Aggregation and stability in parasite–host models

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SUMMARY

This paper generalizes the two-dimensional approximation of models of macroparasites on homogeneous populations developed by Anderson & May (1978), focusing on how the dispersion (the variance to mean ratio) of the equilibrium distribution of parasites on hosts is related to the stability of the equilibrium. We show in the approximate system that the equilibrium is stabilized not by aggregation, but by dispersion which increases as a function of the mean. Computer simulations indicate, however, that this analysis fails to capture properly the dynamics of the full system, raising the question of whether any two-dimensional system could produce an adequate approximation. We discuss the relevance of our results to several empirical studies which have examined the relation of dispersion to the mean.

Key words: aggregation, stability, host–parasite models.

INTRODUCTION

The interaction of the distribution and dynamics of parasites on hosts has been considered from a variety of theoretical angles in recent years (Anderson & May, 1985; Pacala & Dobson, 1988). A theme throughout much of the discussion has been the causes and consequences of the widely observed pattern of aggregated distributions of parasites (clustering of parasites greater than that expected at random), data which have traditionally and successfully been fit by the negative binomial distribution (Anderson & May, 1985). Several reasons have been proposed for this observation, generally falling into the broad categories of host heterogeneity and dynamic factors (Anderson & Gordon, 1982; Anderson *et al.* 1982; McCallum, 1982; Pacala & Dobson, 1988).

In a now-classic paper, Anderson & May (1978) used this empirical observation to reason about the dynamic consequences of such distributions, considering in particular the stability of interactions in which a parasite regulates its host population by increasing the mortality of heavily infected hosts. Their method consists of a reduction of a complete mathematical description of the system to a simple analogue of a predator–prey system in which only total population sizes of hosts and parasites are tracked. To deal with the problem of the distribution of parasites among hosts, they assume that the distribution retains a particular shape regardless of the mean number of parasites per host. They contrast the regular positive binomial, the aggregated negative binomial distribution with fixed clumping parameter k, and the random or Poisson distribution, showing that the equilibrium point is unstable in the first case, stable in the second, and neutrally stable in the last. This result seems to imply that aggregated distributions enhance stability.

In this paper, we show that stability in models of this form is determined not by the degree of dispersion itself, but by the dependence of dispersion on the mean. Computer simulations indicate that the dispersion does not behave like a function of the mean throughout the dynamics, and that the two dimensional system is not an accurate approximation of the dynamics. We postulate that a 3-dimensional system exists, involving the dispersion as a dynamic variable, that more accurately approximates the dynamics. Further discussion of this approach can be found in a companion paper (Kretzschmar & Adler, unpublished). We conclude by reviewing some recent work regarding the expected and observed relation of dispersion to the mean and by commenting on the relevance of our work to these investigations.

THE GENERAL MODEL

Our derivation of the model generalizes that of Kretzschmar (unpublished). The model follows the dynamics of host birth and death, and of parasite death, reproduction and transmission. We ignore age structure in the host population, assume that adult parasites die when their hosts die, and neglect the

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Table 1. Description of variables

Variables	
Variable	Description
i	Number of parasites in a host
t	Time
p_i	Number of hosts with exactly i parasites
H	Total number of hosts
P	Total number of parasites
r_i	Fraction of hosts with exactly i parasites
x	Mean number of parasites per host
π	Variance to mean ratio of the distribution of parasites on hosts

dynamics of the free-living stages of the parasite (Anderson, 1982). The system is described by classifying hosts according to their parasite burden, a technique introduced by Kostizin (1934) and extended to include such factors as age-structure (Hadeler, 1982; Kretzschmar, 1989*a*, *b*) and host heterogeneity (Pacala & Dobson, 1988).

We denote by $p_i(t)$ the number of hosts per unit area with exactly i parasites at time t. See Table 1 for a complete description of variables used in this paper. The following parameters, which can all be density dependent, describe the dynamics: b_i is the rate at which hosts with i parasites die, a_i is the rate at which hosts with i parasites reproduce, s_i is the relative susceptibility of hosts with i parasites, u_i is the *per capita* parasite death rate in hosts with i parasites, λ_i is the *per capita* parasite egg production rate in hosts with i parasites, ϕ_i is the rate at which hosts with i parasites acquire new parasites. We assume that all newborn hosts are born free of parasites, i.e. into class p_0.

