Lecture 6. Mechanisms and evidence for density dependence.

Reading:

Sinclair, A. R. E. 1989. Population regulation in animals. Pages 197-241 *in* J. M. Cherrett, ed. Ecological concepts. Blackwell Scientific Publishers, Oxford, United Kingdom.

Optional:

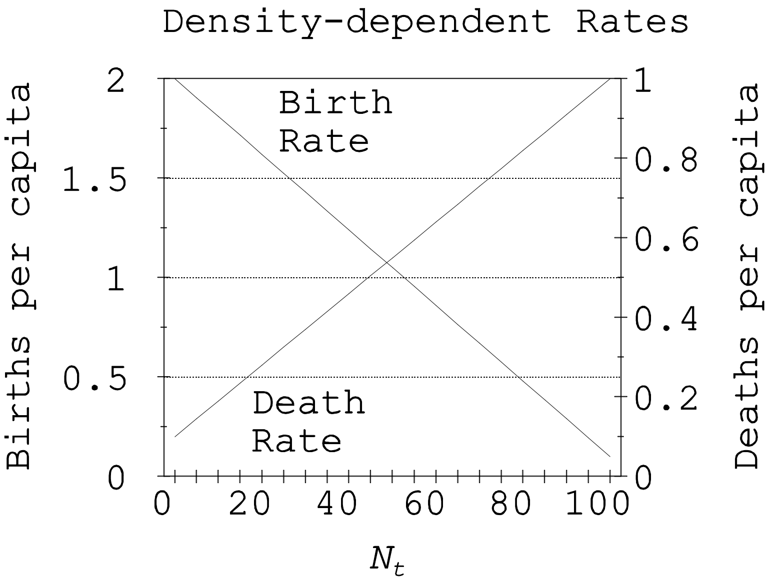
Hixon, M. A., S. W. Pacala, and S. A. Sandin. 2002. Population regulation: historical context and contemporary challenges of open vs. closed systems. Ecology 83:1490-1508.

"By definition, a population is regulated if it persists for many generations with flucuations bounded above zero with high probability. Regulation thus requires density-dependent negative feedback whereby the population has a propensity to increase when small and decrease when large." (Hixon et al. 2002). As stated by Hixon et al. (2002), population regulation requires density dependence. Density dependence need not be omnipresent to regulate a population (Wiens 1977), but is essential at some time and place for long-term persistence (Hassell 1986). What are the mechanisms that generate density dependence?

The possible causes of density dependence can be grouped into 2 categories: competition and predation. Competition for actually or potentially limiting resources (bottom-up regulation) is always density dependent by definition, be it via interference (a direct interaction) or exploitation (an indirect interaction) (Hixon et al. 2002). Predation (broadly including disease, parasitism, parasitoids, and herbivory) is not always density dependent. For predators to cause top-down regulation via density-dependent prey mortality, they must have a regulating total response, which is the combination of a numerical response in predator population size, a functional response in the per capita consumption rate, and other behavioral and developmental responses to changes in prey abundance (Hixon et al. 2002, and references therein).

