UNITED NATIONS EDUCATIONAL, SCIENTIFIC AND CULTURAL ORGANIZATION
INTERNATIONAL ATOMIC ENERGY AGENCY
INTERNATIONAL CENTRE FOR THEORETICAL PHYSICS
I.C.T.P., P.O. BOX 586, 34100 TRIESTE, ITALY, CABLE: CENTRATOM TRIESTE

SMR.940 - 38

THIRD AUTUMN WORKSHOP ON MATHEMATICAL ECOLOGY

(14 October - 1 November 1996)

"Allometry and simple epidemic models for microparasites"

Andrew P. Dobson
Department of Ecology and Evolutionary Biology
Princeton University
Princeton, NJ 08544-1003
U.S.A.

and

Giulio A. de Leo
Dipartimento di Elettronica e Informazione
Politecnico di Milano
20133 Milano
Italy

These are preliminary lecture notes, intended only for distribution to participants.

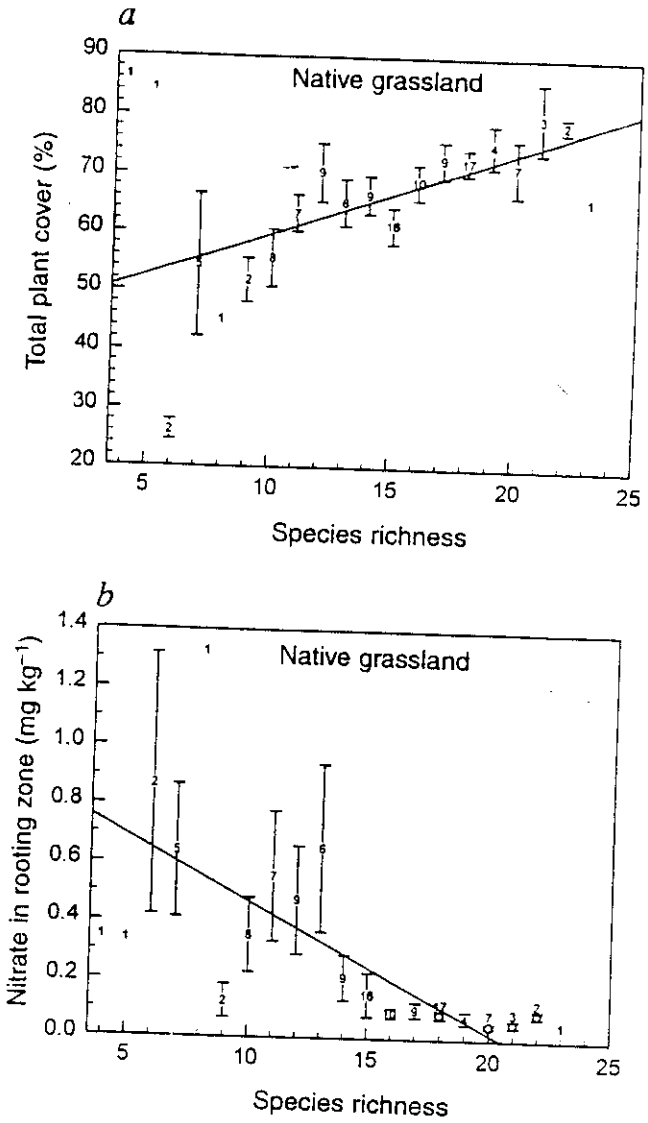

FIG. 2 *a*, Mean ± s.e.m. of total plant cover (fitted curve: $y = 46.1 + 1.40x$, $R^2 = 0.16$, $n = 120$, $P < 0.001$) and *b*, soil nitrate in the rooting zone (fitted curve: $y = 0.92 - 0.045x$, $R^2 = 0.22$, $n = 120$, $P < 0.001$) both plotted against plant-species richness in 120 native grassland plots. Figures indicate the number of plots with a given level of plant species richness. Simple curvilinear fits were no better than lines.
METHODS. Native, undisturbed grassland in Field D (ref. 24) was sampled for plant species cover and species richness (number of vascular plant species per plot) in 120 plots, each 1 m × 1 m, with a block of 4 such plots in each of 30 localities. Four 0–20 cm soil cores per plot were extracted for measuring NO_3^- and NH_4^+ levels.

work is needed to determine how interspecific morphological and physiological differences[25–27] influence the dependence of ecosystem functioning on biodiversity in this and other ecosystems.

