

Lecture 19, 24 Oct 2006
CH6 Genetics, CH7 Populations

Conservation Biology
ECOL 406R/506R
University of Arizona
Fall 2006

Kevin Bonine
Kathy Gerst

Conservation Genetics



PVA etc.

Lab this week:

7am 27-29 7pm October = ORPI, Pinacate, CEDO
(\$, food, see website for lab readings)

Housekeeping, 24 October 2006

Upcoming Readings

today: [Text Ch.6 and 7](#), [PVA](#), [Puma concolor](#)

Thurs 26 Oct: Guy McPherson (web for climate change reading)

Tues 31 Oct: Ed Moll (long web reading)

Thurs 02 Nov: Exam Two

Tues 07 Nov: Don Falk (web reading)

Thurs 09 Nov: Conservation Practices (Ch 10, Donlan EA 2005)

Short oral presentations :

24 Oct Cori and Robert

09 Nov Jon and Laura

14 Nov Dan and Lane

28 Nov Amanda and Fred

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Global Climate Change Lecture Series

All lectures will take place at UA Centennial Hall.

All lectures begin at 7pm and are free to the public. Call 520.621.4090 for more information.

Tuesday, October 17
Global Climate Change: The Evidence
Malcolm Hughes, Professor of Dendrochronology

<http://cos.arizona.edu/climate/>

Tuesday, October 24
Global Climate Change: What's Ahead
Jonathan Overpeck, Director of the Institute for the Study of Planet Earth and Professor of Geosciences

Tuesday, October 31
Global Climate Change: The Role of Living Things
Travis Huxman, Assistant Professor of Ecology and Evolutionary Biology

Tuesday, November 7
Global Climate Change: Ocean Impacts and Feedbacks
Julia Cole, Associate Professor of Geosciences

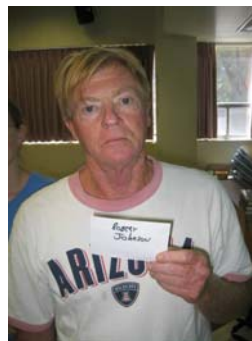
Tuesday, November 14
Global Climate Change: Disease and Society
Andrew Comrie, Dean of the Graduate College and Professor of Geography and Regional Development

Tuesday, November 21
Global Climate Change: Could Geoengineering Reverse It?
Roger Angel, Regents' Professor of Astronomy

Tuesday, November 28
Global Climate Change: Designing Policy Responses
Paul Portney, Dean of the Eller College of Management and Professor of Economics

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Cori and Robert
will speak for 10 minutes on xx



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Applications of Genetics to Conservation Biology

-Molecular Taxonomy

- Populations, Gene Flow, Phylogeography
- Relatedness, Paternity, Individual ID



Dr. Melanie Culver
SNR, UA



Molecular Taxonomy: Molecules versus Morphology

- **Cryptic species** (sibling species)
- Morphological variation without genetic variation

Relatedness (Kinship, Paternity and Individual ID)

Application of molecular genetic techniques, using **hypervariable, repetitive DNA**

(ie. microsatellites, minisatellites)

to questions of kinship, paternity or individual ID

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Populations, Gene Flow, Phylogeography

-**Compare** genetic traits among populations

-Resolve **substructure** among populations

-Infer **movement** patterns among individuals

-Infer **historical events** for species

8

Non-Invasive Sampling

- Allows sampling without disturbance to individual
- Rare or hard to capture species
- Examples (hair, scat, feathers, saliva/cheek swab, regurgitated pellets, dried blood, biopsy dart, museum tissues)

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Subspecies Taxonomy, Phylogeography, Gene Flow:
Puma (cougar, mountain lion)



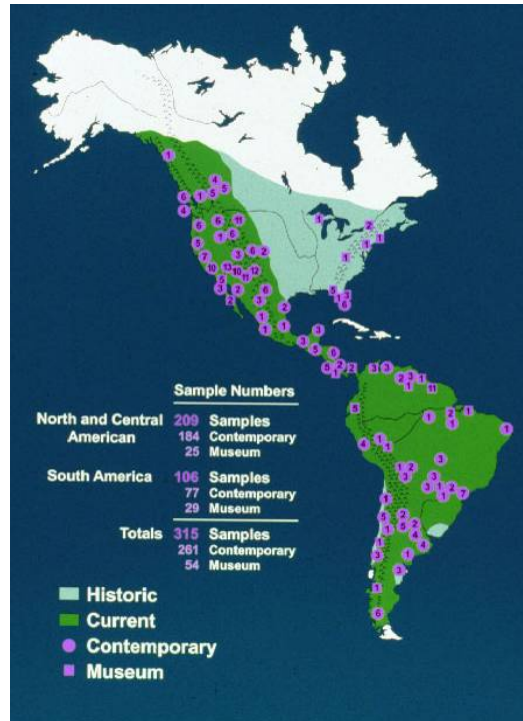
32 Puma subspecies,
as of the early
1900s



Objectives

- Does current population differentiation reflect
 - Trinomial descriptions?
 - Physical or ecological barriers?
 - Isolation by distance?
- Are current levels of genetic variation the same within each population?
- Does population structure and genetic variation reflect
 - Historic migrations?
 - Historic dispersals?
 - Historic bottlenecks?

Modern and museum puma samples collected, total of 315



Molecular Methods Used

- Mitochondrial gene sequencing
 - 16SrRNA
 - NADH-5
 - ATPase8
- Nuclear microsatellite length determination
 - 10 domestic cat microsatellite loci

Neutral Markers often studied.