The model is then described by the following set of differential equations

$$\begin{aligned}\dot{p}_0 &= -(b_0+\phi_0)p_0+u_1p_1+\sum_{i=0}^{\infty}a_ip_i,\\ \dot{p}_i &= -(b_i+\phi_i+iu_i)p_i+(i+1)u_{i+1}p_{i+1}\\ &\quad +\phi_{i-1}p_{i-1},\quad (i=1,\ldots,\infty),\end{aligned} \tag{1}$$

where $\dot{}$ denotes differentiation with respect to time. This set of differential equations describes the time evolution of a parasite population and its host populations starting from a given initial distribution of parasites on the hosts.

We define the aggregated variables

$$H=\sum_{i=0}^{\infty}p_i,\quad P=\sum_{i=0}^{\infty}ip_i,\quad P_l=\sum_{i=0}^{\infty}i\lambda_ip_i. \tag{2}$$

H is the total density of hosts, P is the total number of parasites per unit area and P_l is the total number of eggs produced per unit area per unit time.

We assume that

$$\phi_i=s_iP_lg(H).$$

The infection rate is assumed to depend linearly on the number of eggs produced, with saturation described by the function $g(H)$. $g(H)$ is the probability that a single host is attacked by a single egg as a function of the total density of hosts, and will necessarily be a decreasing function. $Hg(H)$ is the attack probability of a single egg on all hosts in the area and will necessarily be an increasing function since the probability of an egg finding a host increases as the density of hosts increases. Therefore,

$$\frac{d}{dH}Hg(H)>0. \tag{3}$$

We now define the further aggregated variables

$$\begin{aligned}&H_b=\sum_{i=0}^{\infty}b_ip_i,\quad H_a=\sum_{i=0}^{\infty}a_ip_i,\quad H_s=\sum_{i=0}^{\infty}s_ip_i,\\ &P_b=\sum_{i=0}^{\infty}ib_ip_i,\quad P_u=\sum_{i=0}^{\infty}iu_ip_i.\end{aligned} \tag{4}$$

By summing up equations (2) we arrive at the following system for H and P

$$\begin{aligned}\dot{H}&=-H_b+H_a,\\ \dot{P}&=-P_b-P_u+H_sg(H)P_l.\end{aligned} \tag{5}$$

This system of equations is not closed in the sense that knowledge of H and P alone is insufficient to describe the dependence of the dynamics on the other aggregated variables, i.e. these variables cannot be described as functions of H and P alone.

A special case

In this section, we reduce the model to the special case considered by Anderson & May (1978). First of all, we ignore density-dependent effects on parasite mortality and fecundity and on host susceptibility, although some evidence indicates this may not be justified (Michael & Bundy, 1989). We also assume that host fecundity is not diminished by parasite burden. Using these assumptions, we set the subscripted parameters u_i, λ_i, s_i and a_i to the constant values u, λ, s and a, so that $H_a=aH$, $H_s=sH$, $P_l=\lambda P$, and $P_u=uP$. Following Anderson & May (1978), we assume that host mortality increases linearly with parasite burden, or that $b_i=b+\alpha i$.

Substituting these assumptions into the expressions for H_b and P_b, equation (5) becomes

$$\dot{H} = (a-b)H - \alpha P,$$
$$\dot{P} = -(b+u)P + s\lambda H g(H) P - \alpha \sum_{i=0}^{\infty} i^2 p_i. \qquad (6)$$

The only term not expressed in terms of the total population densities H and P is the last. Anderson & May (1978) assumed that the p_i/H have a negative binomial distribution with constant k to close the system. In the following section, we generalize their approach.

THE VARIANCE TO MEAN RATIO

We first reformulate equations (6) to show explicitly how they can be expressed in terms of the variance to mean ratio. Set

$$r_i = p_i/H$$

to be the fraction of hosts carrying a burden of exactly i parasites. The mean number of parasites per host, P/H, we denote by x, and the ratio of the variance to the mean by π. Since the variance V satisfies

$$V = \sum_{i=0}^{\infty} i^2 r_i - x^2,$$

it follows that

$$\sum_{i=0}^{\infty} i^2 r_i = x(\pi + x).$$

By changing variables we can rewrite equations (6) in terms of H and x as

$$\dot{H} = H(a-b-\alpha x),$$
$$\dot{x} = x(s\lambda H g(H) - a - u - \alpha\pi). \qquad (7)$$

To close this system, we assume that π depends only on the mean of the distribution, or that $\pi = \pi(x)$. Note that this is equivalent to assuming that the variance is a function of the mean. This approach was introduced by Taylor (1961), who showed that in many natural populations the variance of population number is a power of the mean with the particular value of the exponent varying from species to species. For the negative binomial distribution with constant k, the variance to mean ratio is again a function of the mean, but this need not hold in general as we discuss in the following section.

