Extinction

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INTRODUCTION

Extinctions are a part of life on earth. Indeed, over 99.9% of all species that once existed have gone extinct. However, humans are doing a good job in helping species into early extinction. In this talk, I fist review some classic mass extinction, and then review ecology and genetic risk factors for extinction. I finish by discussing how to assess (and defend) a species risk assessment and with some general thoughts on management strategies to migrate risk.

MASS EXTINCTIONS

Life as we know it on Earth has been shaped by Mass Extinctions – short periods of geological time when huge numbers of species suddenly went extinct. Classic mass extinctions include:

- Roughly 2 BYA (billion years ago): Most of life on earth wiped out due to pollution (O_2)
- Permian Mass extinction: 250 MYA (million years ago). 90 - 95% of marine species became extinct
- K-T (Cretaceous) Event: 65 MYA. 85% of all species became extinct (including the dinosaurs)
- End-Ice-Age Mass Extinction: (10,000 YA)
- Current on-going mass extinction.

The causes of these events have been debated, and many theories proposed. All are based around massive environmental perturbation, such as extra-terrestrial impacts, volcanoes, climate change, and biological agents. Of course, these potential causes are not mutually exclusive.

Extra-terrestrial impacts have left geologic signatures (iridium layer, shock quartz, microtektites) at the Permian and K-T extinction event boundaries. The major agent for extinction from such impacts is that massive (but short-term) environment change following the impact. There is a very recent example of such an impact: Example: Comet Shoemaker- Levy 9, which hit Jupiter in July 1994.

Super-volcanoes, and their subsequent effect of climate, have also been proposed as agents of mass extinction (indeed, some argue that extra-terrestrial impact triggers a strong cycle of volcanism). A very interesting theory is the **Toba extinction hypothesis**. Lake Toba sits in the middle of the island of Sumatra in Indonesia. Roughly 75,000 years ago, Toba exploited, the largest volcanic event in recent history, with a total energy release around a gigaton of TNT, 3000 times greater than Mount St. Helens. This led to a decrease in average global temperatures of 3 to 3.5 degrees Celsius for several years. It has been hypothesized that this event caused population bottlenecks in all of the homo species that existed at the time (a signature of such a bottleneck is found in human DNA), eventually leading to the extinction of all the other homo species except for the branch that became modern humans.

The most recent mass extinctions are likely human-induced (at least in part). The first is a massive extinction event that occurred following the end of ice-age (called the Quaternary or

Holocene extinctions), which resulted in the sudden extinction of most the North- and South American megafauna. Paul Martin has argued that this event was caused by these mammals being hunted to extinction by man, while others argue for climatic causes. Most likely this was caused by synergistic interaction between climate change coupled with hunting.

Many believe we are currently in "the sixth great mass extinction, one whose rate of loss of species exceeds any of the previous events.

ECOLOGICAL THEORY OF EXTINCTIONS

On a local level, four ecological factors can significantly increase extinction risk:

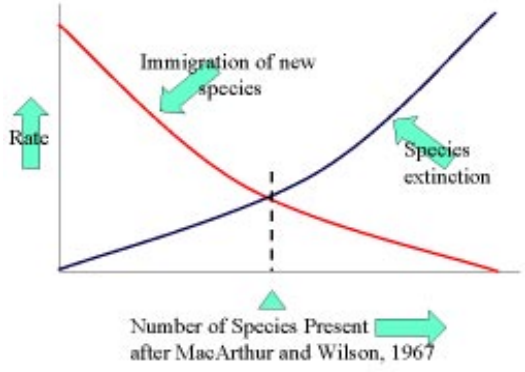
- Small population size
- Declining habitat
- Changes in other species in the ecosystems, such as removal of keystone predators or the appearance of invasive species.
- Disease
- Obviously all of these factors can interact with each other to greatly accentuate the risk.

MacArthur-Wilson Theory of Island Biogeography

Given these (and other) factors, what can theoretical ecology teach us about extinction? One of the most influential ideas in ecology, **Island Biogeography** (due to Robert MacArthur and E. O. Wilson). has widely been used as a model in conservation biology.