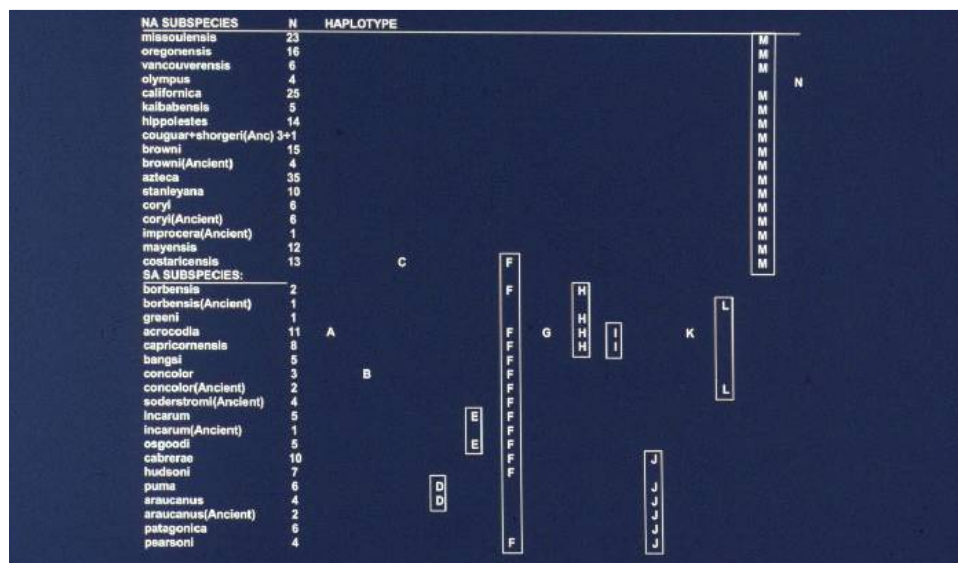
Relevance to natural selection and adaptation?

Ultimately, source of all variation is mutation.

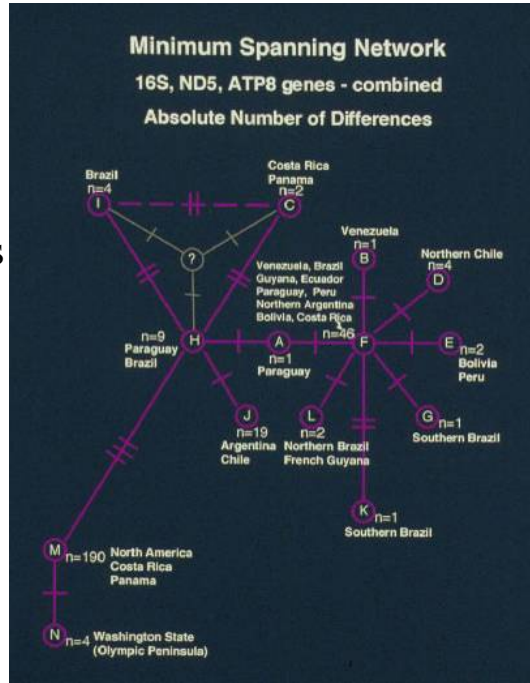
mutation rate = $10^{-4} - 10^{-6}$

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Mitochondrial DNA Haplotypes (in a geographical cline)



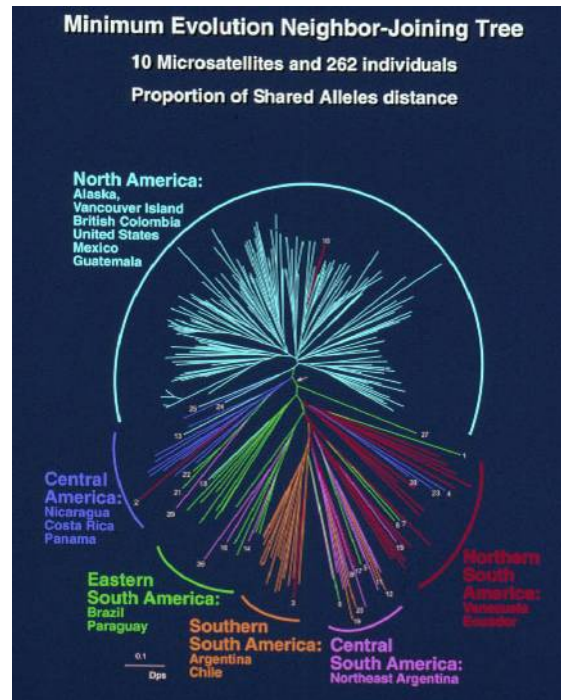
- Ancestral haplotypes
- 2 historical radiations
- NA is most recently founded population



