pollen (with tectal perforations) to semitectate pollen (with a prominent reticulum formed by enlarged tectal perforations) (*15*).

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References and Notes

1. J. A. Doyle, *J. Arnold Arbor. Harv. Univ.* **50**, 1 (1969); ——— and L. J. Hickey, in *Origin and Early Evolution of Angiosperms*, C. B. Beck, Ed. (Columbia Univ. Press, New York, 1976), p. 139.
2. D. L. Dilcher, W. L. Crepet, C. D. Beeker, H. C. Reynolds, *Science* **191**, 854 (1976).
3. R. F. Thorne, *Evol. Biol.* **9**, 35 (1976); R. Dahlgren, *Bot. J. Linn. Soc.* **80**, 91 (1980); A. Takhtajan, *Bot. Rev.* **46**, 225 (1980); A. Cronquist, *An Integrated System of Classification of Flowering Plants* (Columbia Univ. Press, New York, 1981).
4. L. J. Hickey and J. A. Doyle, *Bot. Rev.* **43**, 3 (1977).
5. J. Muller, *ibid.* **47**, 1 (1981).
6. J. W. Walker, paper presented at 5th International Palynological Conference, Cambridge University, Cambridge, England, June 1980; J. W. Walker and A. G. Walker, *Botanical Society of America Miscellaneous Publication 162* (1982) (abstr.), p. 66; *Ann. Mo. Bot. Gard.*, in press.
7. I. W. Bailey and C. G. Nast, *J. Arnold Arbor. Harv. Univ.* **26**, 37 (1945).
8. E. Aharoni, *Isr. J. Earth Sci.* **13**, 63 (1964).
9. M. Greenberg, *Geol. Surv. Isr. Stratigr. Sect.* **5**, 1 (1968).
10. M. Avnimelech, A. Parness, Z. Reiss, *J. Paleontol.* **28**, 835 (1954); Z. Lewy and M. Raab, *Ann. Mus. Hist. Nat. Nice* **4**, 1 (1976).
11. J. Praglowski, *World Pollen and Spore Flora 8* (Almqvist & Wiksell, Stockholm, 1979).
12. G. Erdtman, *Pollen Morphology and Plant Taxonomy, Angiosperms* (Almqvist & Wiksell, Stockholm, 1952).
13. H. A. Leffingwell, D. A. Larson, M. J. Valencia, *Bull. Can. Petrol. Geol.* **18**, 238 (1970).
14. E. A. Stanley, *Mar. Geol.* **4**, 397 (1966).
15. J. W. Walker, *Am. J. Bot.* **61**, 891 (1974).
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When Is an Island Community in Equilibrium?

Abstract. *To determine whether the number of species in a biota is in equilibrium requires a colonization model. In a simple Markov model, each species' extinction and immigration probabilities are estimated independently from available data. For one inland and two island avifaunas, a simulation with these probabilities shows that the trajectories of species richness through time do not manifest the regulatory tendencies expected if species interactions cause species richness to be continuously redressed toward an equilibrium.*

The equilibrium model of island biogeography (*1*) received instant approbation and was quickly applied to many taxa of oceanic and habitat islands (*2*). Early objections were dismissed (*3*), but reconsideration of assumptions and predictions of the model has led to a more tempered judgment of its applicability (*4, 5*).

A persistent problem with the model is subjective assessment of its most fundamental contention—that species number tends toward an equilibrium. The avifauna of the Farne Islands (Fig. 1) seems equilibrial to some (*6*), though number of species varies more than 100 percent. By contrast, the passerine birds of Skokholm Island (Fig. 1) are judged nonequilibrial (*5*), though to me the trajectory of species number does not seem qualitatively different from that of the Farne Islands. How much variation in species number is permitted in an equilibrium biota appears to be arbitrary (*5*). A coefficient of variation less than .20 (*6*) or less than .05 (*7*) may be demanded. Although variation in species number of 16 percent or less may be classed as equilibrial (*8*), the Farne Islands variation far exceeds this percentage.

A colonization model is required before equilibrium can be assessed, just as a null model is needed generally to interpret ecological data (*9*). The model may predict a specific equilibrium and a certain amount of temporal variation about it; the trajectory of species numbers may then be compared to predictions. Or the model may simply state certain characteristics that an equilibrated process should have that an unequilibrated process should lack. In either event, criteria for judging whether given data are equilibrial must be clearly stated; the hypothesis of equilibration must be falsifiable or the entire equilibrium concept will degenerate into a truism.