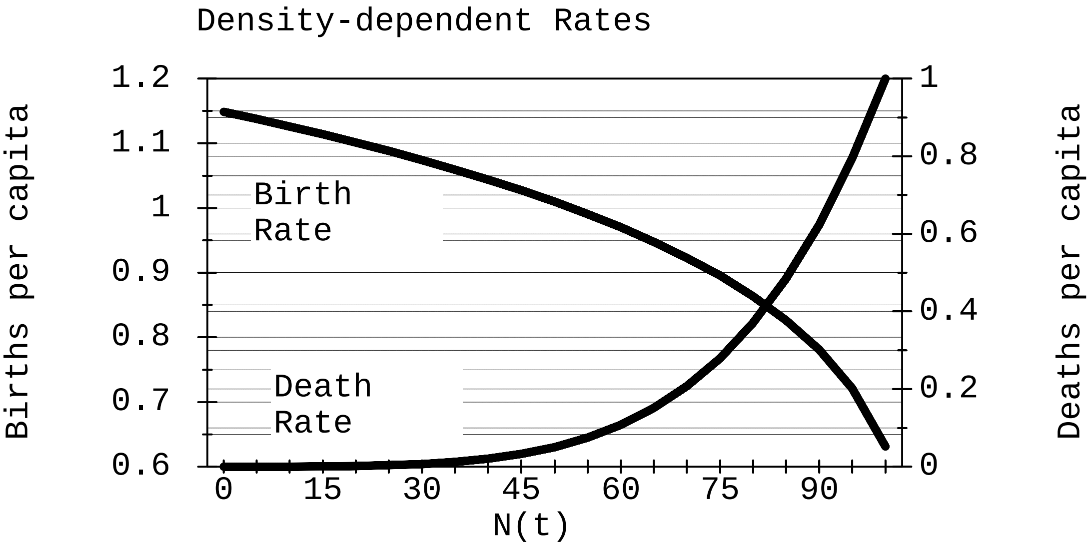
Mechanisms of density dependence

Must have the rates of births, deaths, immigration, or emigration change relative to population size.



Where the per capita birth rate equals the per capita death rate, the resulting population is at K. In the above graph, birth rate = $2 - 0.019N$, and death rate = $0.1 + 0.009N$. By equating the 2 equations and solving for N, we find $K = 67.8571$.

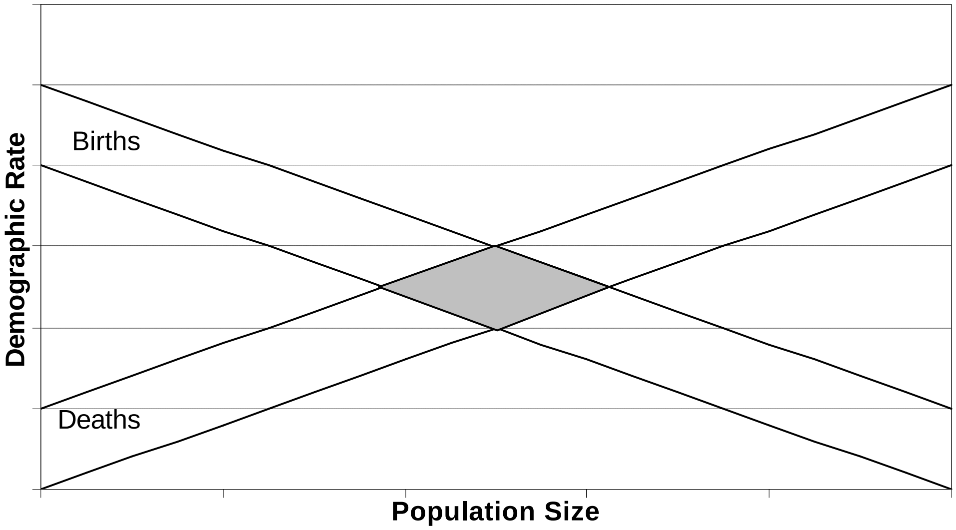
These curves do not have to be linear. See figure 7.1 of Sinclair (1989) for some more examples.



For the above graph, birth rate = $(2 - 0.019N)^{(1/5)}$ and death rate = $(0.1 + 0.009N)^5$.

These equations are equal at $K = 93.52678$.

Likewise, the curves do not have to have a single, constant *K*, if variation is allowed in the density-dependent functions. The idea of "density-vague" dynamics (Strong 1986) results in a cloud of points. Both the birth and death functions can exhibit a range of values, resulting in a range of values of *K*, illustrated by the shaded area in the following graph.