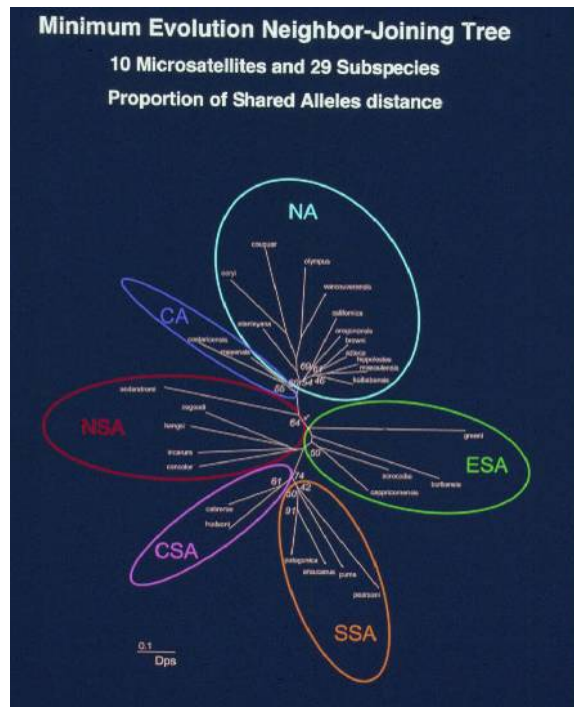
Microsatellite Alleles at FCA008

Group	Subspecies	Tot no/ individual	Allele size																			
			134	136	138	140	142	144	146	148	150	152	154	156	158	160	162	164	166	168	170	172
NA	missoulensis	21												25						16	1	
	* oregonensis	14												23						2		
	* vancouverensis	6												12								
	* olympus	4												8								
	* californica	23												43						3		
	* kalabarensis	3												5						1		
	* hippolestes	12												19						5		
	* cougar	1												2								
	* browni	17												30						4		
	* azteca	33												59	1					6		
* stanleyana	10												20									
* coryi	6												12									
* mayensis	11	3											18						1			
CA	costaricensis	13	9									1	7						3	3	1	2
ESA	borbensis	2											1						1			2
	* greeni	1											1						1			
	* acroodia	12	1		1	2	2				1	1		6	6	1			1		2	
* capricornensis	8	3		1						1	1	1	2	5				1			1	
NSA	bangsi	5	2										2								1	
	* concolor	5	1	1		1							3	2								
	* soderstromi	2											1		1							
	* incarum	5		1									4	1	1			3				
	* osgoodi	5		4									2	1				1	1		1	
CSA	cabrerae	10	2	3	1								2	6					4		1	1
	* hudsoni	7		1									4					3	2		2	2
SSA	puma	3											1	1					1		2	1
	* araucanus	4											1					1		1	3	2
	* patagonica	6											3					2			5	5
	* pearsoni	4											1	1				3			1	2

- Geographic clustering of individuals
- ~Six groups identified
- 2 distance methods agree



- Subspecies associate into same 6 groups
- Statistical support from bootstrap values
- 2 distance methods agree



BOX 11.3 Calculation of F-Statistics

Derrick W. Sugg, University of Georgia, Savannah River Ecology Laboratory

F_{IS} , F_{IT} , F_{ST}

Fixation indices, or F-Statistics, were developed by Sewall Wright (1922, 1965, 1969, 1978) as a means to describe how genetic diversity is partitioned in a population. By partitioning genetic diversity into different components one can determine the relative amounts residing within individuals, subpopulations, and the overall population. Because adaptive evolution requires genetic variation to proceed, it is important to understand how much of the total variation is available for selection acting on individuals. More recently, conservation biologists have shown renewed interest in fixation indices because they provide a means to determine how natural populations maintain genetic variation (beneficial for developing management strategies) and to determine levels of genetic variation in threatened or captive populations (beneficial for assessing the success of management strategies).

Typically when one calculates fixation indices it is for a structured population. The classical approach is to sample individuals from different subpopulations at fairly distinct geographic locations. Such a population is said to consist of three levels of structure: individuals (*I*), subpopulations (*S*), and the total population (*T*). One calculates the average individual heterozygosity by counting the number of heterozygous individuals in a subpopulation and dividing that sum by the total number of individuals in the subpopulation. This calculation is made for every subpopulation, and the average for all subpopulations is called the average individual heterozygosity:

$$H_I = \frac{1}{k} \sum_{i=1}^k \frac{\# \text{Heterozygotes}_i}{N_i}$$

where *k* is the number of subpopulations and *N_i* is the number of individuals in the *i*th subpopulation. At the same time one can use those individuals to determine the frequency of the genes. The gene frequencies are used to calculate the expectations for heterozygosity in the average subpopulation \bar{H}_s and the total population (H_T). The expectation for the average subpopulation is

$$\bar{H}_S = \frac{2}{k} \sum_{i=1}^k p_i - p_i^2$$

where *p_i* is the frequency of the gene in the *i*th subpopulation. The expected number of heterozygous individuals for the entire population is given by $H_T = 2(p - \bar{p}^2)$ where *p* is the frequency of the gene averaged over all individuals in the population without respect to the subpopulation they came from. \bar{H}_s predicts the frequency of heterozygous individuals in subpopulations had they mated at random and H_T predicts the same frequency if individuals are mating at random without respect to subpopulations.

These estimates of the observed and expected frequency of heterozygous individuals can be used to calculate the fixation indices, F_{IS} , F_{IT} , and F_{ST} . Values for F_{IS} determine whether or not subpopulations have fewer or more heterozygous individuals than expected. It is calculated from:

$$F_{IS} = \frac{\bar{H}_S - H_I}{\bar{H}_S}$$

When there are fewer heterozygous individuals than expected ($H_I < \bar{H}_S$), F_{IS}

will be positive. When $H_I > \bar{H}_S$, then F_{IS} will be negative. Therefore, negative values for F_{IS} indicate an excess of heterozygous individuals in subpopulations and positive values indicate the opposite condition. F_{IT} is calculated in a similar manner:

$$F_{IT} = \frac{H_T - H_I}{H_T}$$

and the interpretation of positive and negative values are the same except that they apply to the total population instead of the subpopulations. Finally, the degree of genetic differentiation among subpopulations (how unique they are) is given by:

$$F_{ST} = \frac{H_T - \bar{H}_S}{H_T}$$

which is always greater than or equal to zero. High values for F_{ST} indicate that subpopulations have very different gene frequencies, and when $F_{ST} = 1$ then subpopulations are said to be "fixed" for different genes; each subpopulation has a unique gene for each locus.