With this assumption, the equilibrium must satisfy

$$x^* = (a-b)/\alpha,$$
$$s\lambda H^* g(H^*) = a + u + \alpha\pi(x^*).$$

x^* will be positive as long as $a > b$, and there will be a positive solution for H^* as long as the product of host susceptibility with parasite fecundity, $s\lambda$, is sufficiently large, since $Hg(H)$ is an increasing function (equation 3). We assume henceforth that a positive solution exists.

The Jacobian matrix at this equilibrium, which determines the stability, is

$$\begin{pmatrix} 0 & -\alpha H^* \\ \lambda s x^* \, d/dH \, Hg(H)|_{H=H^*} & -\alpha x^* \pi'(x^*) \end{pmatrix}.$$

Here π' denotes the derivative of π with respect to the mean x. The stability of the equilibrium is determined by the determinant and trace of this matrix (May, 1973). Equation (3) implies that the determinant

$$\alpha\lambda s x^* H^* \, d/dH \, (Hg(H))|_{H=H^*}$$

is positive. The equilibrium is stable if the trace is negative, or

$$\alpha x^* \pi'(x^*) > 0, \qquad (8)$$

and unstable if the inequality is reversed (stability when the trace is zero is not determined by the linearized system). Since $x^* > 0$, the equilibrium will be stable if the variance to mean ratio is an increasing function of the mean, and unstable if the variance to mean ratio is a decreasing function of the mean.

This result complements recent work (Hassell & Pacala, 1991) on stability of host–parasitoid models where hosts experience a range of risks. They show for a wide range of variability-generating mechanisms that the system is stable if the coefficient of variation of the degree of risk is less than 1. In earlier work, Perry & Taylor (1986) showed that variability in the clumping parameter k can modify the stability of the equilibrium. Note that their result resembles that of this paper in that the parameters might depend systematically on the mean rather than being fixed constants.

This simple criterion can be used to demonstrate and re-interpret the results of Anderson & May (1978). In the case of a negative binomial distribution, one has

$$\pi(x) = 1 + x/k,$$

a linearly increasing function and thus a stable equilibrium. A Poisson distribution has constant variance to mean ratio of 1 and this produces a zero derivative of π and neutral stability. In the case of a positive binomial distribution, one has

$$\pi(x) = 1 - x/M$$

for fixed parameter M, a decreasing function of x and thus an unstable equilibrium.

In the case where the variance has a power law relation to the mean (Taylor, 1961), one has

$$\pi(x) = ax^{b-1},$$

where a and b are fixed parameters for a particular system. Here, the variance to mean ratio is a decreasing function of the mean just in case $b < 1$.

It is worthwhile to note that this result depends very much on the assumptions of density inde-