It is known that soil fertility and productivity influence diversity[28,29]. Our results demonstrate that the converse is also true: in our experiment using initially homogeneous soils, plant diversity had a significant effect on productivity, nutrient use, and nutrient retention. The establishment and functioning of these grassland ecosystems depended on their species richness, with more diverse ecosystems being more productive and having lower nutrient losses than less diverse ecosystems. This extends earlier results[8,18] to the field, providing direct evidence that the current rapid loss of species on Earth[30], and management practices that decrease local biodiversity, threaten ecosystem productivity and the sustainability of nutrient cycling. Observational, laboratory and now field experimental evidence supports the hypotheses that biodiversity influences ecosystem productivity[6,8,17,18], sustainability[4,5,17] and stability[3,6,7,14]. □

Received 10 October 1995; accepted 5 January 1996.

1. Ehrlich, P. & Ehrlich, A. *Extinction* (Random House, New York, 1981).
2. Wilson, E. O. *The Diversity of Life* (Belknap, Cambridge, Massachusetts, 1992).
3. McNaughton, S. J. *Am. Nat.* **111**, 515–525 (1977).
4. Ewel, J. J., Mazzanno, M. J. & Bensh, C. W. *Ecol. Applic.* **1**, 289–302 (1991).
5. Vitousek, P. M. & Hooper, D. U. in *Biodiversity and Ecosystem Function* (eds Schulze, E. D. & Mooney, H. A.) 3–14 (Springer, Berlin, 1993).
6. McNaughton, S. J. in *Biodiversity and Ecosystem Function* (eds Schulze, E. D. & Mooney, H. A.) 361–384 (Springer, Berlin, 1993).
7. Tilman, D. & Downing, J. A. *Nature* **367**, 363–365 (1994).
8. Naeem, S., Thompson, L. J., Lawler, S. P., Lawton, J. H. & Woodfin, R. M. *Nature* **368**, 734–737 (1994).
9. Elton, C. S. *The Ecology of Invasion by Animals and Plants* (Chapman and Hall, London, 1958).
10. May, R. M. *Stability and Complexity in Model Ecosystems* (Princeton Univ. Press, 1973).
11. Goodman, D. *Q. Rev. Biol.* **50**, 237–266 (1975).
12. King, A. W. & Pimm, S. L. *Am. Nat.* **122**, 229–239 (1983).
13. Pimm, S. L. *Nature* **307**, 321–326 (1984).
14. Tilman, D. *Ecology* (in the press).
15. Darwin, C. *The Origin of Species by Means of Natural Selection* (Murray, London, 1859).
16. Ehrlich, P. R. & Mooney, H. A. *BioScience* **33**, 248–254 (1983).
17. Swift, M. J. & Anderson, J. M. in *Biodiversity and Ecosystem Function* (eds Schulze, E. D. & Mooney, H. A.) 15–41 (Springer, Berlin, 1993).
18. Naeem, S., Thompson, L. J., Lawler, S. P., Lawton, J. H. & Woodfin, R. M. *Phil. Trans. R. Soc. Lond.* B**347**, 249–262 (1995).
19. Lawton, J. H. & Brown, V. K. in *Biodiversity and Ecosystem Function* (eds Schulze, E. D. & Mooney, H. A.) 255–270 (Springer, Berlin, 1993).
20. Givnish, T. J. *Nature* **371**, 113–114 (1994).
21. Tilman, D., Downing, J. & Wedin, D. *Nature* **371**, 114 (1994).
22. Andre, M., Brechignac, P. & Thibault, P. *Nature* **371**, 565 (1994).
23. Naeem, S., Thompson, L. J., Lawler, S. P., Lawton, J. H. & Woodfin, R. M. *Nature* **371**, 565 (1994).
24. Tilman, D. *Oikos* **58**, 3–15 (1990).
25. McKane, R. B., Grigal, D. F. & Russelle, M. P. *Ecology* **71**, 1126–1132 (1990).
26. Chabot, B. F. & Mooney, H. A. (eds) *Physiological Ecology of North American Plant Communities* (Chapman and Hall, New York, 1985).
27. Givnish, T. J. (ed.) *On the Economy of Plant Form and Function* (Cambridge Univ. Press, 1986).
28. Grime, J. P. *Nature* **242**, 344–347 (1973).
29. Tilman, D. & Pacala, S. in *Species Diversity in Ecological Communities* (eds Ricklefs, R. & Schluter, D.) 13–25 (Univ. of Chicago Press, 1993).
30. Pimm, S. L., Russell, G. J., Gittleman, J. L. & Brooks, T. M. *Science* **269**, 347–350 (1995).