MacArthur and Wilson were interested in species numbers of islands, and they made two general observations. Everything else being equal, larger island have more species, as to islands closer to the mainland. Hence, the most species-rich island should be large islands near a mainland, while the most species-poor should be small very distant islands.

As the figure below shows, they viewed the number of species on an island as a balance between immigration and extinction. Larger islands have a lower extinction rate, while less isolated islands have a higher immigration rate. It was not lost on MacArthur and Wilson that many mainland habitats are really islands in a sea (or matrix) of unusable habitat.



Species-area Curves

MacArthur and Wilson noted that there was a rather predicable relationship between the number of species S on an island and the area A of that island, with S being a power function of A,

$$S = aA^z \tag{1}$$

taking logs, this reduces to a linear relationship

$$\log(S) = \alpha + z * \log(A) \tag{2}$$

The especially interesting observation that MacArthur and Wilson made was that the slope z of the species-area curve tends to fall in a rather narrow range, between 0.1 and 0.4.

Using this relationship, we can try to predict the equilibrium number S^* of species if a habitat is halved in size (from A to $A/2$). Here

$$\frac{S^*}{S} = \frac{a(A/2)^z}{aZ^z} = \left(\frac{A}{2A}\right)^z = \left(\frac{1}{2}\right)^z \tag{3}$$

Hence, the fraction of species under the smaller area relative to the initial numbers would be 93%, 87%, 81%, and 76% for $z = 0.1$, 0.2, 0.3, and 0.4, respectively. Now suppose that you are requesting funds to double or quadruple the size of a reserve. What is the expected (equilibrium) number of species? For doubling, it is 107%, 115%, 123% and 131% of the original number for $z = 0.1$, 0.2, 0.3, and 0.4. For quadrupling, the percentages are 115%, 132%, 151%, and 174%.

While predictions from the species-area curve are helpful, they are not without serious complications. Recall that this estimate represents an equilibrium balance between immigration and extinction. If we focus entirely on the reduction in area (and hence extinction), we are also assuming that immigration will not change. But this is certainly not the case, as the habitat between the reserves can also change, further decreasing immigration and further lowering the predicted equilibrium number of species. Finally, the species-area curve addresses the general number of species, NOT whether a *particular* species will persist.

Island-Biogeography Implications for Conservation

- Isolated patches of habitats are essentially islands.
- Need to maximize patch size
- Need to maximize exchange between patches
- When should you NOT maximize exchange?
  - Risk of disease/pathogens spreading.
  - When patches are sufficiently genetically different
- Many isolated habitats are out of MacArthur-Wilson equilibrium. Their size has been dramatic reduced and they have become increasingly isolated from related habitats. As a result, they will continue to lose species even if no additional habitat is lost.

Metapopulation Dynamics

The more modern version of population structure, which clearly has deep intellectual roots in MacArthur-Wilson theory, is the notion of a **metapopulation**. Essentially, this model is island biogeography with no mainland to serve as a source for immigrants. The metapopulation structure assumes the population is distributed as a series of discrete, largely isolated, patches. Extinction occurs within a patch, and that patch remains empty until re-colonized by immigrants from other patches. At any time, not all patches are occupied, but population persists by being able to colonize patches before all go extinct.

An important concept from the metapopulation model is that ***currently empty habitat can be critical to species persistence***. A further very important concept is that ***not all occupied patches are alike***. An occupied patch can either be a **source** or a **sink**. In a source, the long-term growth rate is positive and this patch contributes immigrants to other (potentially empty) patches. In a sink, that patch simply absorbs immigrants, and has a net negative growth rate. One cannot tell a source from a sink without long term studies, esp. involving population movement.

Actual populations may show departures from the classic metapopulation model

- **Core-satellite model:** A central core source population, with all other patches being sinks.
- **Patchy population model:** Even though the population has a patchy distribution, dispersal events are too frequent to allow for extinctions. Here individual patches support parts of a single population (as opposed to the metapopulation structure, where each population is largely separate)
- **Declining population model:** Here each subpopulation is a sink, so that the entire population is on its way to extinction.

