5 or 50 individuals had lower viability than those initiated with higher numbers. Thus, while populations that experienced an extreme founder event (5 individuals) did not go extinct within this experiment, they did appear to have a decreased capacity to adapt to stressful environments compared to populations that had not experienced a similar founder event. In a similar study conducted with *Drosophila melanogaster*, Frankham *et al*. (1999) compared tolerance of 35 inbred and 31 outbred lines to increasing levels of NaCl. Inbred lines went extinct at lower levels of the stressful NaCl treatment than outbred lines. These studies imply that while inbred populations are capable of rebounding and persisting in benign environments, they are less capable of coping with stressful conditions. Given that most natural populations are confronted with variable and stressful conditions, inbreeding may increase extinction risk in natural populations by this route.

Further, inbreeding depression and environmental stress can interact. Bijlsma *et al*. (2000) demonstrated that extinction probability increased for *Drosophila melanogaster* populations with increasing levels of inbreeding or stress from thermal or ethanol exposure. However, when populations that had previously experienced inbreeding were subject to environmental stress, extinction rates were greater than when either occurred alone. Similarly, Reed *et al*. (2002) showed that inbreeding and chemical stress (copper sulphate or methanol) interacted to diminish *Drosophila melanogaster* population persistence.

Migration

As with demographic effects, genetic effects resulting from low population size can be prevented by migration between subpopulations. Early theoretical work suggested that one migrant per generation would maintain genetic diversity (Spieth 1974), though it has been suggested that realistically as many as 10 migrants per generation may be required (Mills & Allendorf 1996; Vucetich & Waite 2000). One experiment under controlled conditions, in which migration of bean beetles *Callosobruchus maculatus* was manipulated between zero and five females per generation, examined the rate of migration needed to reduce population extinction rate resulting from genetic constraints in small populations (Waite *et al*. 2005). A single migrant per generation between populations with a carrying capacity of 10 individuals greatly reduced the rate of population extinction, and additional migration provided no additional benefit, supporting the generalization that a single migrant per generation is sufficient to maintain genetic diversity. However, caution should be used in extending these results to field conditions where survival to reproduction is less likely. It should also be recognized that reduced population size can potentially benefit population persistence by purging deleterious alleles, and that migration between populations caries the risk of outbreeding depression.

In conclusion, experimental results confirm that genetic effects contribute to population extinction. As with

demographic effects that should decrease with total population size, avoiding extinction caused by genetic effects requires increasing the effective population size above levels where significant amounts of inbreeding occur. Also, as with demographic effects, this trap may be avoided by migration between populations.

ENVIRONMENTAL EFFECTS

As mentioned above, environmental factors commonly interact with each other or with genetic and/or demographic effects to influence extinction. Environmental influence on population extinction may be divided into two types: static environmental effects and variable environmental effects. Not surprisingly, consistently poor habitats with few resources or space can only support small populations, which in turn are susceptible to extinction via demographic or genetic effects. Thus, habitat degradation or destruction is postulated to lead to greater population extinction by decreasing the amount or quality of resources (Klok & De Roos 1998). We divide our review of environmental factors influencing population extinction into several categories: habitat quality, habitat fragmentation or destruction, environmental stressors, temporal variation, temporal autocorrelation, and spatial variation.

Habitat quality

Multiple studies have examined extinction in habitats of different quality, by reducing the amount of food available or the area of habitat. Thus, extinction rates increased for *Artemia franciscana* when populations were given lower amounts of food (Belovsky *et al*. 1999), and for *Drosophila* when food levels were reduced by more than 90% (Philippi *et al*. 1987). In the latter, the authors attributed higher extinction at low food levels to large fluctuations in population size resulting from environmental stochasticity. However, given that experimental environments were constant, demographic stochasticity or population cycles may also have caused observed fluctuations. Similarly, populations of both *Drosophila pseudoobscura* and *D. hydei* exhibited higher extinction rates due to demographic stochasticity in small habitats than populations in habitats that were twice as large (Forney & Gilpin 1989).