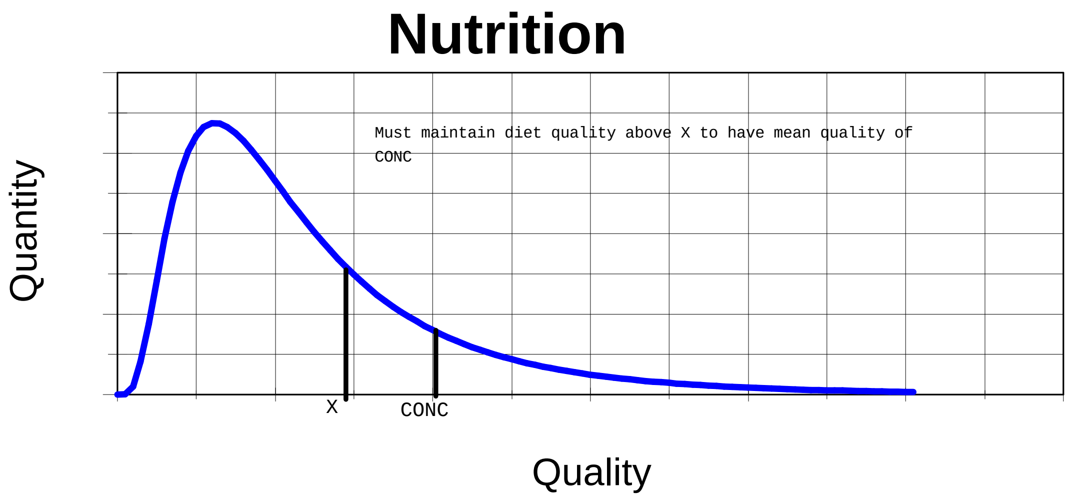


r vs. *K* selection. The theory of *r*- and *K*-selection was one of the first predictive models for life-history evolution, and has recently been discussed as a paradigm of ecology by Reznick et al. (2002). *r*- and *K*-selection has been displaced by the kinds of demographic models presented in this class. However, *r*- and *K*-selection still has its place, even in these models. The shape of the birth and death per capita curve reflects the strategy of the organism on the *r*- and *K*-selection continuum.

Indirect confrontations (scramble or exploitation competition).

Nutrition -- affects births, deaths.

(Clutton-Brock et al. 1985, Bartmann et al. 1992, McCullough 1979, Lack 1954). Hobbs and Swift (1985) defined quantity of forage as a function of its quality. Generally, a lot of poor quality forage exists relative to the amount of good quality forage. As a result, animals must maintain a diet of quality greater than *X* to be able to maintain a mean concentration in their diet of CONC.



Must maintain diet quality above X to have mean quality of CONC

- Social Behavior. Direct confrontations (contest or interference competition).
  - Territory size -- territory provides a link between resources and population processes.
  - Space available.
    - Plants (Harper 1977)
  - General Adaptive Syndrome, Calhoun's rats, H. Selye.
  - Chitty's genetic-feedback hypothesis
  - Wynne-Edwards (1962, 1986), group selection, advantage accruing to the individual.

- Other resources limiting than food
  - Nest sites
- Predation and Parasitism
  - More on this later! However, one example is Competition for shelter space causes density-dependent predation in damselfishes. Holbrook and Schmitt (2002) demonstrated that competition for shelter spaces caused more predation in damselfishes. These species shelter in branching corals or anemones, and when refuge spaces to protect them were filled with their intra-specific competitors, more mortality from predation was found.
- Andrewartha and Birch (1954) -- density independence

Biological techniques for detecting density dependence.

- Time series of observations with and without perturbation
  - No spatial control
  - Spatial control
- Quasi-experiments
- Experimental perturbations (Sinclair 1989)

Statistical techniques for detecting density dependence.

Tests of density dependence have been developed for a time series of population sizes, and as tests of a relationship between birth rates and/or death rates and population size.

Most tests of density dependence are set up with the null hypothesis of density independence, and the alternative hypothesis of density dependence. Failure to reject the null hypothesis does not constitute evidence of density independence in these cases, but only evidence to suggest the test lacked sample size, or the experimental variance was too high to reject the null hypothesis. In cases where the null hypothesis of density independence is not rejected, the investigator should report the confidence interval on the parameter being tested. This confidence interval will include the value of the parameter that suggests density independence, because the test failed to reject this hypothesis. However, if this confidence interval is narrow, evidence is provided that the true parameter value may not differ much from density independence. If the interval is wide, evidence is provided that the test lacked power to reject the null hypothesis, and hence little information is contained in the data relative to the hypothesis of density dependence.