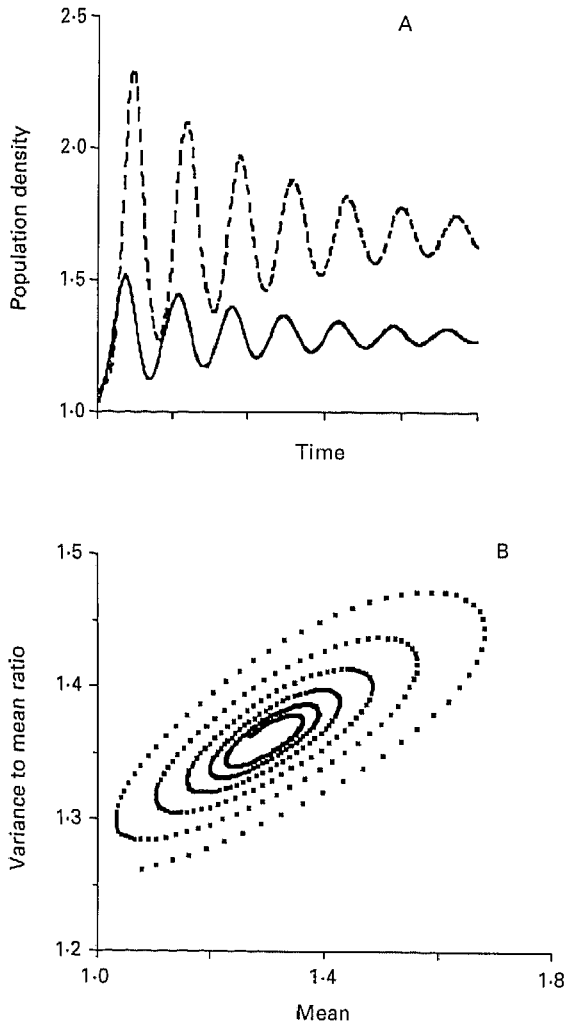

Fig. 1. (A) Dynamics of the total density of hosts H (– – –) and the total density of parasites P (——) as a function of time. (B) Variance to mean ratio (π) plotted against the mean x in a simulation of equation (2) with density-independent parasite mortality ($\gamma = 0$). Note that the variance to mean ratio is roughly an increasing function of the mean, in accord with the stability of the equilibrium. Other parameter values are $b = 0{\cdot}2$, $a = 1{\cdot}5$, $u = 1$, $\lambda = 3$, $s = 1$ and $\alpha = 1$. The function $g(H)$ is simply the constant value 1.

pendence and on the particular form of parasite-enhanced mortality rates used in equations (6). Firstly, if the host mortality rates do not increase linearly with parasite burden, then the variance to mean ratio does not arise naturally from the analysis. Secondly, even making the parasite death rate a linearly increasing function of parasite burden, which preserves the general form of the equations, breaks down the simplicity of the above result. In particular, assume

$$u_i = u + \gamma i.$$

Then

$$P_u = uP + \gamma \sum_{i=0}^{\infty} i^2 p_i.$$

Substituting this into equation (5) and transforming into the variables H and x as before gives the system

$$\dot{H} = H(a - b - \alpha x),$$
$$\dot{x} = x(s\lambda H g(H) - a - u - \alpha\pi - \gamma\pi + \gamma x).$$

The Jacobian matrix of this system at this equilibrium is

$$\begin{pmatrix} 0 & -\alpha H^* \\ \lambda s x^* \, d/dH \, Hg(H)|_{H=H^*} & (-(\alpha+\gamma)\pi'(x^*) + \gamma)\, x^* \end{pmatrix}$$

The determinant of this system is unchanged from that of the earlier system and therefore is positive. The trace at equilibrium is now

$$(-(\alpha+\gamma)\pi'(x^*) + \gamma)\, x^*.$$

Thus the equilibrium is stable if

$$\pi'(x^*) > \frac{\gamma}{\gamma + \alpha} \tag{9}$$

and unstable if the inequality is reversed. The equilibrium is necessarily unstable if $\pi'(x^*) < 0$ as before, and is necessarily stable if $\pi'(x^*) > 1$. However, if $0 < \pi'(x^*) < 1$ the equilibrium will be destabilized by sufficiently large γ. That is, an increasing variance to mean ratio may not be sufficient to stabilize the system in the presence of sufficiently high density-dependent parasite mortality (Anderson & May, 1978).

THE DYNAMICS OF THE FULL MODEL

Two mechanisms could constrain the variance to mean ratio to be precisely a function of the mean. First, it could be maintained by rapidly acting forces not accounted for in the dynamics described by the model equations (2); factors which might cause the system to quickly equilibrate to, perhaps, a negative binomial distribution with constant k. Second, this function, and indeed the entire distribution, arises only from processes described in model equations (2). Using a generating function approach, Kretzschmar (unpublished observation) has found the equilibrium distribution of parasites on hosts for equations (2) with assumptions embodied in equation (6) and shown that it is indeed aggregated, though not exactly negative binomial. In order to apply the result of this paper, one must ask whether the dispersion acts like a function of the mean during the course of the dynamics, and whether this functional dependence correctly predicts stability. In addition, it is interesting to consider whether stability is determined by the dispersion itself, as hypothesized by Anderson & May (1978).