The influences of habitat quality can differ when multiple trophic levels are considered. Predator–prey models predict that nutrient enrichment can increase the amplitude of population oscillations (the 'paradox of enrichment', Rosenzweig 1971; May 1972), resulting in extinction. Using the predatory protozoan *Didinium nasutum* preying on the protozoan *Colpidium striatum*, Holyoak (2000) demonstrated that higher nutrient levels resulted in higher predator densities, which in turn drove more prey populations extinct than in low nutrient, low predator density conditions. However, the paradox-of-enrichment effect was reduced by habitat fragmentation so that prey populations were able to persist longer in subdivided habitats than in undivided habitats. In a similar study, increasing food (bacteria) available for the prey *Paramecium aurelia* resulted in its extinction due to predation by *D. nasutum* (Luckinbill 1973). However, adding methyl cellulose, which slows the rate of movement and thus the rate of contact between the species, increased time to extinction. When intraguild predation occurs, habitat quality can have the opposite effect. For example, Morin (1999) found that an intermediate predator (*C. striatum*) competitively excluded a top predator (*Blepharisma americanum*) at low levels of bacterial food production, while the two protists coexisted at higher levels of bacterial production. Thus, when populations are considered within the context of normal predator–prey interactions, greater food availability does not universally diminish extinction risk, and food abundance further interacts with the ability to escape immediate predation (whether through migration or changes of the habitat) to influence population extinction.

By including trophic interactions, the paradox of enrichment can also negate the benefits of increased habitat size. Using the *Paramecium–Didinium* system, Luckinbill (1974) showed that predators could capture all prey, in small habitats, leading to extinction. When habitat size was increased, predators depressed prey population size, but were unable to capture all individuals. However, when prey food supply was increased, predator abundance also increased via trophic transfer, and predators were subsequently able to drive prey to extinction, even in large habitats. Stability of the predator–prey system was therefore only possible in large habitats where prey populations were limited by food availability.

Habitat fragmentation or destruction

The breaking up of large habitats via habitat fragmentation can also result in population extinction. Using a series of experiments with grain beetles *Oryzaephilus surinamensis*, Bancroft & Turchin (2003) examined the influence of habitat fragmentation and decreasing food abundance: 6% of 247 populations went extinct in their experiments, all from low food treatments, suggesting that habitat quality is more important for their system than habitat size.

Two studies have incorporated community dynamics in the study of habitat fragmentation. Warren (1996) applied three levels of habitat destruction to microcosms initially containing 13 species of protists. The number of surviving species per patch greatly declined with increasing habitat destruction. However, when migration between habitat patches was possible, low habitat destruction did not reduce survival, while high habitat destruction did reduce survival, but much less so than when migration was not possible. In contrast, in communities with three trophic levels, Burkey (1997), discussed above, found that habitat destruction (by fragmentation or habitat loss) reduced survival of top predator populations, but that the presence of migration increased rather than reduced extinction risk.

Environmental stressors

One possibility for a population exposed to environmental stress is to relocate. To test this hypothesis, Shirley & Sibly

(2001) examined *Drosophila melanogaster* metapopulation extinction (the entire extinction of a set of subpopulations connected by migration) with variable exposure to cadmium. Metapopulations, consisting of populations in 10 subhabitats, persisted with or without migration in unpolluted conditions. But when exposed to cadmium, metapopulations without migration went extinct by the sixth generation. Metapopulations with migration initially persisted in unpolluted habitats only, but recolonized polluted habitats by the end of the experiment. The authors concluded that migration facilitated the spread of a previously recognized cadmium-resistant gene originally present at low levels within the population. They further concluded that a combination of recolonization of extinct subpopulations and the spread of the cadmium-resistant gene, both facilitated by migration, allowed metapopulations to adapt to the stressful environment, reducing extinction risk.

In addition, environmental stresses that do not cause extinction in isolation may cause extinction when combined with community ecological interactions. For example, Beketov & Liess (2006) examined the interaction between exposure to toxic insecticides and simulated predation (i.e. removing individuals from the population) for *Artemia* survival. Even at the highest concentration of insecticides, *Artemia* populations were able to persist, though at lower densities than populations that had not been exposed to insecticides. However, when combined with predation, population extinction occurred at all but the lowest toxicant concentration examined.