The original equilibrium model (*1*) connotes a regulation of species number by interactions among species, such that the presence of species A modifies the probability that species B will suffer extinction (*10*). Though for mutualistic or commensal species pairs this extinction probability may be lowered, advocates of the model argue that on average for all species pairs, one species' presence raises the extinction probability of the other (*11*). Consequently, high species numbers will, on average, increase extinction probabilities for all species, and species numbers will fall. Low species numbers will lower extinction probabilities, and species number will be redressed upward. Higher order interactions such as diffuse competition can modify the details of any particular scheme, but not the general expectation of regulation: high species numbers are followed by increased extinctions per species, and low species numbers by low extinctions per species.

One cannot simply correlate the extinction rates per species with number of species present, since a positive correlation would be expected as an artifact (*10*). In a bounded sequence of random, independent numbers, S_i, one would expect a positive correlation between S_i and $(S_i - S_{i+1})/S_i$. One might, however, be able to perceive regulation (as opposed to a null hypothesis of no regulation) by a version of a runs test (*12*) that allows for the event of "no change."

One appropriate model to test against the regulatory equilibrium model is a Markov model, recently termed the "molecular theory of island biogeography" (*13*). If every species in a pool of size p has constant immigration and extinction probabilities (i_k and e_k), an equilibrium number of species ultimately obtains (*14*):

$$S_{eq} = \sum_{k=1}^{p} \frac{i_k}{i_k + e_k} \quad (1)$$

The colonization curve is a sum of independent Markov processes, where each species has constant transition probabilities of absent to present (i_k), present to absent (e_k), absent to absent ($1 - i_k$), and present to present ($1 - e_k$). To take this model further one must know the distributions of i_k and e_k (*15*), but the only species pool for which these distributions are available is Florida Keys mangrove insects (*16*). Since the data on birds of Skokholm and the Farne Islands were tabulated species by species (*17*), they can be used to estimate Markov transition probabilities. For any species k, the fraction of presences followed by absences is an estimate of e_k, and the fraction of absences followed by presences is an estimate of i_k. For example, if species k were absent during 20 censuses, and for 10 of these it was present at the next census, one would estimate i_k as $10/20 = 0.5$. In addition to the two insular avifaunas, I also tabulated transition probabilities for birds of Eastern Wood (*18*), a 16-ha wood in 112-ha Bookham Common, an inland "habitat island" in Surrey, England (*19*).

For each avifauna, I simulated colonization. Each simulation began with the

species present at the beginning of the study. Each year I determined for each species whether it remained present, remained absent, immigrated, or went extinct by noting its status the year before and a uniform-random number that was generated on the interval [0, 1]. This number was compared to the transition probabilities. If the species was present and the random number was less than e_k, extinction occurred; if it was greater than or equal to e_k, it remained present. If the species was absent and the random number was less than i_k, the species immigrated; otherwise it remained absent. Each year the number of species present was tallied. Observed colonization curves (Fig. 1) were compared with 100 simulated curves in three ways: variance in species number, number of runs up and down, and number of "no changes." If species interactions generated a regulated species number equilibrium, then observed variance should be smaller than that produced by the Markov model. If there were more "no changes" in the observed data than in the Markov simulations, one would suspect tight regulation. Finally, a "run" is defined as a sequence of species counts that has at least one increase and is nondecreasing or has at least one decrease and is nonincreasing; it may include one or more "no changes." A regulated equilibrium ought to have more runs than the Markov model would predict, since an increase should be quickly redressed by a decrease, and vice versa. Number of runs and number of "no changes' might be partially complementary, since a larger number of "no changes" could contribute to a lower number of runs, and vice versa.

The observed variance, number of runs, and number of "no changes" for the three avifaunas are shown in Table 1 together with the percentages of 100 Markov simulations for which variance was less than or equal to that observed, and numbers of runs and "no changes" were greater than or equal to those observed. These were the distributional tails for which a system undergoing interactive species number regulation, as opposed to just a sum of independent Markov processes, should produce extreme values. There should be few simulations with variance as small as observed and number of runs and "no changes" as large as observed if species number is regulated. In fact, most values are in the wrong tail for the regulation hypothesis, though only species number variance on Skokholm is very extreme.