ACKNOWLEDGEMENTS. We thank C. Lehman, T. Davis, C. Bristow, N. Larson, T. Mielke and G. Abbegian for assistance, and E. Siemann, C. Mitchell, C. Lehman, J. Huisman, F. S. Chapin, G. Daily, S. Naeem, S. Lavorel and J. Lichter for comments. Supported by the US NSF, the Andrew Mellon Foundation, and the Canadian NSERC.

Allometry and simple epidemic models for microparasites

Giulio A. De Leo & Andy P. Dobson

Ecology and Evolutionary Biology, Princeton University, Princeton, New Jersey 08544-1003, USA

SIMPLE mathematical models for microparasites offer a useful way to examine the population dynamics of different viral and bacterial pathogens. One constraint in applying these models in free-living host populations is the paucity of data with which to estimate transmission rates. Here we recast a standard epidemiological model by setting the birth and death rates of the host population and its density as simple allometric functions of host body weight. We then use standard threshold theorems for the model in order to estimate the minimum rate of transmission for the parasite to establish itself in a mammalian host population. Transmission rates that produce different comparable values of the parasites' basic reproductive number, R_0, are themselves allometric functions of host body size. We have extended the model to show that hosts having different body sizes suffer epidemic outbreaks whose frequency scales with body size. The expected epidemic periods for pathogens in different mammalian populations correspond to cycles observed in free-living populations.

The basic microparasite model takes the form[1] $dS/dt = (\nu - \mu)(1 - S/K)S - \lambda(I)S$, and $dI/dt = \lambda(I)S - (\mu + \alpha)I$, where S and I are the density of susceptible and infected individuals respectively, K the carrying capacity in absence of the pathogen[2,3],