Estimating Species Diversity in a Patch

Suppose you are trying to estimate the number of species (say moths) in a patch. You have done a number of surveys and have recorded a total of S species. Clearly S is an underestimate for the actual number T of species that use the patch. How can we estimate this? Simple jackknife estimator: Our estimate of T is just S plus the number species C seen on just one sampling period,

$$T = S + C \tag{4}$$

For example, if we have seen 250 species, 40 of which we only seen in one sampling period, our estimate of T is 250 + 40 = 290.

Key Implications from Metapopulation Model

- A static snapshot of the population distribution is very misleading.
- Currently unoccupied habitat may be critical for future success.
- An occupied habitat may in fact be a sink, so setting only this area aside as the reserve will doom the species.
- With human intervention, even sink populations are critical, as these can serve as sources to export individuals to currently unoccupied patches.

Demographic Stochasticity

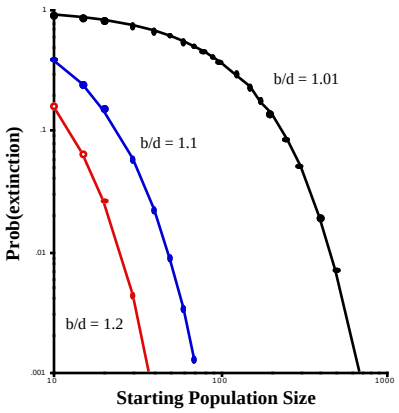
Random fluctuations of birth and death rates can lead to extinction, even in a population with a positive growth rate. Such random changes are called **demographic stochasticity**. The simplest model of this (but a classic) is due Ludwig (1971). Recall that the population growth rate r is the difference between the birth b and death d rates,

$$r = b - d \tag{5}$$

Ludwig showed that if the population starts at size n_0, probability of persistence is

$$P = \begin{cases} 1 - \left(\frac{d}{b}\right)^{n_0} & b > d \\ 0 & b < d \end{cases} \tag{6}$$

Note that when $b > d$, $r > 1$, and the population will (if it survives) grow without limit. Thus, ***small populations under positive exponential growth can still go extinct.*** Some sample values from Equation 6 are plotted below.



Extinction Times Tend to be Exponentially Distributed

All populations are finite, and as such all will eventually be driven to extinction by demographic stochasticity. The key here is the expected time. For some populations, this might be on the order of 10^{30} or more generations, so we have nothing to worry about. Thus, the notion of eventually extinction probability has been replaced with the probability of extinction in some time window (say 200 years) or the expected time to extinction.

Under fairly general conditions, extinction times tend to follow an exponential distribution, so that if $E[T]$ denotes the expected (or mean) time to extinction, the probability of extinction within t generations is just

$$\Pr(\text{extinction} \leq t) = 1 - \exp\left(-\frac{t}{E[T]}\right) \tag{7}$$

One important feature of the exponential is that it has a long tail, so that the mean value is somewhat misleading, as it is inflated by a very long (and lucky) runs of persistence. The table below shows that the probability of extinction by the mean time is 63%.

Time	$E[T] = 100$	$E[T] = 200$	$E[T] = 500$
25	22.1%	11.8%	4.9%
50	39.3%	22.1%	9.5%
100	62.2%	39.3%	18.1%
200	86.4%	63.2%	33.0%
500	99.3%	91.8%	63.2%

GENETIC RISKS

Genetic factors leading to extinction pose a significant challenge to conservation biologists as ***even an apparently large population may harbor significant genetic risks due to it past history***. There are two major classes of genetic risk factors, both resulting from small population size (at some point in an organism's recent history). The first in ***inbreeding***, the mating of close relatives (as occurs by default in small populations). Inbreeding often results in inbreeding depression wherein mean population fitness is significantly reduced relative to an outbred population. The second is ***lack of sufficient genetic variation*** to response to environmental changes. This can occur two ways. First, if most individuals are genetically similar, a single strain of pathogen can essentially attack the entire population at once, as opposed to only attacking a factor of a more genetically-diverse population. Likewise, environment changes (and this includes the interactions with other species, perhaps newly introduced ones) pose a constant challenge to any population, and response in the population to these challenges requires sufficient genetic variation, which may be lacking.