Models by Wright make simplifying assumptions including equal reproductive contributions among breeding adults and a large number of subpopulation of equal and constant size contributing dispersers to the pool of migrants. More recently, Wright's models have been recast using different methodologies or by emphasizing the importance of different evolutionary forces. Readers interested in this subject area are encouraged to read additional literature in this area including Slatkin (1991), Crow and Aoki (1984), Chesser (1991a,b), Wade and McCauley (1988), and Whitlock and McCauley (1999).

Groom, Meffe, & Carroll 2006

Wright's Fst Estimates and Slatkin's Migration Estimates

mtDNA	NA	CA	ESA	NSA	CSA	SSA
NA	-	0.1	0.1	0.02	0.03	0.1
CA	*0.784	-	8.3	0.5	1.6	1.6
ESA	*0.815	0.057	-	0.8	2.3	2.2
NSA	*0.958	*0.492	0.384	-	4.2	0.5
CSA	*0.935	0.233	*0.177	*0.107	-	1.3
SSA	*0.835	0.240	*0.186	*0.526	*0.281	-

(Fst near 0 = little divergence)

(Migrants/generation)

microsatellites	NA	CA	ESA	NSA	CSA	SSA
NA	-	4.0	4.4	8.0	2.2	0.9
CA	*0.110	-	2.3	3.5	3.5	1.2
ESA	*0.103	*0.179	-	15.7	4.8	1.0
NSA	*0.059	*0.126	*0.031	-	6.0	1.1
CSA	*0.186	*0.126	*0.094	*0.077	-	2.4
SSA	*0.367	*0.288	*0.330	*0.316	*0.172	-

Summary:

- 6 groups identified using microsatellites
- mtDNA haplotypes overlaid onto map, supports 6 groups
- Location of 2 ancestral haplotypes

Major restrictions to gene flow:

- Amazon River
- Rio Parana
- Rio Negro
- Andes?



Fossil Record versus Molecular Divergence Estimates

- Oldest fossils in North and South America date to 0.2-0.3 Mya
- From mtDNA mutation rate of 1.15%/My, divergence for extant puma lineages is 390,000 years ago
- From mutation rate of 5×10^{-9} /yr for microsatellite flanking regions, pumas are less than 230,000 years old

Historical Inferences

- Extant pumas originated in Brazilian Highlands (ancestral haplotypes)
- Fossil record suggests dispersal to NA soon after the common origin in Brazil
- 2 historical radiation events occurred

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-Ancestor to puma crosses land-bridge ~2-3 Mya

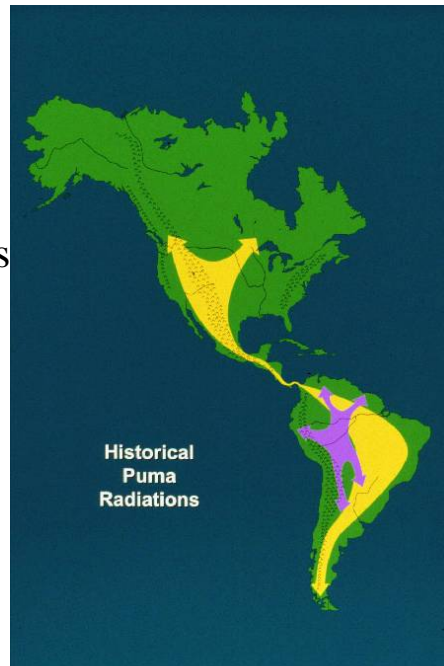
-Puma origin in Brazilian Highlands ~300,000 ya



2 Major historical radiations

-One locally distributed

-One broad ranging



Puma Bottlenecks

- Subspecies-level
 - North America low overall genetic variation
- Population-level
 - Florida monomorphic at 8/10 microsatellite loci
 - Olympic Peninsula and Vancouver Island, monomorphic at 5/10 microsatellite loci

Puma Conclusions

- Pumas originated in Brazil approximately 300,000 years ago
- Possible extirpation and recolonization in North America (Pleistocene age?)
- Molecular data does not support 32 subdivisions, instead 6 groups
- Pumas are fairly panmictic within 6 groups

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Conservation Implications

- Maintain habitat connectivity within 6 large groups
- Management should consider effects of bottlenecked populations
- Eastern cougar, Florida panther and Yuma puma management take into account revised subspecies

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What is population viability analysis? (PVA)

Thanks to Margaret Evans, 2003

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Population Dynamics

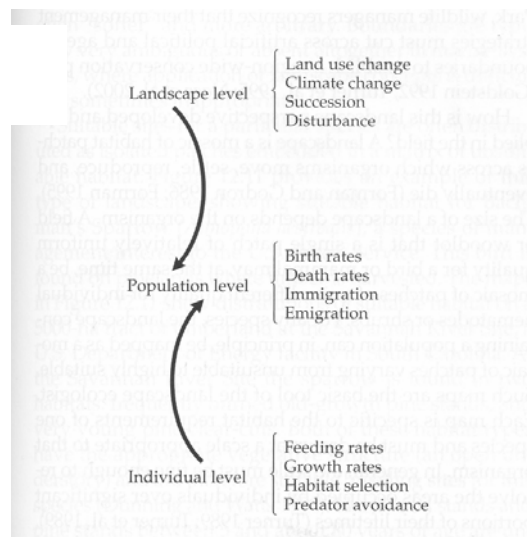
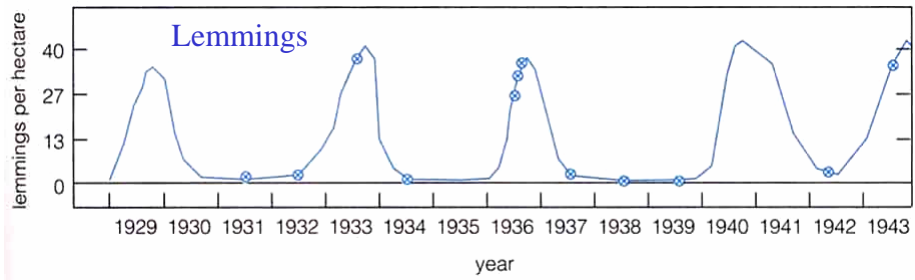


Figure 12.10 Population dynamics should be understood as resulting from a hierarchy of processes affecting populations at different levels. Landscape-level changes in the availability of habitat determine how much suitable habitat exists for a given species, and its configuration (and therefore its accessibility). The availability of suitable habitat and the behavior and physiology of individual organisms combine to influence the dynamics of populations.