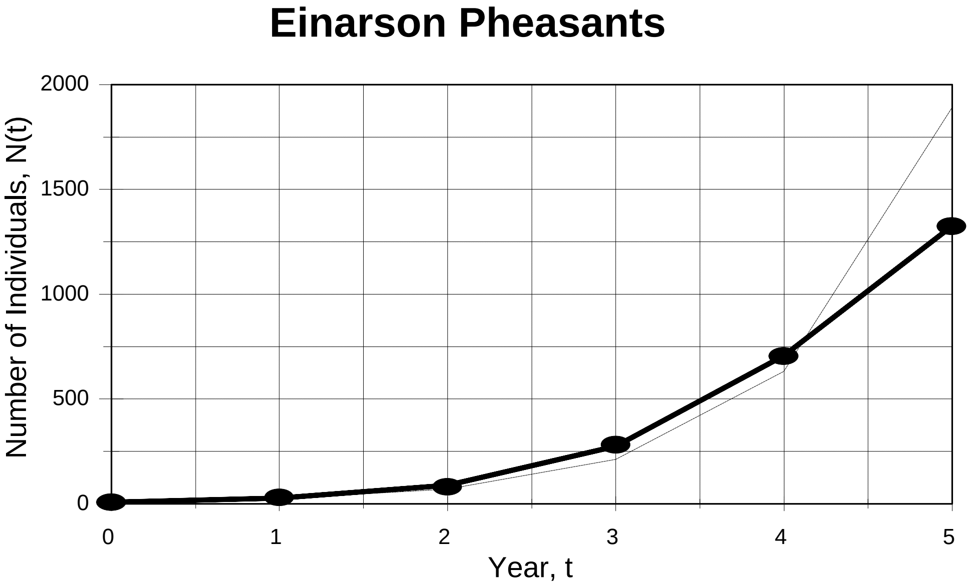
No perturbation of population size.

Procedures for testing density dependence in a time series of population sizes where the population density has not been manipulated have been developed in the entomology literature. Bulmer (1975) developed the first test. Pollard et al. (1987) extended the procedure, and Dennis and Taper (1994) developed the procedure further. All of these tests try to detect a “return” to K carrying capacity. That is, if the population is density dependent, then it should vary around K, and not grow indefinitely either direction from K.

These procedures have not been particularly useful in vertebrate research because the long time series of population sizes needed by these tests have not been available. Further, all the procedures suffer increased Type I errors when the population sizes are only estimated, and hence include sampling error (Dennis and Taper 1994, Shenk et al. 1998). Considerable controversy has developed over the usefulness of these tests (Wolda and Dennis 1993, Holyoak and Lawton 1993, Hanski et al. 1993, and Wolda et al. 1994). In general, their low power makes them ineffective even when the true population size is available (Murdoch 1994, Shenk et al. 1998).

Manipulation of population size.

When the population size has been manipulated to a level below K and observed as it increases, or alternatively, just manipulated to different levels and the amount of growth to the next year observed, a much more powerful approach to detecting density dependence is provided. Examples include the introduction of a species to an area previously uninhabited, or intensive harvest. As an example consider the growth of a ring-necked pheasant (*Phaisianus colchicus*) population introduced to an island off the coast of the state of Washington. Initially, in 1937, 2 cocks and 6 hens were introduced. The data on population sizes from 1937-42 are {8, 30, 81, 282, 705, 1325}.



Lack (1954) commented: "The figures suggest that the increase was slowing down and was about to cease, but at this point the island was occupied by the military and many of the birds were shot." The dashed line in the figure represents density-independent population growth, i.e., the model $N = N_0 e^{rt}$. The solid line represents density-dependent population growth, i.e., the differential equation model

$$N = \frac{K}{1 + \left(\frac{K}{N_0} - 1\right) e^{-rt}} .$$

The density-dependent model provides the best fit of the data, but then it should. It has 2 parameters, compared to only 1 parameter for the density-independent model. Is the improvement of the more complex model statistically significant, and hence, is density-dependence supported by these data? Was Lack correct in suggesting that growth had indeed slowed down? To answer these questions, we can either take a hypothesis testing paradigm and construct a statistical test of the null hypothesis that the 2 models fit equally well, or use information-theoretic methods and consider the weight of the evidence for each model. First, compute the sum of squared deviations for each model, or the SSE for each model:

Model	SSE
Dependence	142.79
Independence	59600.02

Clearly, the difference in SSE suggests a difference. To test the null hypothesis of no difference between the 2 models, construct an *F*-test as follows:

$$F_{df_I - df_D,\ df_D} = \frac{\dfrac{(SSE_I - SSE_D)}{df_I - df_D}}{\dfrac{SSE_D}{df_D}}$$

where SSE_D is the sum of squared errors for the density-dependent model, SSE_I is the sum of squared errors for the density-independent model, and df_D and df_I are the respective degrees of freedom of the 2 models. The F statistics equals 1249.2 with 1 and 3 degrees of freedom ($P < 0.001$). Thus, we conclude that Lack was correct.