With genetic risks, larger ***effective*** population sizes are better (i.e., fewer genetic risks). However, the key concept is that the apparent population size (for example, census size) can be an extremely misleading indicator of effective population size and hence genetic risk, as past history is critical. The good news is that molecular markers can provide significant information about an organism's past and hence its potent genetic risk.

Inbreeding

The critical parameter for describing inbreeding is the ***inbreeding coefficient F***, *the probability that the two alleles at a locus in an individual are identical by descent (descent from a single copy in a recent ancestor)*. In an individual inbred to amount F, a randomly-chosen locus has both alleles IBD with probability F and hence is a homozygote.

Finite population size, even with random mating, generates inbreeding. In particular, if the size of an ideal population (more on this shortly) is N, then the amount of inbreeding in generation $t+1$ given the amount of inbreeding in generation t is given by

$$F(t+1) = \frac{1}{2N} + \left[1 - \frac{1}{2N}\right] \cdot F(t) \tag{8}$$

If particular, if the level of inbreeding at generation 0 is 0, then

$$F(t) \simeq 1 - \exp\left(-\frac{t}{2N}\right) \tag{9}$$

To compute the genotypic probabilities under inbreeding, suppose we chose a locus at random. Denote the frequency of allele A_1 by p and the freq(A_2) by q. With probability F the two alleles are IBD, and hence this locus is always homozygous, with freq(A_1A_1) = p and freq(A_2A_2) = $q = 1-p$. If the alleles are not IBD, then the genotypic frequencies follow Hardy-Weinberg. Thus, the expected genotypic frequencies under inbreeding become

Genotype	Alleles IBD	Alleles not IBD	Population frequency
A_1A_1	$F \cdot p$	$(1-F)p^2$	$p^2 + Fpq$
A_2A_1	0	$(1-F)2pq$	$(1-F)2pq$
A_2A_2	$F \cdot q$	$(1-F)q^2$	$q^2 + Fpq$

If the genotypes A_1A_1, A_1A_2, A_2A_2 have values of $a, d, -a$, then the mean under inbreeding becomes

$$\begin{aligned}\mu_F &= a \cdot (p^2 + Fpq) + d \cdot (1-F)2pq - a \cdot (q^2 + Fqq) \\ &= a(2p-1) + 2(1-F)pqd\end{aligned}$$

Noting that the mean character value in a random mating population ($F = 0$) is

$$\mu_0 = a(2p-1) + 2pqd,$$

then the mean under inbreeding can be expressed as

$$\mu_F = \mu_0 - 2Fpqd \tag{10a}$$

More generally, if there are k loci, the mean is

$$\mu_F = \mu_0 - 2F\sum_{i=1}^{k} p_i\, q_i\, d_i = \mu_0 - B\,F \tag{10b}$$

where $B = 2\sum p_i\, q_i\, d_i$ is the reduction in the mean under complete inbreeding ($F = 1$).

Estimating the Inbreeding Depression Coefficient, B

If we can estimate B for traits of interest, we can estimate the effects of inbreeding depression. Suppose we have a series of populations with different values of F (estimation of this is covered next). Recalling that

$$\mu_F = \mu_o - BF \tag{11}$$

it immediately follows that the slope of the regression of the population mean μ_F on the inbreeding coefficient F estimates the inbreeding depression coefficient B.