That the statistical tests all reject the interactive regulatory hypothesis in favor of the Markov hypothesis need not mean that the Markov hypothesis is correct. But the fact that observed and expected mean number of species are similar (Table 1) suggests that the Markov model may be on the right track. One can think of innumerable ways to embellish the model and make it more realistic, but realism is not necessarily a virtue, especially if the number of parameters required to render the model realistic also renders it unfalsifiable (*3*). On biological grounds, one objectionable assumption of the Markov model is that transition probabilities do not change, but probably the weakest aspect of the Markov model, and in fact of the whole gamut of versions of the equilibrium model, is that population sizes are not taken into account (*20*). In two recent studies of bird communities (*21*) population sizes of all species have been examined, but in neither were the data related to extinction and immigration probabilities.

Table 1. Observed mean number of species, variance, number of runs, and number of "no changes" for Skokholm, the Farne Islands, and Eastern Wood, along with percentages of 100 Markov simulations (that is, the percentages in the tail) with equal or smaller variance, equal or greater number of runs, and equal or greater number of "no changes." Expected mean is calculated from Eq. 1.

Habitat	Observed mean	Expected mean	Observed variance	Tail (%)	Observed runs (*N*)	Tail (%)	Observed no changes (*N*)	Tail (%)
Skokholm	7.088	8.403*	2.375	98	7	91	19	44
Farne	5.862	5.873	1.360	50	13	54	8	83
Eastern Wood	31.654	32.354	5.611	91	13	90	4	58

*Arbitrarily assigned e_k = .5 to one species and i_k = .01 for one species (*19*).

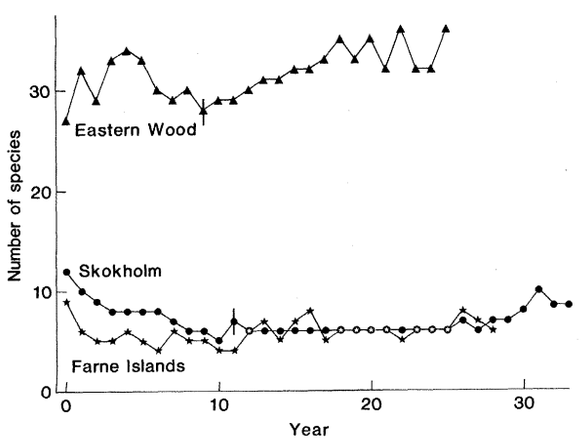

Fig. 1. Colonization curves for passerine birds of Skokholm and land birds of the Farne Islands and Eastern Wood. Vertical lines indicate one or more years when censuses were not performed.

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References and Notes

1. R. H. MacArthur and E. O. Wilson, *Evolution* **17**, 373 (1963); _____, *The Theory of Island Biogeography* (Princeton Univ. Press, Princeton, N.J., 1967).
2. D. Simberloff, *Annu. Rev. Ecol. Syst.* **5**, 161 (1974).
3. _____, in *Perspectives in Ornithology*, A. Brush and G. Clark, Eds. (Cambridge Univ. Press, New York, in press).
4. J. F. Lynch and N. K. Johnson, *Condor* **76**, 370 (1974); D. Simberloff, *Science* **194**, 572 (1976); _____, *Ekologiya* (Moscow) **4**, 3 (1982); P. Slud, *Smithson. Contrib. Zool.* **212**, 1 (1976); I. Abbott, *Adv. Ecol. Res.* **11**, 329 (1980); F. S. Gilbert, *J. Biogeogr.* **7**, 209 (1980).
5. I. Abbott and P. R. Grant, *Am. Nat.* **110**, 507 (1976).
6. J. M. Diamond and R. M. May. *Science* **197**, 266 (1977).
7. I. Abbott and R. Black. *J. Biogeogr.* **7**, 399 (1980).
8. J. M. Diamond, *Proc. Natl. Acad. Sci. U.S.A.* **64**, 57 (1969).
9. D. R. Strong, *Synthèse* **43**, 271 (1980).
10. D. Simberloff, in *Evolution, Time and Space: the Emergence of the Biosphere*, R. W. Sims, J. H. Price, P. E. S. Whalley, Eds. (Academic Press, London, 1983), pp. 57–83.
11. See R. H. MacArthur, in *Geographical Ecology* (Harper & Row, New York, 1972); E. C. Pielou, in *Biogeography* (Wiley, New York, 1979); B. A. Wilcox, in *Conservation Biology: An Evolutionary-Ecological Perspective*, M. E. Soulé and B. A. Wilcox, Eds. (Sinauer, Sunderland, Mass., 1980), pp. 95–117; and M. Williamson, in *Island Populations* (Oxford University Press, Oxford, 1981).
12. For a description of a runs test, see F. S. Swed and C. Eisenhart, *Ann. Math. Stat.* **14**, 66 (1943).
13. The model, introduced by P. W. Holland [*Memo. Dep. Stat. Harv. Univ. NS-107* (1968)] and D. Simberloff [*Ecology* **50**, 296 (1969)] was termed "molecular" by M. E. Gilpin and J. M. Diamond [*Proc. Nat. Acad. Sci. U.S.A.* **78**, 393 (1981)].
14. See Simberloff in (*13*).
15. See Simberloff in (*13*) and MacArthur in (*11*).
16. D. Simberloff, in *Insect Life History Patterns: Habitat and Geographic Variation*, R. F. Denno and H. Dingle, Eds. (Springer, New York, 1981), pp. 195–206.
17. The Skokholm data are from D. Lack [*J. Anim. Ecol.* **38**, 211 (1969)] and the Farne Islands data are from Diamond and May (*6*).
18. See Williamson in (*11*).
19. The following conventions were established. For Skokholm, a census was not performed in