Some readers may question the use of normal theory to develop this test. Note that this assumption can be changed by fitting a model with multiplicative, and hence lognormally distributed errors, or by randomization or permutation tests. For multiplicative errors, the *F*-test is 38.155 ($P = 0.009$), still supporting the conclusion that the population was exhibiting density dependence and that Lack was correct. However, the attempt here is to demonstrate the technique, not provide a full treatment of the statistical analyses.

For an information-theoretic approach, we compute the AIC$_c$ and Akaike weights for each model (Burnham and Anderson 2002). The AIC$_c$ and Akaike weight for each model can be computed directly from the SSE for the 2 models.

Model	SSE	AIC$_c$	Weight

Dependence	142.79	46.76	0.994
Independence	59600.02	56.93	0.006

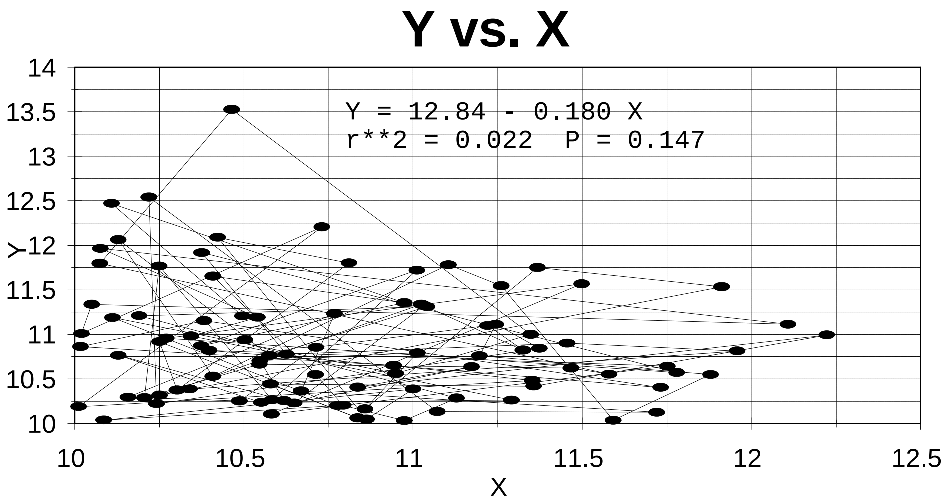
As with the hypothesis test paradigm, the hypothesis of density dependence is strongly supported.

Given that the population has been manipulated, the birth and death rates may also be observed, and tests of density dependence developed for changes in these rates as a function of population size. Such an approach was used by Bartmann et al. (1992) to test for changes in over-winter survival of mule deer fawns. In their experiment, fawn survival was estimated by radio-tracking animals, and density was known because a set number of animals were stocked in each enclosure. Often, however, attempts are made to estimate per capita recruitment to the next time step from a sequence of observed population estimates. As discussed next, such an approach must be implemented carefully.

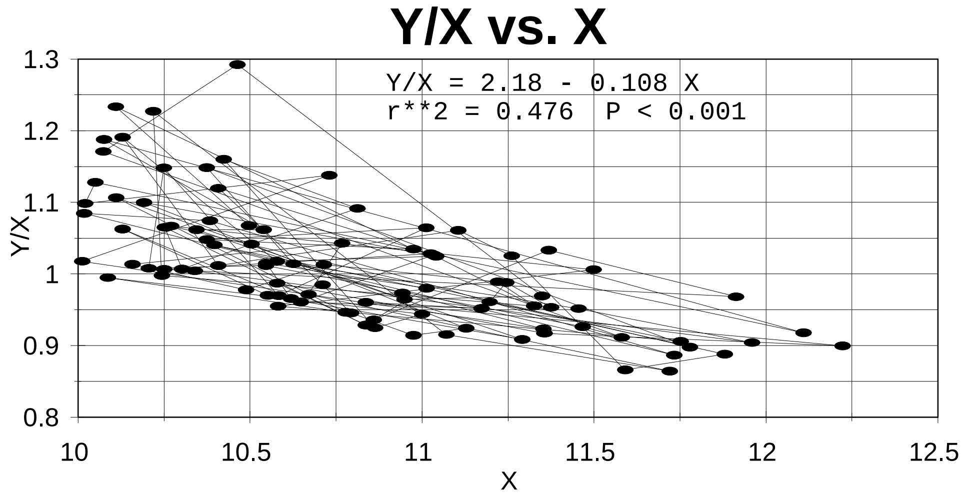
Problem of induced correlation.