Temporal variation

While the above studies demonstrate that static features of environments can significantly influence population extinction, many environmental factors are highly variable (e.g. food availability, temperature, etc.). Temporal variation in food abundance is predicted to increase extinction risk by decreasing the long-run growth rate of the population (Davis, Pech & Catchpole 2003) and by increasing the risk of encountering depleted conditions. The experiment with *Artemia* discussed above (Belovsky *et al*. 1999) also manipulated variation in food abundance (coefficient of variation in food abundance of 0, 1 and 3). As hypothesized, they found that populations persisted for longer periods when food abundance was constant (no variation). However, there was no difference in extinction risk when food variation increased from low (CV = 1) to high (CV = 3) levels. A similar experiment with *Daphnia magna* found that variation in food abundance increased the probability of extinction and decreased the time to extinction (Drake & Lodge 2004). Similarly, in two studies in the soil mite system described above, Benton *et al*. (2001, 2002) showed that increasing the variation in food supply increased population variation, so that populations fluctuated in synchrony with environmental variations. While no extinctions were observed in either of these studies, mean population size decreased with increasing environmental variation, with effect size depending on the life stage examined. Environmental variation may therefore also increase extinction risk by the combined effects of decreasing population size and increasing population variability.

In contrast, when community interactions are considered, environmental variation in resource supply may decrease extinction risk, for instance, by prohibiting competitive exclusion (the 'paradox of the plankton', Hutchinson 1961). In two separate studies, Grover (1988, 1991) combined two species of microalgae, and found that competitive exclusion was much slower and less common when phosphorus addition was variable than when phosphorus addition was constant.

Temporal autocorrelation

Theoretical arguments have been advanced predicting that 'red shifted' (i.e. temporally autocorrelated) environments may either increase or decrease the risk of population extinction, depending on the time scale of environmental variation (Lawton 1997; Heino, Ripa & Kaitala 2000). In general, temporal environmental autocorrelation is predicted to increase extinction risk if environmental variation is high (indicating strong likelihood of catastrophic events) or if high sensitivity to environmental variation leads to overcompensatory density dependence (Schwager, Johst & Jeltsch 2006). Two studies have experimentally shown that the time scale of autocorrelated environments can influence population dynamics.

Using *Colpidium striatum*, Orland & Lawler (2004) showed that when environmental cycling between low food and high food conditions increased in frequency, population size increased and population variability decreased. However, the influence of environmental fluctuation frequency can depend on population growth rates, which may in turn depend on the size of the organism. Luckinbill & Fenton (1978) showed that a small protozoan (*Colpidium campylum*) adjusted its population size quickly in response to food levels, increasing in population variability with increasing frequency of food fluctuation, until populations eventually became extinct. In contrast, population sizes of a larger protozoan (*Paramecium primaurelia*) tracked food abundance much more slowly and thus was little influenced by the frequency of environmental fluctuation.

Autocorrelation in other environmental factors may also influence extinction risk. Petchey (2000) found that *Colpidium* and *Paramecium* populations also tracked temperature fluctuations, leading to increased population variation. Finally, Pike *et al*. (2004) directly controlled population size by culling and showed that autocorrelated culling decreased the time to extinction for springtails *Folsomia candida*.

Spatial variation

In contrast to temporal variation in food or habitat quality, spatial variation is predicted to decrease extinction risk by decoupling population dynamics across spatially segregated subpopulations, reducing the potential for simultaneous reduction of subpopulations to small sizes (Allen, Schaffer & Rosko 1993). Further, even though sink habitats are by

definition not self-sustaining, they may increase metapopulation size and thus reduce extinction risk (Howe & Davis 1991). Experimental evidence both supports and contradicts this prediction. In a study with *Paramecium tetraurelia*, Gonzalez & Holt (2002) examined interactions between autocorrelation in temperature and spatial variation in habitat quality. They created 'sink' habitats by periodically diluting cultures (i.e. imposing density reductions), which were then replenished by transferring inoculations from the undiluted 'source' populations. Consistent with theoretical predictions, they found that mean population sizes were larger in autocorrelated than in randomly variable habitats. In a study with *Tribolium castaneum*, Godoy & Costa (2005) established populations that each dispersed between two habitats. Pairs of habitats had a constant total amount of food, but differed in the extent to which food was evenly distributed or concentrated within one of the two habitats. The authors manually transferred a proportion of individuals between habitats at regular intervals. However, the authors did not detect a difference in the risk of extinction in populations that had different levels of contrast between high food and low food habitats. Further empirical studies on metapopulation extinction in variable environments and with source–sink dynamics are needed.