Estimating the Inbreeding Coefficient, F

Recall (for one locus with two alleles) that under inbreeding the frequency of heterozygotes is just freq(Heter) = $(1 - F)2pq$. More generally, with any number of alleles at a locus, the frequency of heterozygotes equals $(1-F)$ times the frequency expected under Hardy-Weinberg (where freq$(A_iA_j) = 2p_ip_j$). Thus it follows that a simple estimate of F is just

$$F = 1 - \frac{\text{Observed freq hetrozygotes}}{\text{Expected Hardy-Weinberg freq hetrozygotes}} \tag{12}$$

Inbreeding Depression in Fitness Traits

Fitness-related traits (such as viability, offspring number, and body size) often display significant inbreeding depression, with B/μ_0 often on the order of 0.4 - 0.9 (i.e., the reduction in mean under complete inbreeding is 40 - 90% of the outbred mean.

Why do Traits Associated with Fitness Show Inbreeding Depression?

Two competing hypotheses have been proposed:

- **Overdominance Hypothesis:** Genetic variance for fitness is caused by loci at which heterozygotes are more fit than either homozygote. Inbreeding decreases the frequency of heterozygotes, increases the frequency of homozygotes, so fitness is reduced. Since some inbred lines have means for fitness traits equal to the base population, this explanation cannot be generally true.
- **"Dominance" Hypothesis:** Genetic variance for fitness is caused by rare deleterious alleles that are recessive or partly recessive (heterozygote fitness closer to the fitness of the wildtype). Such alleles persist in populations because of recurrent mutation. Most copies of deleterious alleles in the base population are in heterozygotes. Inbreeding increases the frequency of homozygotes for deleterious alleles, so fitness is reduced.

While the dominance hypothesis is sufficient to account for inbreeding depression, even a very small fraction of overdominant loci can have a major effect on the level B. Hence, even though most loci that contribute to inbreeding depression may due to uncovering of deleterious recessives, the bulk of the contribution to inbreeding depression could theoretically come from a much smaller fraction of overdominant loci.

Purging Inbreeding Depression

If inbreeding depression is caused by deleterious recessives, it may be possible to purge lines of these alleles, provided they are not yet fixed. Strategies have been proposed (expand the population and inbred) to attempt to purge captive populations of inbreeding depression, but these remain controversial. Natural populations that historically have had small populations may have already purged themselves (to at least some degree) of inbreeding depression. Otherwise, they likely would have already gone extinct.

Effective Population Size, N_e

When the population is not ideal (e.g., changes over time, unequal sex ratio, uneven contribution from individuals), we can still compute an effective population size N_e which gives the size of an ideal population that behaves the same as our population.

First, suppose that the ***population size changes over time***. In such cases, the effective population size is highly skewed towards the smallest value. If the populations sizes have been $N(1), N(2), \cdots, N(k)$, the effective population size for this series is given by the **harmonic mean**,

$$N_e = \frac{k}{\sum_{i=1}^{k} \frac{1}{N(i)}} \tag{13}$$

For example, if the population size have been 10000, 10000, 10000, and 100, the arthimetric mean population size is 7525. However, the harmonic mean is

$$N_e = \frac{4}{\frac{1}{10,000} + \frac{1}{10,000} + \frac{1}{10,000} + \frac{1}{100} +} = 399$$

Thus, for a population with this history, inbreeding accumulates at the same rate as an ideal population of size 399.

Populations can also depart from ideal by having ***unequal sex ratios***, with N_m males and N_f females. In such cases,

$$N_e = \frac{4N_m \cdot N_f}{N_m + N_f} \tag{14}$$

Notice that if $N_f = N_m = N/2$ (equal sex ratio in a population of size N), then Equation 14 gives $N_e = N$. By contrast, suppose we used 2 male salmon to fertilize the eggs of 1000 females. What is N_e in this case?

$$N_e = \frac{4 \cdot 2 \cdot 1000}{1002} = 8$$

Using this few males wastes the effort of using all of those females.