years 13 through 18. The simulation omitted those years and began with the observed species present when the census was resumed. For Eastern Wood the ninth year was treated similarly. The robin on Skokholm was present only the 12th year (just before the missing censuses) and therefore had no estimated extinction probability. This was arbitrarily reset to .5. The starling on Skokholm was absent before the hiatus but always present afterward. Its estimated immigration probability of 0 was arbitrarily reset to .01. A few species whose presence was questioned in the Skokholm censuses were changed to present, and the dunnock, which was not censused the first year to Eastern Wood but was present, usually with several breeding pairs during 23 of the 25 remaining years, was assumed to be present initially. Simulations were run with all of these conventions modified in a variety of biologically reasonable ways, with no substantive change in the results.

20. Y. Haila and O. Järvinen, *Stud. Avian Biol.* **6**, 559 (1981).
21. F. C. James and W. J. Boecklen, in preparation; K. Smith, *Ecology* **63**, 952 (1982).

22. I am indebted to L. G. Abele, W. J. Boecklen, J. Farr, N. Gotelli, F. C. James, M. Keough, and D. R. Strong for comments on this manuscript and particularly to C. E. McCulloch and D. A. Meeter for statistical discussion.

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Mutations Affecting Programmed Cell Deaths in the Nematode *Caenorhabditis elegans*

Abstract. *Mutations in two nonessential genes specifically block the phagocytosis of cells programmed to die during development. With few exceptions, these cells still die, suggesting that, in nematodes, engulfment is not necessary for most programmed deaths. Instead, these deaths appear to occur by cell suicide.*

During development, the nematode *Caenorhabditis elegans* follows an essentially invariant pattern of cell divisions which produces cells of rigidly specified fates (*1–3*). One fate is for cells to die, usually within 1 to 2 hours of their birth, in mitosis. For example, of 671 cells produced during embryogenesis of the hermaphrodite, 113 cells die before hatching. An additional 18 cells die during larval growth as the number of somatic nuclei increases from 558 to an eventual 959. Normally, these cells are engulfed and degraded by neighboring cells at the time of their deaths (*1, 4*). Mutations in two genes, described below, prevent the elimination of dead cells by blocking their phagocytosis. With certain exceptions, cells die at their normal times in these mutants, suggesting that most programmed deaths occur by cell suicide, not from phagocytosis. Neither mutation disrupts development.

Cell divisions and deaths can be observed in living nematodes by Nomarski differential interference contrast microscopy (*2*). Overt signs of cell death, a darkening of the cytoplasm and nucleoplasm in electron micrographs or a corresponding increase in refractility in Nomarski optics, appear 30 minutes or more after cytokinesis (*1–4*). A period of high, uniform refractility, during which the entire cell has the appearance of a flat raised disk in Nomarski optical section, persists for 10 to 30 minutes. Engulfed cells then lose refractility and shrink, eventually disappearing. The entire process, from the first increase in refractility to the disappearance of the cell, takes about an hour. A sequence of Nomarski photographs showing the death of a presumptive ventral cord motor neuron is given in Sulston and Horvitz (*2*). A corresponding series of electron micrographs is shown in (*4*).