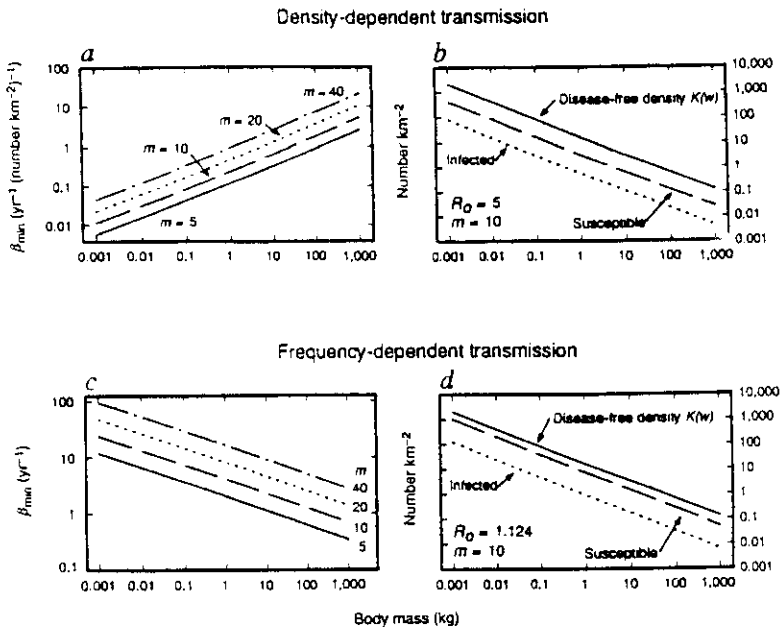

FIG. 1 Allometric relationships of β_{min} for density-dependent transmission (in *a*, $\beta_{min} = 0.0247 m w^{0.44}$) and frequency-dependent transmission (in c, $\beta_{min} = 0.4 m w^{-0.26}$). Host demographic parameters are: $K = 16.2 w^{-0.70}$ (number km^{-2}), $r = \nu - \mu = 0.6 w^{-0.27}$ (years^{-1}), $\mu = 0.4 w^{-0.26}$ (years^{-1}); m indicates the diseased induced mortality factor for infected individuals. If infection spreads ($\beta > \beta_{min}$), host density will drop below the natural carrying capacity. Densities of susceptibles and infectives at equilibrium are inversely related to w (*b* and *d*). In contrast, the fraction of infected hosts and the degree of depression, $d = N_{eq}(w)/K(w)$, below the carrying capacity are independent of w. To derive the relationships in *b* and *d*, transmission rates have been chosen to produce comparable values of R_0 for mammals of different size, namely $\beta(w) = 5\beta_{min}(w)$, and thus $R_0 = K\beta/(\alpha + \mu) = 5$ in the case of density-dependent transmission, and $\beta(w) = 1.124\beta_{min}(w)$, and thus $R_0 = \beta/(\alpha + \mu) = 1.124$ in the case of frequency-dependent transmission. In the latter case, the host population is always driven to extinction when $R_0 \geq 1.22$. The allometric exponents of $I_{eq}(w)$ and $S_{eq}(w)$ are, to a large extent, determined by the carrying capacity $K(w)$ of the disease-free population, and are largely insensitive to the actual values of R_0 and α (provided $R_0 > 1$). Although transmission parameters combine several different epidemiological, environmental and social factors, the scaling of β_{min} is basically due to the following reasons: β_{min} increases with w in the density-dependent case because the number of encounters per unit time decreases with body size as a consequence of the lower population density, thus higher transmission rates are required for the disease to spread. In contrast, when transmission is frequency-dependent, the probability of successfully transmitting the disease does not depend upon density, but is proportional to the time spent in the infective class, $(\alpha + \mu)^{-1}$; as larger individuals have a longer infectious period, β_{min} decreases with w.

μ the host natural mortality rate, ν the maximum birth rate, α the disease-induced mortality, and $\lambda(I)$ the 'force of infection', that is, the rate at which animals become infected. The actual relationship between λ and I depends upon the type of interaction among infected and susceptibles. We examine two main types of transmission: density-dependent, in which the probability of a susceptible becoming infected is proportional to the density of infectives through the transmission rate β, namely $\lambda(I) = \beta I$; and frequency-dependent, in which the same probability is a function of the proportion of infectives, namely $\lambda(I) = \beta I/N$ (where $N = I + S$). Density-dependent transmission is usually assumed for wild-animal diseases[4,5], but frequency dependence may characterize sexually and vectorially transmitted pathogens[6,7]. It may also apply to species that are strongly territorial.

Following the introduction of a single infective into a population of K susceptibles, the disease will only spread provided $dI/dt > 0$. For this to occur, β must exceed a critical value β_{min}, where $\beta_{min} = (\mu + \alpha)/K$ for density-dependent transmission, and $\beta_{min} = \mu + \alpha$ for frequency-dependent transmission. These thresholds are directly related to the basic reproductive number R_0, defined as the average number of secondary cases generated by one primary case in a susceptible population[5,8]. The disease spreads in the population if $R_0 \geq 1$.