Groom, Meffe, & Carroll 2006

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populations are dynamic, not static

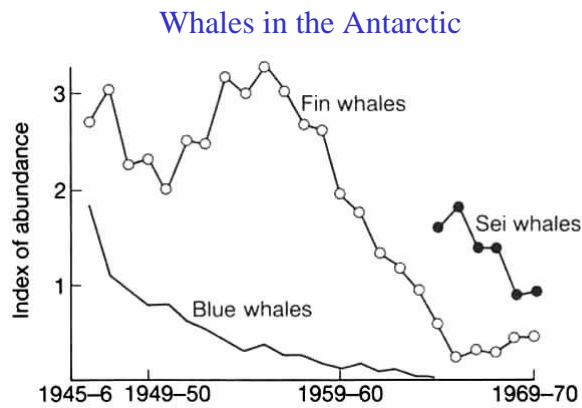


Cause of cyclic change in population not completely understood. Cycle length average 3.8 years Mass migration in response to high density with decreasing food supply, sometimes swimming involved.



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populations are dynamic, not static



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Population sizes change over time

Why?

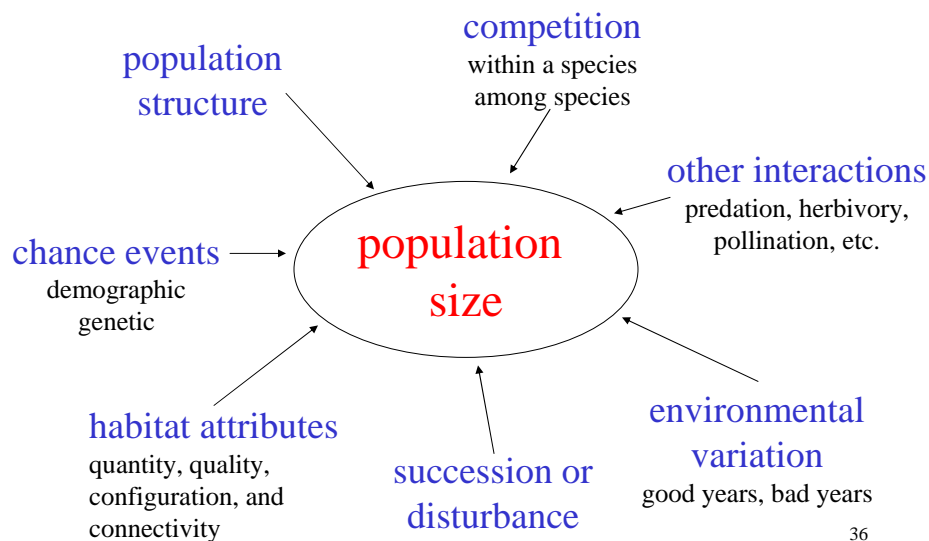
What causes change in population size?

What regulates population size?

If we can answer these questions, we might be able to make changes that increase populations of declining (endangered) species

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Many things affect population size

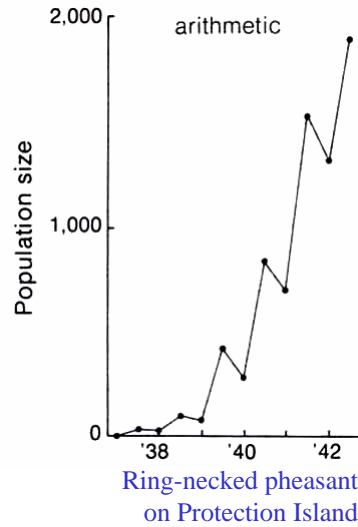


1. Exponential growth

density-independent, deterministic

In a closed population (no immigration or emigration), population growth is a function of birth and death rates

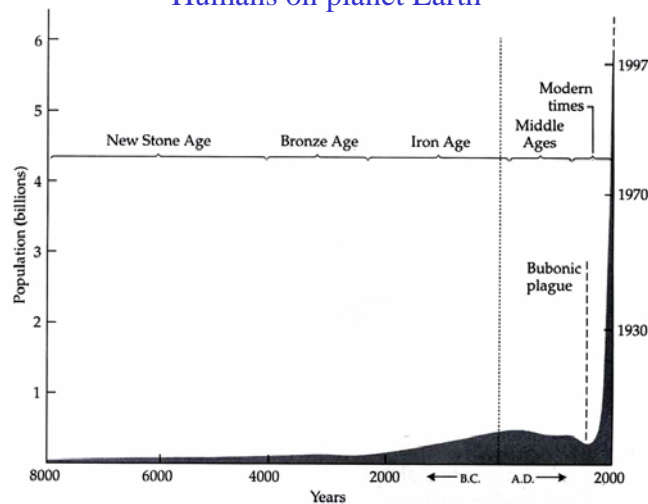
$$\frac{dN}{dt} = (b-d)N$$



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exponential growth: an unrealistic model?