Many natural populations experience multiple environmental factors that may act simultaneously to influence population persistence. Mora *et al*. (2007) examined the simultaneous influence of overexploitation, habitat destruction, and environmental warming using rotifers (*Brachionus plicatilis*). Each of these factors individually caused population declines and overexploitation and habitat fragmentation had additive effects. However, both of these factors interacted with environmental warming. Thus, environmental factors may combine synergistically to induce population extinction.

While the studies described above largely support ecological theory for single populations under duress from environmental factors, population responses to these factors may change when placed into a community context with multiple interacting populations. For example, Bull *et al*. (2007) simultaneously examined extinction time with constant vs. variable food supply, single-habitat vs. metapopulations, and the presence/absence of apparent competition in a host–parasitoid relationship. When a single species of the host bruchid beetle (either *Callobruchus maculatus* or *C. calandrae*) was examined alone, and when the Hymenopteran parasitoid (*Anisopteromalus calandrae*) was combined with a single host species, food variation decreased time to extinction relative to constant food conditions and metapopulations increased time to extinction relative to single-patch conditions. However, when both host species were combined so that apparent competition occurred, parasitoid densities increased and parasitoids were no longer spatially constrained by the location of a single host species. As a result, none of the host species were able to escape parasitoid attack through asynchronous metapopulation dynamics, and thus food variation no longer influenced extinction times.

Community interactions themselves may also be altered by environmental factors, thereby altering extinction risk. Indeed, theory predicts that the strengths of community interactions vary along environmental stress gradients (Menge & Sutherland 1987). Long *et al*. (2007) examined competition between two protozoans (*Tetrahymena thermophila* and *Colpidium striatum*) that could migrate between habitats with constant, randomly variable, or temporally autocorrelated temperatures. When migration was not possible, competitive exclusion occurred under all environmental conditions. However, competitive exclusion did not occur when migration was possible, and the positive effect of migration was further enhanced by autocorrelated temperatures.

Similar examples of environmental factors altering community interactions that result in population extinction can be found in early studies that established the competitive exclusion principle. Park (1954) examined competition between *Tribolium confusum* and *Tribolium castaneum* and found that temperature and humidity conditions dictated the species that was driven to extinction. Thus, community interactions can alter the influence of environmental factors and in turn environmental factors may influence the outcome of community interactions that influence extinction risk.

Further, not only must the presence of community interactions be considered, but also the time scale of these interactions relative to other factors that influence population persistence. In a study with protists, Orland (2003) examined how population variability was influenced by the frequency of density perturbations through simulated predation or introductions and by the frequency of resource renewal. Population variability was lowest when both density perturbation and resource renewal occurred on the same time scale.

Finally, though it does not fit neatly into any of the three main categories of influences reviewed here, direct intervention by humans can also lead to extinction. Fryxell and colleagues demonstrated in two different studies with the ciliate *Tetrahymena thermophila* that extinction risk of harvested species depends on the harvesting strategy used (Fryxell, Smith & Lynn 2005) and decreases with the use of harvesting refuges (Fryxell, Lynn & Chris 2006) (i.e. reserves).

KNOWLEDGE GAPS

Several studies reviewed here have highlighted areas for future research to begin filling knowledge gaps. These include: (1) determining how demographic stochasticity scales with population size (Drake 2006); (2) development of methods for identifying the optimal timing and magnitude of introductions to maximize colonization success (Pike *et al*. 2004); (3) the long-term relative contributions of inbreeding depression, the loss of genetic diversity, and mutation accumulation to extinction risk (Frankham 2005); (4) the relative contributions of and interactions between genetic and non-genetic processes leading to extinction (Bijlsma *et al*. 2000; Frankham 2005); (5) the genetic impacts of habitat fragmentation (Frankham 2005); (6) factors that influence outbreeding depression (Frankham 2005); (7) effects of non-random variable environments on population growth and decline (Drake 2006); (8) the interaction between environmental variation and migration

on different time scales (Drake *et al*. 2005; Long *et al*. 2007); (9) the interaction between demographic and environmental stochasticity (Philippi *et al*. 1987); and (10) the interaction between multiple limiting resources for population growth (Bancroft & Turchin 2003).