Computer simulations of truncated versions of equations (2) indicate that all of these questions must be answered in the negative. The simulations were truncated at $i = 20$, though due to the low average number of parasites per host there were no detectable differences among truncation levels above $i = 10$. Initially, each simulated host was infected with

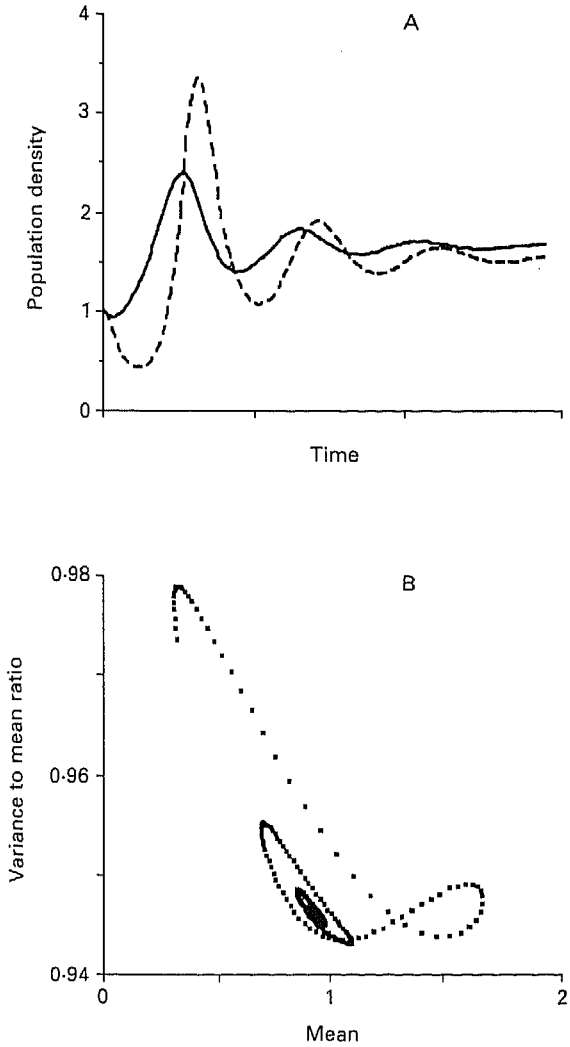

Fig. 2. The dynamics in a case with density-dependent parasite mortality ($\gamma = 1$). Notation and parameter values are otherwise identical with Fig. 1. Note that here the variance to mean ratio is roughly a decreasing function of the mean, in contrast to the prediction of equation (9).

exactly 1 parasite. Fig. 1A illustrates the dynamics of H and P for the case $\gamma = 0$, the special case of density-independent mortality analysed by Anderson & May (1978). Fig. 1B shows that the variance to mean ratio is generally increasing with the mean, in accord with the stability of the equilibrium as indicated by equation (8). In the case of density-independent parasite mortality ($\gamma = 0$) we were unable to find parameter values producing an unstable equilibrium. Given the finding of Kretzschmar (unpublished observation) that the equilibrium distribution in this case is always aggregated, this is consistent with the original hypothesis of Anderson & May (1978) that aggregation leads to stability. Fig. 2A and B show the dynamics with a positive value of γ for which the dynamics of H and P (Fig. 2A) are still stable, but for which the variance to mean ratio is roughly a decreasing function of the mean (Fig. 2B). Accord-

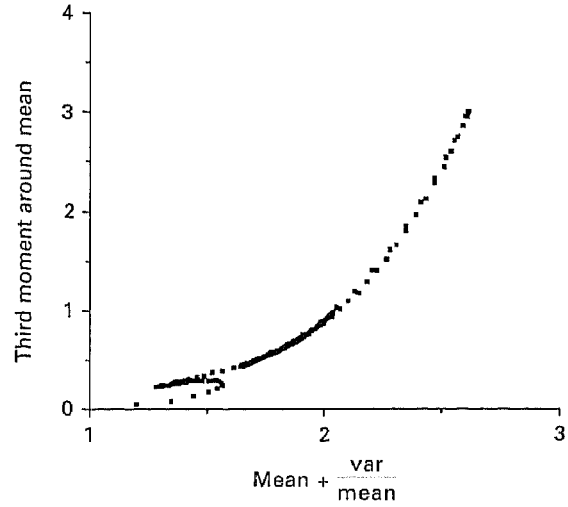

Fig. 3. The dynamics of the third moment around the mean plotted against the sum of the mean and the variance to mean ratio for the same simulation shown in Fig. 2. Note how the third moment appears to behave like a function of the lower moments.

ing to equation (9) such a relation should produce an unstable equilibrium. This discrepancy is due to the fact that the variance to mean ratio fails to be very well described by a function of the mean. Note that the equilibrium distribution is under-dispersed in this case, which, according the reasoning of Anderson & May (1978) should produce instability, in conflict with simulation results.