Humans on planet Earth



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2. Logistic growth

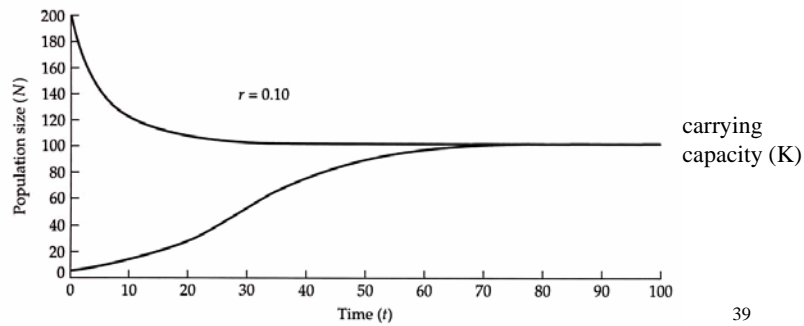
density-dependent, deterministic

$$\frac{dN}{dt} = rN \left(\frac{K-N}{K} \right)$$

intraspecific competition

stabilizes population size

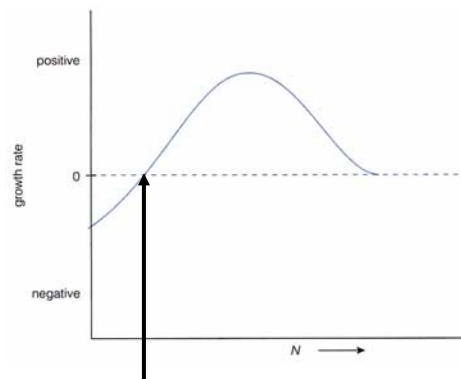
birth rates go down and/or death rates
go up with increasing population size



Alternatively,

The population growth rate may increase with
population size (positive density-dependence)

Allee effect



minimum viable population size

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Allee effect

How?

In animals:

- group defense against predators
- group attack of prey
- mates difficult to find
- critical number to stimulate breeding behavior

In plants:

- pollinator limitation
- self-incompatibility
- inbreeding depression

37 Passenger Pigeon (adult male).



Allee effect

How?

group defense against predators



Figure 7.6

The sage grouse (*Centrocercus urophasianus*), a gallinaceous bird of the western United States, gathers for mating on communal display and breeding grounds known as leks. If numbers are insufficient to promote lek formation, displays and breeding may not take place.

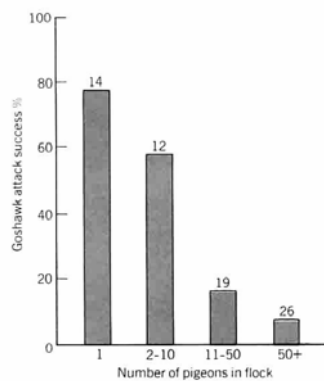


FIGURE 13.17
Success rate of goshawk attacking pigeons in flocks.

Attack by a trained goshawk rarely resulted in capture of a pigeon from a large flock, although most attacks on single pigeons were successful.

.2

The two categories of models we have considered thus far **assume** that

- all individuals in a population have the **same birth and death rates** (no genetic, developmental, or physiological differences among individuals)

under some circumstances, this might cause us to inaccurately predict population size

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3. **Structured** population models density-independent, deterministic

This is the type of model most often used in population viability analysis

What is meant by “structure”?

A population is **unstructured** if all individuals have the same rates of survival and fertility.

A population is **structured** if differences among individuals in **age**, developmental **stage**, or **size** cause them to have different survival or fertility rates.

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TABLE 6.3 Survival data for red-cockaded woodpeckers in different reproductive stages, from Walters (1990)

Stage	Total number of bird-years	Fate at the end of a one-year interval		Proportion surviving one year
		Dead	Alive	
Fledglings	616	345	271	0.44
Solitary males	131	50	81	0.62
Helpers-at-the-nest	273	60	213	0.78
Breeding males	838	201	637	0.76
Floater	29	11	18	0.62

Life Tables

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Table 7.1 A Life Table for Belding's Ground Squirrel (*Spermophilus beldingi*). Life tables, properly constructed from appropriate data, provide important summaries of age-specific demographic characteristics of plant and animal populations; n_x is the actual number of individual squirrels alive in each age interval; d_x is the number dying during the interval; l_x is the proportion of the original cohort alive at the beginning of the age interval; q_x is the mortality rate from interval x to interval $x + 1$; e_x is the life expectancy of individuals in the age interval; and x is the age interval to which the value refers. Calculations of l do not include individuals first marked as adults.

AGE (YEARS)	FEMALES					MALES				
	n_x	d_x	l_x	q_x	e_x	n_x	d_x	l_x	q_x	e_x
0-1	337	207	1.000	0.61	1.33	349	227	1.000	0.65	1.07
1-2	252*	125	0.386	0.50	1.56	248 [†]	140	0.350	0.56	1.12
2-3	127	60	0.197	0.47	1.60	108	74	0.152	0.69	0.93
3-4	67	32	0.106	0.48	1.59	34	23	0.048	0.68	0.89
4-5	35	16	0.054	0.46	1.59	11	9	0.015	0.82	0.68
5-6	19	10	0.029	0.53	1.50	2	0	0.003	1.00	0.50
6-7	9	4	0.014	0.44	1.61	0	—	—	—	—
7-8	5	1	0.008	0.20	1.50	—	—	—	—	—
8-9	4	3	0.006	0.75	0.75	—	—	—	—	—
9-10	1	1	0.002	1.00	0.50	—	—	—	—	—

Source: Sherman and Merton 1984.

*Includes 122 females first captured as yearlings.