Our final complication is subtle, but important. Individuals, on average, contribute different number of offspring to the next generation. For example, the 2 male salmon above might have left 50 and 1500 offspring, respectively. One might expect that this gives a smaller N_e than if they, say, contributed essentially the same number of offspring. This thought is indeed correct, and we formalize it here. Let σ_o^2 be the variance in offspring number left by parents. Then

$$N_e \simeq \frac{2N}{\sigma_o^2 + 1} \tag{15}$$

One common model is for parents to contribute, on average, two offspring (for a replacement of the male and female parent), with offspring number given by a Poisson distribution with mean 2 (this means each parent has the same chance as any other parent of leaving offspring). In this

case, $\sigma_o^2 = 2$ and $N_e \simeq N$. However, suppose we force the population so that each parent leaves EXACTLY one male and one female. In this case, $\sigma_o^2 = 0$ and $N_e = 2N$. This is a rare case where the effective population size is larger than the actual population size. In a survey of reproductive success in birds, Grant found that $\sigma_o^2/2$ ranged from 1.2 to 4.2, giving an N_e of only 40 - 90% of the actual number of females.

Insufficient Genetic Variation: Response to Selection

A population that has had a small effective population size not only has a large $F(t)$ value (and hence the potential for significantly inbreeding depression), but also has reduced genetic variation to boot. The latter has implication for the population's ability to adapt to environmental changes.

The expected selection response R (change in mean) in a trait under selection is given by the **breeders' equation** $R = h^2 S$. Here S is the within-generation change in the mean and h^2 the heritability of the trait (runs from 0 - 1, typical value around 0.2 - 0.4). For example, suppose a very hot summer selects for smaller adult mice, so that mean body size before the heat was 10cm, while the mean of heat survivors is 6cm. If h^2 for body size is zero, NONE of this within-generation change is passed onto their offspring, and mice in the next generation are the same size as the pervious generation. If $h^2 = 0.05$, the mice are (on average) $Sh^2 = (6 - 10) \cdot 0.05 = -0.2$cm smaller. If h^2 = 0.4 (a typical value for body size, then $Sh^2 = -1.6$, and the mice are 1.6 cm smaller. Obviously, if the response is not large enough, the population cannot keep pace with environmental change.

The effect of finite population size is to reduce heritability in a very large population h_0^2 to

$$h_t^2 = h_0^2 \, \frac{1 - F_t}{1 - h_0^2 \, F_t} \tag{16}$$

For example, suppose the base population heritability is $h_0^2 = 0.4$ and the level of inbreeding if $F_t = 0.5$, then $h_t^2 = 0.25$.

Genetic Measures of Subpopulation Isolation

Knowledge of the amount of genetic differentiation between (apparently) isolated populations is critical. If the populations are sufficiently distinct, we need to treat populations as separate entities. If they are sufficiently similar, a metapopulation approach can be considered.

How can we access just how different two populations are genetically? The key is that DNA from natural populations is highly **polymorphic**, in that if we looked at the DNA sequence for a random collection of the same chromosome (say the X chromosome), no two sequences would be the same (except for identical twins). In Humans, one polymorphism occurs roughly every 100 to 1000 bases. Any two random humans differ by over 20 million DNA differences. This natural variation in DNA provides us with a richly abundance set of **genetic** (or **molecular**) **markers**. One commonly used class of marker in conservation biology are **STRs** or **simple tandem repeats** (also called **microsatellites**). STRs are variations in the lengths of short repeated regions. For example, –ACACAC— vs. –ACACACAC— (e.g., AC_3 vs AC_4). Such differences are easily scored with a variety of DNA sequencing technologies. STR loci tend to be very highly polymorphic.

One standard measure of genetic differentiation between populations using the allele frequencies at molecular markers is Wright's F_{st} statistic. Suppose we have two (or more) populations. We

can partition the total variation into the fraction within each population and the fraction due to between-population differences. The later is Wrights F_{st}. A small F_{st} value implies very little genetic differentiation between populations.

While F_{st} is widely used as a measure of population-level genetic divergence, it is not without problems. The STR markers that are typically used are neutral (not under selection), in which case F_{st} is a measure of the amount of time that populations have been separated. However, divergence time (by itself) may be very poorly correlated with the amount of adaptive genetic differences between populations, which is the more critical question from a conservation biology standpoint.