Several authors also suggest investigating interactions between multiple factors that affect population extinction. We agree, and several studies that we reviewed here have made progress in this regard. We also extend the list given above as follows. Bijlsma *et al*. (2000) recommended examining interactions between genetic and non-genetic factors. Under this umbrella, there is a specific need to develop greater understanding of the relative importance, as well as interactions between, demographic and genetic processes leading to extinction at small population size. Much of the previous work on dynamics of small populations has used asexually reproducing species, such as *Daphnia*, which are ideal for isolating demographic effects. However, other studies that specifically focused on either demographic or genetic effects may have confounded the two by using sexually reproducing species (*Drosophila*, *Tribolium*, house flies, etc.) (Table 1). A successful approach for examining the relative importance of demographic and genetic effects could be to study closely related species that reproduce sexually and asexually under identical experimental treatments.

In addition to the above suggestions, results of some studies are contradictory and require additional investigation. For example, both Burkey (1997) and Belovsky *et al*. (1999) found that time to extinction did not depend on initial population size, yet a nearly universal prediction of theory is that population persistence should increase monotonically with initial population size, and field experimental introductions of garlic mustard *Alliaria petiolata* (Drayton & Primack 1999), bush-crickets *Metrioptera roeseli* (Berggren 2001), and two species of beetles (*Galerucella calmariensis* and *G. pusilla*) (Grevstad 1999) all found lower extinction risk in larger introduced populations. The study systems used by Burkey (1997) and Belovsky *et al*. (1999) (protists and *Artemia*, respectively) both have high reproductive capacities, and under benign conditions such as those used in their laboratory experiments, low numbers of individuals may be expected to increase rapidly. This discrepancy might be explained in part by environmental variation (which is probably higher in field experiments) that could increase the influence of demographic stochasticity on small populations. For example, encounter between a single male and a single female *Artemia* in a laboratory experiment will be much more frequent than in a lake or pond. We conclude that further experiments are needed to clarify the influence of initial population size on time to extinction.

SYNTHESES AND FUTURE DIRECTIONS

Reconciling laboratory and field experiments

The examples in the preceding paragraph highlight the fact that laboratory studies sometimes conflict with results from field studies. As an additional example, in a laboratory study described above, Bengtsson & Milbrink (1995) found that competition between *Daphnia* species increased population variability, and that variability in turn was positively correlated with extinction risk. However, the same researchers using the same species, but conducting experiments in field rock pools, found that population variability did not increase the risk of extinction (Bengtsson 1989). The underlying reasons for these differences between field and laboratory studies are not clear. Presumably, there were uncontrolled factors present in the field (such as environmental variability) that were not present in the laboratory. Identifying these factors may provide valuable information on the relative influence of different contributors to population extinction.

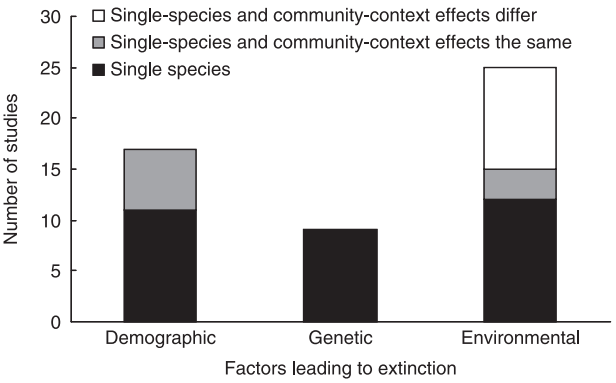

Fig. 1. Number of studies that have examined demographic, genetic, and environmental factors leading to population extinction within a single species context and within multiple species contexts where community interactions have or have not altered extinction risk.