Mutations were induced in hermaphrodites by exposure to ethyl methanesulfonate (*5*). The F_2 progeny were screened under Nomarski optics for abnormal persistence of embryonic cell deaths. Eight independent strains were obtained in which dead cells were not resorbed. We designated these *ced* mutants, mnemonic for programmed *c*ell *d*eath. All eight mutations were recessive and, together, defined two complementation groups, *ced-1 (el735, el754, el797, el798, el799, el801,* and *el814)* and *ced-2 (el752).*

Mutations *el735 (ced-1)* and *el752 (ced-2)* were mapped to linkage groups I and IV, respectively. Recombination frequencies were determined from cis double heterozygotes. The distances, given in recombinant chromosomes per total chromosomes examined, were *dpy-5 (e61) ced-1,* 2/24; *unc-75 (e950) ced-1,* 0/24; and *ced-2 dpy-13(el84),* 5/24. Trans three-factor crosses gave the following gene orders: *dpy-5*[*unc-13(e51),ced-1*], 12 recombinants; *unc-75*(1/21)*ced-1*(20/21) *lev-11(x12);* [*ced-2,unc-17(el13)*]*dpy-13,* 5 recombinants; and *dpy-9(el2)*(13/23) *ced-2*(10/23)*lin-1(el275).*

The phenotypes of *ced-1 (el735), ced-2 (el752),* and the *ced-1, ced-2* double mutant are indistinguishable by Nomarski and electron microscopy and by Feulgen staining, suggesting that *ced-1* and *ced-2* mutations affect closely related steps in the removal of dead cells, though neither gene product can substitute for the other.

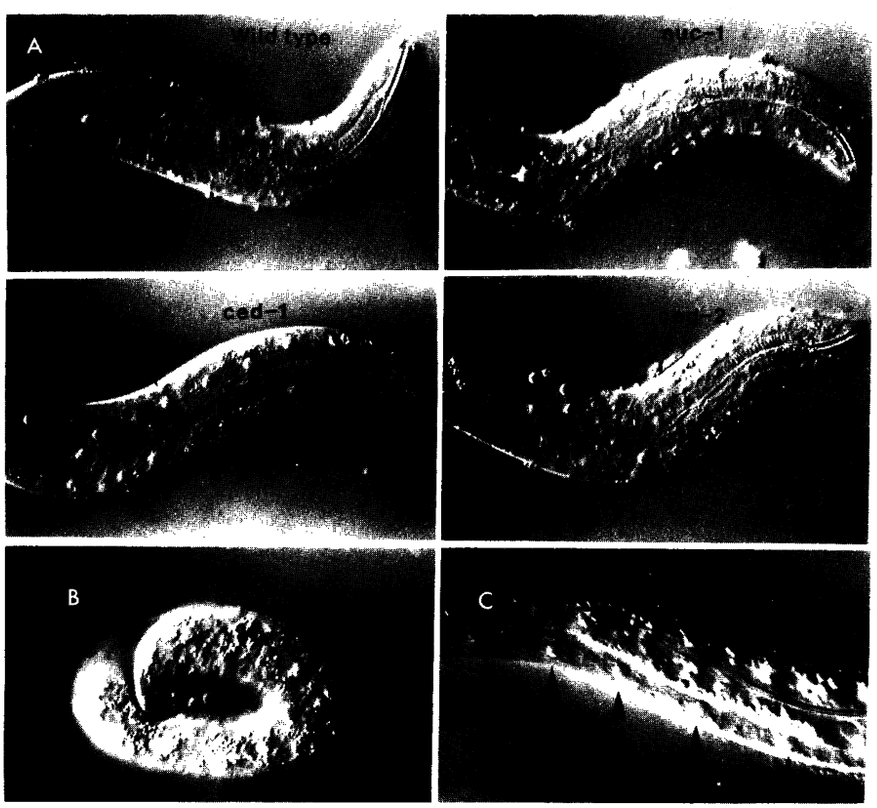

Fig. 1. (A) Nomarski photographs of newly hatched wild-type (N2), *nuc-1 (el392), ced-1 (el735),* and *ced-2 (el752)* larvae showing normal cell nuclei and refractile cell deaths (arrows) in the head. (B) Embryonated egg *(el735)* with two dead cells shed into the egg fluid (arrow). (C) Posterior ventral nerve cord of 15-hour first-stage larva *(el752).* Arrows mark (from right to left) refractile dying cells P9.aap, P10.aap, P11.aap, and P12.aap [for nomenclature, see (*2*)]. Scale bar, 20 μm.