Sea Turtles: A Cautionary Tale

Bowen et al (2005) used molecular markers to look at population structure of loggerhead turtles on the east coast of the US. While autosomal microsatellites showed no population structure, mtDNA showed strong population structure. Recalling the mtDNA is passed on only by the mother, these results imply that females home faithfully to their natal nesting colony, but males migrate between nesting colonies. If the authors had only looked at autosomal markers, they would have inferred a very different population structure, and hence a very different conservation strategy.

TOOLS FOR ASSESSING EXTINCTION RISK

Population Viability Analysis, PVA

The standard approach for assessing the extinction risk of a population is to perform a ***Population Viability Analysis*** (or ***PVA)***. Basically, a PVA is a model, often complex, that attempts to incorporate the ecological and genetic risk factors to obtain a probability (or mean time) of extinction. The PVA model for the species under consideration has to incorporate **population size** (effects on demographic stochasticity, inbreeding, standing levels of genetic variation), **demographic parameters** (stage-specific birth and death rates, population carrying capacity), and **population structure** (geometry of patches and their connectiveness).

A Simple Example: Leslie matrices

As an example of some of the complexities introduced into a PVA, we consider the simplest model with age structure, a ***Leslie***(or projection) ***matrix***. Different age groups (on average) likely produce different numbers of offspring. Likewise, different age groups likely have different probabilities of surviving into the next age class. A Leslie matrix model allows us to model these age-class differences in viability and fecundity.

Suppose there are k age classes, where age class 1 are new-borns and no individuals live past age class k. We represent the number in each age class at time t by the vector $\mathbf{n}(t)$ where

$$\mathbf{n}(t) = \begin{pmatrix} n_1(t) \\ n_2(t) \\ \vdots \\ n_k(t) \end{pmatrix}$$

here $n_i(t)$ are the number of individuals in age class i at time t.

Let v_i be the probability that an individual in stage class i survives to stage class $i+1$. Hence,

$$n_{i+1}(t+1) = v_i n_i(t) \tag{17a}$$

Likewise an individual in age class i contributes (on average) b_i offspring, so that the new value of for the number of new borns is

$$n_1(t+1) = b_1 n_1(t) + b_2 n_2(t) + \cdots b_k n_k(t) = \sum_{i=1}^{k} b_i \, n_i(t) \tag{17b}$$

We can put these birth and death parameters into matrix form. For the matrix P, let the element in the ith row and jth column be the transition from class j into class i,

$$\mathbf{P} = \begin{pmatrix} b_1 & b_2 & b_3 & \cdots & b_{k-1} & b_k \\ v_1 & 0 & 0 & \cdots & 0 & 0 \\ 0 & v_2 & 0 & \cdots & 0 & 0 \\ 0 & 0 & v_3 & \cdots & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & \cdots & v_{k-1} & 0 \end{pmatrix} \tag{17c}$$

For example, Row 1, column 3 = contribution to class 1 from class 3, which occurs by age class 3 individuals having offspring, b_3. Likewise, Row 3, column 2 = moving from class 2 to class 3. This occurs by surviving class 2, v_2.

The numbers of individuals in the age classes in time t+1 given by matrix multiplication,

$$\mathbf{n}(t+1) = \mathbf{P}\mathbf{n}(t) \tag{18a}$$

In particular, the vector of age-class numbers in time t, given starting values, $\mathbf{n}(0)$, is given by

$$\mathbf{n}(t) = \mathbf{P}^t \mathbf{n}(0) \tag{18b}$$

Formally, the population growth rate is given by λ_{max}, the leading (largest) ***eigenvalue*** of $\mathbf{P}$. If $\lambda_{max} > 1$, the population grows, otherwise it goes extinct.