[†]Includes 126 males first captured as yearlings.

3. Density-independent, deterministic, structured population growth

What else can structured population models tell us?

Sensitivity

The sensitivity of λ to each matrix element describes how much λ will be affected by a change in that transition probability

Would it be better to focus conservation efforts on improving the survival of hatchlings or large juveniles or adults???

(λ = population growth rate)

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When λ is **greater** than 1 the population **increases** in size

When λ is **less** than 1 the population **decreases** in size

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3. Density-independent, deterministic, structured population growth

What else can structured population models tell us?

Elasticity

Elasticities quantify the proportional change (e.g., 1%) in the asymptotic growth rate that can be expected given a particular change (1%) in each life history transition.

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Van Dyke p. 178

“Four Horsemen of the Extinction Apocalypse:”

1. Genetic Stochasticity
2. Environmental Stochasticity
3. Demographic Stochasticity
4. Natural Catastrophes

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Population Viability Analysis

TABLE A Potential Uses of PVA "Products"

Category of use	Specific use	Sources for examples
Assessment of extinction risk	Assessing the extinction risk of a single population	Shaffer 1981, Shaffer and Samson 1985, Lande 1988
	Comparing relative risks of two or more populations	Forsman et al. 1996, Menges 1990, Allendorf et al. 1997
	Analyzing and synthesizing monitoring data	Menges and Gordon 1996, Gerber et al. 1999
Guiding management	Identifying key life stages or demographic processes as management targets	Crouse et al. 1987
	Determining how large a reserve needs to be to gain a desired level of protection from extinction	Shaffer 1981, Armbruster and Lande 1993
	Determining how many individuals to release to establish a new population	Bustamante 1996, Howells and Edwards-Jones 1997, Marshall and Edwards-Jones 1998, South et al. 2000
	Setting limits on the harvest or "take" from a population that are compatible with its continued existence	Nantel et al. 1996, Ratsirarson et al. 1996, Tufto et al. 1999, Caswell et al. 1998
	Deciding how many populations are needed to protect a species from regional or global extinction	Menges 1990, Lindenmayer and Possingham 1996

Groom, Meffe, & Carroll 2006

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Evolution of Population Viability Assessments for the Florida Panther: A Multiperspective Approach
David S. Maehr, Robert C. Lacy, E. Darrell Land, Oron L. Bass Jr., and Thomas S. Hootor

IN: Population Viability Analysis.
 Steven R. Beissinger and Dale R. McCullough, eds. Univ. of Chicago Press, Chicago. xvi + 577 pps.

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-Panther Article on PVAs over time



- VORTEX
- data
- population size?
- source and sink?
- inbreeding problems?
- captive breeding?
- introgression?
- time scale?
- HABITAT LOSS



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Table 14.1 Comparison of VORTEX Model Inputs Provided Independently by the Five Authors and the Outputs Generated from These Simulations

Model Inputs and Output	Originator of Variable Estimates for the VORTEX Simulation				
	Population Ecologist (Lacy)	State Field Biologist (Land)	Federal Field Biologist (Bass)	University Landscape Ecologist (Hector)	University Conservation Biologist (Maehr)
<i>Inputs</i>					
Inbreeding depression?	Yes	No	No	No	No
Lethal equivalents	3.14	—	—	—	—
% due to recessive lethals	50	—	—	—	—
Reproduction correlated with survival?	Yes	No	No	No	No
Polygynous mating system?	Yes	Yes	Yes	Yes	Yes
Age 1st female reproduction	2	1	3	2	2
Age 1st male reproduction	4	3	2	3	3
Maximum individual age	12	12	12	9	12
Reproduction density dependent?	No	No	No	No	No
Sex ratio at birth	50:50	50:50	50:50	50:50	50:50
Maximum litter size	4	4	2	3	4
% females with litter/year	50	50	50	60	50
SD of above	20	5	10	10	5
% litter of size 1	32.5	17.5	50	20.0	10.0
% litter of size 2	40.0	50.0	50	50.0	50.0
% litter of size 3	20.0	30.0	—	30.0	30.0
% litter of size 4	7.5	2.5	—	0	10.0
Female mortality in year 1	26.5	20	0	20	20
SD in female mortality, year 1	6.625	2.0	4	10.0	5.0
Female mortality in year 2	10.1	—	0	10	20
SD in female mortality, year 2	—	—	—	—	—

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Table 14.1 (continued)

Model Inputs and Output	Originator of Variable Estimates for the VORTEX Simulation				
	Population Ecologist (Lacy)	State Field Biologist (Land)	Federal Field Biologist (Bass)	University Landscape Ecologist (Hector)	University Conservation Biologist (Maurer)
Male mortality in adults	21.7	20	66	20	20
SD in male mortality, adults	5.425	3.0	6	5.0	10
Number of catastrophe types	0	0	0	2	1
Probability for catastrophe 1	—	—	—	0.05	0.02
Probability for catastrophe 2	—	—	—	0.01	—
Reproduction rate for catastrophe 1*	—	—	—	0.80	0.95
Reproduction rate for catastrophe 2*	—	—	—	0.50	—
Survival for catastrophe 1*	—	—	—	0.80	0.95
Survival for catastrophe 2*	—	—	—	0.50	—
% of adult males breeding	100	50	100	50	40
Starting population size	50	50	6	60	70
Habitat carrying capacity	50	60	8	70	85
SD of above	0	5	2	10	5
Change in habitat	Lost	0	0	Lost	0
# of years of habitat lost	25	0	0	20	0
% habitat change per year	-1.0	0	0	-1.5	0
Will panthers be removed?	No	No	No	Yes	No
At what annual interval?	—	—	—	1	—
For how many years?	—	—	—	10	—
# males removed/year	—	—	—	1	—
# females removed/year	—	—	—	1	—
Population augmentation?	Yes	—	Yes	—	No
If yes, at what interval?	20 years	10 years	10 years	—	—
For how many years?	100	100	100	—	—
# males added per event	0	0	1	—	—
# females added per event	6	1	2	—	—
Outputs					
Expected heterozygosity	0.682	0.507	0.650	0.537	0.635
Number of extant alleles	6.38	4.58	3.89	3.58	4.68
Probability of persistence to 100 years over 500 iterations	0.998	1.00	0.0689	0.98	1.00
Mean final population	34.19	50.41	5.32	50.24	83.20
Median time to extinction	—	—	7.13 years	—	—

Note: SD = standard deviation.