These threshold theorems are very useful in investigating disease dynamics and control policies like culling and vaccination, but their use is hampered in practice because direct measurements of β are difficult, if not impossible, to obtain without extensive field data[1,9]. In contrast, estimates of the basic host demography (ν, μ and K) are available for many species. Extensive comparative studies[10–14] relate ν, μ and K to body size and show that they scale with height w as simple allometric relationships.

We can thus derive a similar allometric relationship for the transmission rate and predict the threshold values of β for species over a wide range of body sizes. To generalize this calculation we rescale the disease-induced mortality rate α with respect to the average lifespan of the species. By assuming that α is $(m - 1)$ times the natural mortality μ, more (or less) virulent pathogens will be characterized by larger (or smaller) values of m, as this produces an m-fold reduction of the $(\mu + \alpha)^{-1}$ life expectancy of infected individuals.

After replacing $\nu(w)$, $\mu(w)$, $K(w)$ and $\alpha(w)$ into the equations for

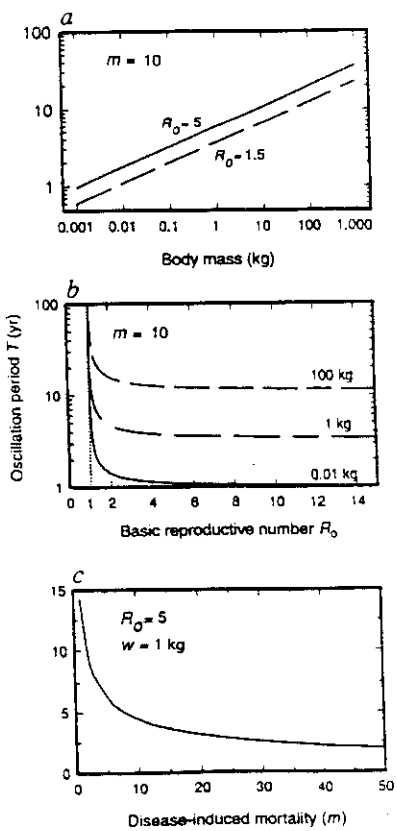

FIG. 2 The oscillation period T (in years) of the host population as a function of the body mass w (in *a*), the basic reproductive number $R_0 = K\beta/(\alpha + \mu)$ (in *b*) and the disease-induced mortality factor m (in c) in the case of density-dependent transmission. Note that for a pathogen that produces a 5–20-fold reduction of life expectancy of a 1-kg animal, the oscillation period ranges between 3 and 7 years, whereas T is practically constant for $R_0 > 3$. Scaling of the oscillation period is basically determined by the scaling of the intrinsic growth rate[16]. The stability properties and the transient dynamics to equilibrium are different when transmission is frequency-dependent: when $w < 10$ kg, the system approaches equilibrium without oscillations, whereas for $w > 10$ kg, damped oscillations with a very long period (>60 years) may occur. In any case, no significant relationship can be detected between body size and equilibrium eigenvalues.

TABLE 1 Threshold conditions on β for five epidemiological models assuming different courses of infection and the density dependence of the host