$(N_{t+1} - N_t)/N_t$ regressed against N_t

N_{t+1}/N_t regressed against N_t even worse. Eberhardt (1970) demonstrated that a correlation of r=-0.7 is expected. In the following graph, Y and X are random normal variables with mean 10 and standard deviation 1. As the first graph demonstrates, the 2 variables were independent, i.e., no relationship exists.



As the following graph shows, the regression of Y/X versus X is significant, and the correlation is close to the 0.7 that Eberhardt (1970) suggested it should be. That is, $0.475^{1/2} = 0.689$.



Procedures to test for a relationship between recruitment (including per capita birth or death rates) and population size have been extensively used to test for density dependence (Tanner 1966, McCullough 1979). The approach is to regress population growth rate against population size. If population growth is density independent, the expected slope of the regression is 0. If density dependent growth is occurring, the slope of the regression should be negative, with a negative correlation. However, as first pointed out by Eberhardt (1970), population growth rate (recruitment) must be estimated independently of population size. When population growth rate is estimated from the time series of population sizes as

$$\hat{R}_t = \frac{N_{t+1} - N_t}{N_t}$$

and $\hat{R}_t$ is regressed against N_t, a correlation is induced because N_t occurs on both sides of the regression. Eberhardt (1970) pointed out that correlations of about -0.7 are expected for sequences of random numbers when tested with the regression procedure used by Tanner (1966).

Review of evidence by major animal groups: good review by Sinclair (1989).

- 18th Century Humans (Matessi and Menozzi 1979)
  - Birth rates did not depend on population size, but did increase from mountains to hills to plains, whereas death rates increase with population size, and also increase from mountains to hills to plains. Consequently, population growth rates were density dependent, with spatial variation.
- Large Herbivores — nutrition (Fowler 1987). Eberhardt (2002) suggested a

paradigm for population regulation of long-lived vertebrates. A sequence of changes in vital rates observed as populations approach maximal levels has been used as the basis for a “paradigm” for population analysis. Past work indicates that early survival (survival of young animals) decreases first, followed by lower reproductive rates; ultimately, adult female survival may decrease. Sensitivity of population growth rates as measured by partial derivatives of Leslie matrix models follow the same sequence, suggesting that population regulation follows this sequence, and implying some evolutionary significance in the sequence.

Large Carnivores — social (territory size)

Raptors (Newton 1991)
- Stability of breeding population
- Existence of surplus adult
- Re-establishment of populations to same level prior to removal
- Regular spacing of nests where nest sites are not limited

Small Mammals
- Meadow voles — strong density-dependent regulation via birth rates (Ostfeld et al. 1993). They suggest cycles not caused by lagged effects of resource exploitation.

Grouse
- Red grouse — cycles not stopped based on maternal nutrition, a version of Chitty’s genetic hypothesis, host-parasite (caecal threadworm), or predator-prey relationships (Moss et al. 1996). They suggest the changing age structure in the population as one explanation.

Insects

Why is the evidence so weak (Sinclair 1989)?

Discussion: Bring to class an example of a good test of density-dependence, and be prepared to discuss what critical information is lacking to produce a valid population model that incorporates density-dependence.

Literature Cited

Andrewartha, H. G., and L. C. Birch. 1954. The distribution and abundance of animals. University Chicago Press, Chicago, Illinois, USA. 782 pp.

Bartmann, R. M., G. C. White, and L. H. Carpenter. 1992. Compensatory mortality in a Colorado mule deer population. Wildlife Monograph 121:1-39.

Bulmer, M. G. 1975. The statistical analysis of density dependence. Biometrica 31:901-911.

Burnham, K. P., and D. R. Anderson. 2002. Model selection and multimodel inference: a practical information theoretic approach. Second Edition. Springer Verlag, New York, New York, USA.

Christian, J. J., and D. E. Davis. 1964, Endocrines, behavior and population. Science 146:1550-1560.

Clutton-Brock, T. H., M. Major, and F. E. Guinness. 1985. Population regulation in male and female red deer. Journal of Animal Ecology 54:831-846.

Dennis, B., and M. Taper. 1994. Density dependence in time series observations of natural populations: detecting stability in stochastic systems. Ecological Monograph 64:205-224.