This is a ***determinstic*** analysis (assumes no random effects or sampling). We can easily add demographic stochasticity to the model by adding random sampling to the birth and death process. For example, survival is a binomial random variable, so that if n_i is the current number in age class i, the probability that $n_{i+1} = k$ (for $0 \le k \le n_i$) is given from the binomial probability

$$\Pr(n_{t+1} = k \,|\, n_i) = \frac{n_i!}{(n_i - k)!\, k!} \, v_i^k \, (1 - v_i)^{n_i - k} \tag{19a}$$

Likewise, the number of offspring contributed by class i is given by a Poisson random variable with success parameter $b_i n_i$, so that the probability of k offspring ($0 \le k < \infty$) is given by

$$\Pr(\text{class } i \text{ contributes } k \text{ offspring}) = \frac{(b_i n_i)^k \; e^{(-b_i n_i)}}{k!} \tag{19b}$$

Thus in a PVA, one starts with some initial age-class numbers vector and then has a computer draw random variables using Equations 19a/b to generate the updated age class vector. This can be run over some specific time period and we record (for each run) either extinction or no extinction to give an estimate of the probability of extinction in the time window. Likewise, we could continue each run until extinction, and the time to extinction recorded. Obviously, one must do a fairly large number of runs (at least 500 to 1,000) to obtain accurate estimates of either the probability of extinction or the time to extinction for the given set of life-history parameters.

Using this basic structure, we can build up a complex model. For example, a typical model might have a metapopulation structure. Within each population, dynamics given by a stochastic Leslie matrix, and migration then occurs between subpopulations. As above, the model is run with a set of parameters to generate a probability of extinction or a time to extinction.

Comparing (and Defending) PVAs

A PVA is an attempt to model a complex process, typically with very incomplete (and potential rather inaccurate) data. This is done in an environment wherein the results of just above any PVA are likely to be challenged for being both an underestimate (conservationists) AND an overestimate (developers) of the risk. Besides using "the best possible data" (the legal mandate) what else can be done to support the findings of a PVA?

Any analysis should examine the ***sensitivity*** of the modeling assumptions — if we make small changes in the parameters, how robust are our findings? Besides examining sensitivity of the parameters, one also needs to examine the sensitivity of the *general structure* of the model. For example, in an assumed population structure, what happens if we change a 0 value of immigration between two demes to some very small number? Any summary result from a PVA (such as time or probability of extinction) should (at a minimum) be reported as a ***confidence interval*** rather than a point estimate.

A scientifically, and statistically, justifiable approach that jointly deals with BOTH model sensitivity and model outcome uncertainty is offered by generating a ***Bayesian posterior distribution*** for the PVA parameter of interest. One can also use this approach to justifiably contrast the PVAs under two different actions (for example, before and after building a campground) to formalize the impact of a proposed project/action.

Here is how one generates such a posterior distribution. One has prior distributions for model parameters (such as viability and fecundity). These distribution can reflect statistical uncertainty in the estimation of the model parameters and/or any prior assumptions we have about these distribution. One them samples a vector of the model parameters from the distribution, using these values in a PVA to generate a summary statistic (probability or extinction time). One then repeats this process of randomly sampling a vector of model parameters and using thse to generate a PVA several thousand times.

The net result is a posterior distribution of the PVA summary statistic that reflects both model uncertainty and also incorporates a sensitivity analysis.

Mimimal Viable Population, MVP

A closely-related approach to PVA is a ***minimal viable population*** size (and structure when a metapopulation is assumed). One can obtain this by setting some criteria for viability (e.g., >

80% probability of not being extinct in 300 year) and then running different population sizes (and potentially population structures) through a PVA to estimate this value.

MANAGEMENT STRATEGIES TO MIGRATE RISK

The major take-home points from the ecological and genetic theory are as follows:

- The larger N (and N_e) the better. This usually means a larger area.
- Even populations with a large N can be doomed because of lack of genetic variation from previous events (large N, small N_e).
- In such cases, crosses to closely related populations might be considered. F_{st} values can help determine which crosses to make.
- Population structure is critical.
- Currently empty patches of habitat may still be critical.
- Patches can be sinks or sources, and it is critical to be able to distinguish between these. Takes long-term data.
- The habitat between patches may also be very critical to species success.
- Bottom line: Need ***dynamic management***, constantly updating a survival strategy as new information is obtained. This needs to be built into a PVA.

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