Model	Basic reproductive number	Threshold criterion for transmission rate	Allometric relationship $\beta(w)$
a, SI model with nonlinear density-dependent fecundity $S' = (\nu - \mu)[1 - (N/K)^c]S - \beta SI$ $N = I + S$ $c > 1$ $I' = \beta SI - (\alpha + \mu)I$ $\alpha = (m - 1)\mu$	$R_0 = \frac{K\beta}{\alpha + \mu}$	$\beta \geq \beta_{min} = \frac{\alpha + \mu}{K}$	$\beta_{min} = 2.47\ 10^{-2} m w^{0.44}$
b, SI model with density-dependent adult mortality $S' = (\nu - \mu)[1 - N/K]S - \beta SI$ $N = I + S$ $I' = \beta SI - (\alpha + \mu + \gamma N)I$ $\gamma = (\nu - \mu)/K$	$R_0 = \frac{K\beta}{\alpha + \nu}$	$\beta \geq \beta_{min} = \frac{\alpha + \nu}{K}$	$\beta_{min} = (2.47\, m + 8.02)\ 10^{-2} w^{0.44}$
c, SIR: susceptible, infective and recovered $S' = (\nu - \mu)[1 - F/K]S - \beta SI$ $F = S + R$ $I' = \beta SI - (\alpha + \mu + \rho)I$ $R' = \rho I - \mu R$	$R_0 = \frac{K\beta}{(\alpha + \mu + \rho)}$	$\beta \geq \beta_{min} = \frac{\alpha + \mu + \rho}{K}$	$\beta_{min} = 2.47\ 10^{-2}\ (m + n) w^{0.44}$ where $n \equiv \rho/\mu$
d, SEI: susceptible, exposed and infective $S' = (\nu - \mu)[1 - F/K]S - \beta SI$ $F = S + E + I$ $E' = \beta SI - (\sigma + \mu)E$ $I' = \sigma E - (\alpha + \mu)I$	$R_0 = \frac{\sigma\beta K}{(\sigma + \mu)(\alpha + \mu)}$	$\beta \geq \beta_{min} = \left(1 + \frac{\mu}{\sigma}\right)\frac{\alpha + \mu}{K}$	$\beta_{min} \approx 2.47\ 10^{-2} m w^{0.44}$ when $\sigma \gg \mu$
e, SEIR: susceptible, exposed, infective and recovered $S' = (\nu - \mu)[1 - F/K]S - \beta SI$ $F = S + E + R$ $E' = \beta SI - (\sigma + \mu)E$ $I' = \sigma E - (\alpha + \mu + \rho)I$ $R' = \rho I - \mu R$	$R_0 = \frac{\sigma\beta K}{(\sigma + \mu)(\alpha + \mu + \rho)}$	$\beta \geq \beta_{min} = \left(1 + \frac{\mu}{\sigma}\right) \times \frac{\alpha + \mu + \rho}{K}$	$\beta_{min} \approx 2.47\ 10^{-2}\ (m + n)\ w^{0.44}$ where $n \equiv \rho/\mu$ and $\sigma \gg \mu$

ν and μ are the maximum fecundity and minimum mortality, respectively. The allometric relaitionship $\beta(w)$ between transmission and body size does not change with respect to the basic SI model when infected individuals can reproduce and the density-dependent reduction in host fecundity is not linear (model *a*). If density affects adult mortality rather than fecundity (model *b*), the threshold condition becomes $\beta \geq \beta_{min} = (\nu + \alpha)/K$. As fecundity has roughly the same allometric coefficient as that of host mortality, β_{min} scales with body mass as in model *a*. Furthermore, if the disease-induced mortality factor m equals 20, β_{min} is only about 10% larger than in model *a* for the same m. The introduction of an immune class (model *c*) or a latent class (models *d* and *e*) slightly complicates the formula for the β threshold, but does not modify the allometry of β_{min} with respect to the previous models. In fact, the average time spent in the latent class, $1/\sigma$, is usually much smaller than the average life expectancy $1/\mu$, therefore $\mu/\sigma \approx 0$. Also, the rate of recovery ρ for the immune class can reasonably be assumed to scale with body size as μ. This explains why the exponent of the w–β relationship is the same as usual. Although the allometric property of the threshold criterion is basically insensitive to variation in the model structure, the actual dynamics may change considerably as models with a latent class may show complex dynamics (such as limit cycles and chaos) if the basic reproductive number is sufficiently high, whereas the simple SI model cannot, unless exogenous seasonal factors are explicitly introduced.