Eberhardt, L. L. 2002. A paradigm for population analysis of long-lived vertebrates. Ecology 83:2841-2854.

Eberhardt, L. L. 1970. Correlation, regression, and density dependence. Ecology 51:306-310.

Fowler, C. R. 1987. Overview of density dependence in populations of large mammals. Pages 401-441 *in* H. H. Genownys, ed. Current Mammalogy, volume 1. Plenum, New York, New York, USA.

Hanski, I., I. Woiwod, and J. Perry. 1993. Density dependence, population persistence, and largely futile arguments. Oecologia 95:595-598.

Harper, J. L. 1977. Population biology of plants. Academic Press, New York, New York, USA. 892 pp.

Hassell, M. P. 1986. Detecting density dependence. Trends in Ecology and Evolution 1:90-93.

Hixon, M. A., S. W. Pacala, and S. A. Sandin. 2002. Population regulation: historical context and contemporary challenges of open vs. closed systems. Ecology 83:1490-1508.

Hobbs, N. T., and D. M. Swift. 1985. Estimates of habitat carrying capacity incorporating explicit nutritional constraints. Journal of Wildlife Management 49:814-822.

Holbrook, S. J., and R. J. Schmitt. 2002. Competition for shelter space causes density-dependent predation mortality in damselfishes. Ecology 83:2855-2868.

Holyoak, M., and J. H. Lawton. Comment arising from a paper by Wolda and Dennis: using and interpreting the results of tests for density dependence. Oecologia 95:592-594.

Lack, D. L. 1954. The natural regulation of animal numbers. Clarendon Press, Oxford, United Kingdom. 343 pp.

Matessi, C. and P. Menozzi. 1979. Environment, population size and vital statistics: an analysis of demographic data from 18th century villages in the province of Reggio Emilia (Italy). Ecology 60:486-493.

McCullough, D. R. 1979. The George Reserve deer herd. University Michigan Press, Ann Arbor, Michigan, USA. 271 pp.

Moss, R., A. Watson, and R. Parr. 1996. Experimental prevention of a population cycle in red grouse. Ecology 77:1512-1530.

Murdoch, W. W. 1994. Population regulation in theory and practice. Ecology 75:271-287.

Newton, I. 1991. Population limitation in birds of prey: a comparative approach. Pages 3-21 *in* C. M. Perrins, J-D. Lebreton, and G. J. M. Hirons, eds. Bird Population Studies, Oxford, New York, New York, USA.

Ostfeld, R. S., C. D. Canham, and S. R. Pugh. 1993. Intrinsic density-dependent regulation of vole populations. Nature 366:259-261.

Pollard, E., K. H. Lakhani, and P. Rothery. 1987. The detection of density dependence from a series of annual censuses. Ecology 68:2046-2055.

Resnick, D., M. J. Bryant, and F. Bashey. 2002. *r*- and *K*-selection revisited: the role of population regulation in life-history evolution. Ecology 83:1509-1520.

Shenk, T. M., G. C. White, and K. P. Burnham. 1998. Sampling-variance effects on detecting density dependence from temporal trends in natural populations. Ecological Monographs 86:445-463.

Sinclair, A. R. E. 1989. Population regulation in animals. Pages 197-241 *in* J. M. Cherrett, ed. Ecological concepts. Blackwell Scientific Publishers, Oxford, United Kingdom.

Strong, D. R. 1986. Density-vague population change. Trends in Ecology and Evolution 1:39-42.

Tanner, J. T. 1966. Effects of population density on growth rates of animal populations. Ecology 47:733-745.

Wiens, J. A. 1977. On competition and variable environments. American Scientist 65:590-597.

Wolda, H., and B. Dennis. 1993. Density dependence tests, are they? Oecologia 95:581-591.

Wolda, H., B. Dennis, and M. Taper. 1993. Density dependence tests, and largely futile comments: Answers to Holyoak and Lawton (1993) and Hanski, Woiwod and Perry (1993). Oecologia 98:229-234.

Wynne-Edwards, V. C. 1962. Animal dispersion in relation to social behavior. Oliver and Boyd Limited, Edinburgh, Scotland.

Wynne-Edwards, V. C. 1986. Evolution through group selection. Blackwell Scientific, Palto Alto, California, USA. 386 pp.