Extinction in a community context

Several examples given above highlight that expected extinction outcomes in isolated populations are altered or reversed when populations are placed within a community context (Fig. 1). Specifically, the influences of environmental factors leading to population extinction were reversed in 9 of 12 studies where community interactions were included. However, community interactions did not reverse the influence of demographic effects in any of the six studies in which they were examined. We found no studies that examine how community interactions influence genetic factors leading to population extinction. More studies are needed to examine whether genetic drivers of extinction are influenced by community interactions, and to determine why community interactions often reverse environmental effects but do not seem to interact with demographic effects.

Quantitative tests of extinction theory

There is also a need for numerical tests of theoretical predictions. Most of the studies reviewed here examined specific hypothesized factors that influence population extinction. The majority of these studies only made qualitative

comparisons between hypothesized effects and experimental results (e.g. experimental manipulation decreased time to extinction). Very few studies have compared experimental results to quantitative predictions of population extinction (Ellner *et al*. 2001; Drake 2006; Fryxell *et al*. 2006; for examples of quantitative comparisons; but see Leslie *et al*. 1968; Molofsky & Ferdy 2005). The lack of quantitative knowledge hampers the application of theory to natural populations. For example, we know that increasing variation in food abundance can increase extinction risk (Drake & Lodge 2004; Godoy & Costa 2005), but not how sensitive populations are to increasing variation or if there are thresholds. Partly this may result from system-specific idiosyncrasies. However, an approach that may apply across different systems is to identify broad generalities that can be iteratively honed by comparison against experimental results, i.e. models that maximize generality and realism (Levins 1966). This approach has been used in establishing minimum viable population sizes (Shaffer 1981) for overcoming inbreeding depression (an initial benchmark was set at 50 individuals, or the theoretical minimum number needed to decrease the rate of inbreeding to 1% per generation, Franklin 1980) and to maintain adequate genetic variation for evolution of a typical quantitative trait (an initial benchmark was set at 500 individuals as a sufficient number to balance new variation arising through mutation and the loss of variation through selection and drift, Franklin 1980; Lande & Barrowclough 1987). Subsequent investigations suggest that these benchmarks need to increase to much higher levels (Reed & Bryant 2000). Additionally, more applicable quantitative results might be achieved by examining experimental treatments over several levels in order to detect the functional shapes of relationships and the presence of threshold effects. In addition to quantitative tests of extinction, we also need studies that explore the relative importance of various determinants of population extinction.

Migration as a unifying process

Finally, previous work has demonstrated that migration between populations can reduce extinction risk within each of the three main categories discussed here: demographic (Drake *et al*. 2005; Molofsky & Ferdy 2005), genetic (Waite *et al*. 2005), and environmental factors (Shirley & Sibly 2001). In this sense, migration is a unifying process that decreases the threat of extinction under many conditions. However, it is not the best solution in every instance. For example, increasing habitat size may improve population persistence more effectively than increasing migration between small isolated habitats (Forney & Gilpin 1989) (an issue related to the 'single large or several small' debate, Soulé & Simberloff 1986). More research is needed on the amount, timing and frequency of migration required to overcome various threats, including interactions between multiple threats, to population persistence. Further, a balance presumably exists between sufficient migration to overcome these threats and too much migration that can synchronize dynamics of subpopulations, thereby increasing extinction risk (Molofsky & Ferdy 2005; Dey & Joshi 2006). Additional research is needed to identify factors that establish the level of migration where this balance may be attained, and to determine the relative contributions of, and potential interactions between, environmental variation and migration in synchronizing populations. Finally, future research should use experimental techniques that allow for mortality associated with unsuccessful migration.

Conclusion

Laboratory studies have been instrumental in expanding and refining our understanding of population extinction. This has largely been a product of the strengths of laboratory systems: elegant experimental designs that manipulate population and environmental characteristics, and a capacity for replication at the population level. Simplified laboratory systems thus provide a rigorous first test of extinction theory. Future laboratory studies can continue to advance our understanding of extinction by incorporating increased levels of environmental and community complexity. Understanding how this complexity influences extinction risk is undoubtedly one of the great outstanding questions for population ecology.

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