*These values represent multipliers that reduce survival and reproduction due to catastrophe.

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Table 14.2 Comparison of Variables Used in the PVA Models

Model Inputs and Outputs	1989	1992	1992	1999
	Panther PVA	Panther PVA Consensus	Panther PVA Optimistic	Consensus Simulation
<i>Inputs</i>				
Inbreeding depression?	Yes	Yes	No	Yes
Lethal equivalents	3.4	3.0	0	3.14
% due to recessive lethals	0	0	0	50
Reproduction correlated with survival?	Yes	Yes	No	No
Polygamous mating system?	Yes	Yes	Yes	Yes
Age 1st female reproduction	3	2	2	2
Age 1st male reproduction	3	2	2	4
Maximum individual age	15	12	12	12
Reproduction density dependent?	No	No	No	No
Sex ratio at birth	50:50	50:50	50:50	50:50
Maximum litter size	5	3	3	4
% females with litter/year	50	50	50	50
SD of above	1	0	0	10
% litter of size 1	10	25	25	17.5
% litter of size 2	20	50	50	50.0
% litter of size 3	40	25	25	30.0
% litter of size 4	20	—	—	2.5
% litter of size 5	10	—	—	—
Female mortality in year 1	50	50	20	20
SD in female mortality, year 1	5	0	0	6
Female mortality in year 2	30	20	20	20
SD in female mortality, year 2	3	0	0	3
Female mortality in year 3	25	—	—	—
SD in female mortality, year 3	3	—	—	—
Female mortality in adults	25	20	20	17
SD in female mortality, adults	3	0	0	3
Male mortality in year 1	50	50	50	20
SD in male mortality, year 1	5	0	0	6
Male mortality in year 2	30	20	20	30
SD in male mortality, year 2	3	0	0	5
Male mortality in year 3	25	—	—	20
SD in male mortality, year 3	3	—	—	5
Male mortality in adults	25	20	20	15
SD in male mortality, adults	3	0	0	5
Number of catastrophes	2	0	0	1
Probability for catastrophe 1	0.01	—	—	0.5
Probability for catastrophe 2	0.02	—	—	—
Reproduction rate for catastrophe 1	—	—	—	0.95
Reproduction rate for catastrophe 2	—	—	—	—
Survival for catastrophe 1	—	—	—	—
Survival for catastrophe 2	—	—	—	0.95
% of adult males breeding	100	50	50	50
Starting population size	45	50	50	60
Habitat carrying capacity	45	50	50	70
SD of above	1	0	0	5

(continued)

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Table 14.4 Effects of Increasing Carrying Capacity on Genetic Heterozygosity after 100 Years, Using the Consensus VORTEX Simulation

Carrying Capacity	Predicted Heterozygosity (%) ^a
70	72.2
100	80.6
150	84.1
200	86.5
250	87.5
300	89.6
400	90.7
500	92.4

^a As percentage of initial value of H .

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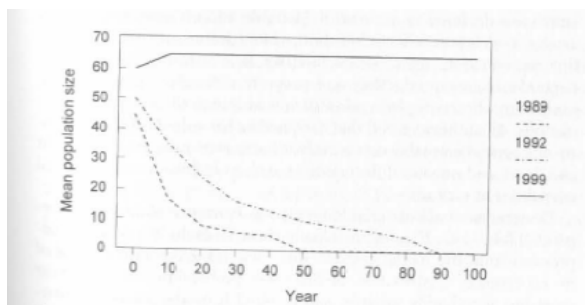


Fig. 14.3 Mean extant population sizes per ten-year intervals for Florida panther PVAs conducted in 1989 and 1992, and the consensus simulation from 1999.

~data

-time scale?

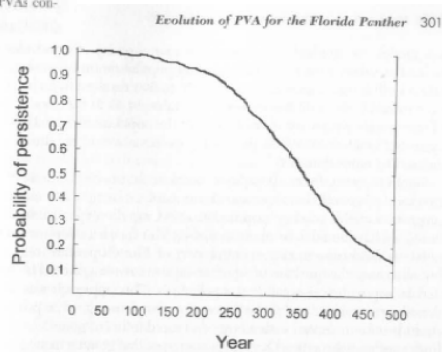


Fig. 14.4 Probability of persistence of the Florida panther based on the consensus simulation run for 500 years.

Last thoughts on PVA

PVA requires lots of **data**, which takes time, work, and money, whereas managers want answers (predictions about extinction) now. Few species will get through PVA. When should PVA be used and what type of PVA (how complex)?

Predictions from PVA can only be as good as the **data** that go into the analysis. We can only have **degrees of confidence** in the predictions from PVA. Populations should not be managed to their “minimum viable population” size.

One of the greatest strengths of PVA is the ability to play “**what if**” games with the model. That is, what if management were to increase patch sizes or connectivity? What if adult survival were improved?

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END

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