β_{min}, we find that it scales allometrically with body size. β_{min} increases with w when transmission is density-dependent and decreases when transmission is frequency-dependent (Fig. 1). With density-dependent transmission, the model exhibits damped oscillations towards equilibrium densities for any body size (Fig. 1), provided $\beta(w) > \beta_{min}(w)$. The oscillation period is allometric (Fig. 2), with an exponent of 0.260. Peterson *et al.*[15] analysed population cycles of 41 species of birds and mammals: they found that populations fluctuate with a period related to body mass as $8.51w^{-0.263}$. Although our finding matches their data very well, other factors such as spatial and age structure, predation, plant–herbivore relationships and seasonality may be important in explaining the observed patterns. Oscillations are not detectable in the frequency-dependent case, which actually applies only to a minority of wild-animal diseases.

This model does not incorporate many features of the population at the epizoological level, such as different forms of density-dependent growth and the presence of latent and/or recovered classes. But several logical embellishments do not significantly modify the relationship between the body size of the host and the characteristic transmission rate: allometry holds for a wide class of epidemiological models (Table 1). These relations should not be interpreted as deterministic laws giving the exact transmission rate for any species, however, because other important details must be considered. For instance, there is considerable variability in the relationship between population density and body size[14]. Several studies[10,13] have shown that carnivore populations are significantly less dense than those of herbivores and frugivores, so the relationships we described should be modified to include these differences. Also, the two components of the transmission rate should be included: production of infective agents and actual transmission of those agents from infective to susceptible. Large body size may allow greater production of infective viral particles—this would boost the transmission rate in the absence of any compensatory decrease in the actual transmission rate of pathogens.

The allometric relationships shown in Table 1 are a good indication of the general trends underlying the available data and provide a useful initial estimate of β for pathogens of mammals over a wide range of body sizes and population densities. The results in Fig. 1*a*,*c* offer an important insight into whether emergent diseases such as Hantavirus and Ebola virus could become established in the human population. If pathogens use mammals as their normal hosts and transmission is density-dependent, then outbreaks of these pathogens should die out in all but the most crowded human populations; in contrast, if transmission is frequency-dependent, as in the case of human immunodeficiency virus, they should be able to become established. □

Received 2 August; accepted 15 November 1995.

1. Anderson, R. M. & May, R. M. *Infectious Diseases of Humans: Dynamics and Control* (Oxford Univ. Press, 1992).
2. Anderson, R. M. & Trewhella, W. *Phil. Trans R. Soc. Lond.* B **310,** 327–381 (1985).
3. Fowler, C. W. *Ecology* **62,** 602–610 (1981).
4. Kermack, W. O. & McKendrick, A. G. *Proc. R. Soc. Lond.* A **115,** 700–721 (1927).
5. Anderson, F. M. & May, R. M. *Nature* **280,** 361–367 (1979).
6. Antonovics, J., Iwasa, J. Y. & Hassell, M. P. *Am. Nat.* **145,** 661–675 (1995).
7. Thrall, P. H., Biere, A. & Uyenoyama, M. K. *Am. Nat.* **145,** 43–62 (1995).
8. Anderson, R. M. & May, R. M. *Phil. Trans. R. Soc. Lond.* B **291,** 451–524 (1981).
9. Grenfell, B. T. & Dobson, A. P. *Infectious Diseases in Natural Populations* (Cambridge Univ. Press, 1995).
10. Peters, R. H. *The Ecological Implications of Body Size* (Cambridge Univ. Press, 1983).
11. Calder, W. A. *Size, Functions and Life History* (Harvard Univ. Press, Boston, 1984).
12. Schmidt-Nielsen, K. *Scaling: Why is Animal Size so Important?* (Cambridge Univ. Press, 1984).
13. Charnov, E. L. *Life History Invariants* (Oxford Univ. Press, 1993).
14. Silva, M. & Downing, J. A. *Am. Nat.* **145,** 704–727 (1995).
15. Peterson, R. O., Page, R. E. & Dodge, K. M. *Science* **224,** 1350–1352 (1984).
16. Mollison, D. *Nature* **310,** 224–225 (1984).

ACKNOWLEDGEMENTS. A.P.D. thanks the Geraldine R. Dodge Foundation for financial support; G.D.L. thanks S. Levin and M. Gatto for their support.