Fig. 3 illustrates simulation results which indicate that there may be a 3-dimensional system tracking the dynamics of H, x and π which accurately describes the dynamics of the infinite system. Shown are the same dynamics as in Fig. 2, but here the third moment around the mean is plotted against the sum of the mean and the variance to mean ratio. The third moment of the distribution does seem to be well approximated by this function of the lowest moments, and the same appears to hold for higher moments not illustrated. If this is true in general, then the whole distribution, perhaps after a brief transient, could be described by only these lower moments. We are currently investigating this approach. A related approach to designing a 3-dimensional system is to assume, as with the 2-dimensional approximation of Anderson & May (1978), that the distribution remains constrained to a fixed family throughout the dynamics, but to allow two of the parameters describing that distribution to vary rather than just one. For instance, in the case of the negative binomial, this amounts to allowing the clumping parameter k to vary along with the mean (Kretzschmar & Adler, unpublished observation), as in Perry & Taylor (1986).

DISCUSSION

Our results indicate that the dependence of the degree of aggregation on the mean may have more important consequences for the dynamics than does the simple presence or absence of aggregation. This result meshes well with a number of observations regarding the relation between the dispersion and the mean. Note that our model considers this relation over time in a single population, as opposed to across age classes within a population or across different populations. Measurements have been made in all three of these cases. Most closely related to the approach presented in this paper are the results of Scott (1987), who found a roughly linear increase of the variance to mean ratio as a function of the mean in a system of monogeneans infecting guppies. The host dynamics in this case depended upon pulsed immigration rather than breeding as in our model, and tended to produce oscillatory dynamics. Whether this is a consequence solely of the immigration of naive individuals, a failure of our method of analysis, or an indication of the presence of important density-dependent factors such as parasite mortality is an issue worthy of further consideration. In earlier work, Scott & Anderson (1984) analysed this data using a negative binomial distribution, concluding that the variance to mean ratio proved to be a more sensitive statistic.

Pacala & Dobson (1988) used the behaviour of the variance to mean ratio as a function of the mean as a test of density independence, showing under very general conditions that aggregation within age classes increases linearly with the mean burden in those classes when density-dependent processes are not acting. Variance among individuals is generated in their model by host heterogeneity in susceptibility or response to parasites. Since the negative binomial distribution with constant k produces a linear increase of dispersion as a function of the mean, it is thus singled out as an appropriate null model. McCallum (1982) and McCallum & Anderson (1984) also considered models with fixed host heterogeneity, showing that an estimate of the negative binomial parameter k remains constant even in the presence of some density dependence in the host dynamics. McCallum (1982) found no trend toward increasing or decreasing k as a function of the mean burden in his data. In related empirical work, Evans, Whitfield & Dobson (1981) showed that both the mean and the variance to mean ratio of the number of metacercerial cysts on molluscs increased with the size of the host, once again indicating the importance of host heterogeneity upon distribution patterns.

Gordon & Rau (1982) and Lemly & Esch (1984) argued that a decrease in the variance to mean ratio at high mean infection levels provides evidence for parasite-induced mortality. Although such an argument is consistent with the approach presented in this paper, the results derived here are based on the assumption of parasite-enhanced mortality rates and cannot be used to test their argument. Guyatt *et al.* (1990) considered the relation between aggregation and mean helminth burden in human populations. In the case of humans, it is unlikely that the primary form of density dependence is parasite-enhanced host mortality, but it is interesting to note that they find a linear increase in the negative binomial parameter k as a function of the mean. Although it might seem that this implies decreased aggregation with increased mean, computation of the variance to mean ratio in this case shows that this ratio increases as long as the intercept of the regression of k on the mean is positive, as found in their study. Studies of this sort could cast light on dynamics if they could be thought of as a cross-section through several similar systems at different stages of an oscillation, but it seems much more likely that differences are generated by differences between populations.

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