Critical Behavior in Population Biology

Zigurts K. Majumdar

May 5, 2000

Abstract

This essay seeks to give an overview of several methods employed from the theory of critical phenomena in order to understand the behavior of biological populations. In particular, properties which exhibit power-law scaling are studied. Derivations of scaling laws through lattice models and self-similarity postulates are discussed as well as a dynamical models .

1 Introduction

The behavior of biological populations has been studied in a number of ways. As members of the human population we may wonder how much our own social ties and behavior might affect the dynamics and trends in a population, the current answer seems to be that it's all a matter of scale.

There has been great interest for a number of pracitcal reasons such as predicting the population size, the spread of epidemics, the interaction of different species and their evolution as well as a number of others. An interesting feature of a great number of phenomena in the dynamics of populations have been observed to exhibit power law scaling.

As early as 1921 Olaf Arrhenius discovered the first ecological scaling law, as a candidate for the well known species-area relationship (SAR), relating how the distribution of species goes with area. He found that the number of species in a given area followed a power law : $S \sim A^z$ where S is the number of species in an area A and z is the exponent. This particular power law and many others, have been studied through a variety of approaches and models. One of the foremost current approaches is the assumption of self-similarity, which is well known as a property of critical phenomena where the response functions display power law scaling near the critical point. Fluctuations occur on all length scales and the system is then scale invariant. Another characteristic is that of an *order parameter* which is zero in a disordered phase and becomes nonzero at the critical point. Such a quantity is useful to identify characteristic changes in population dynamics.

We shall examine some approaches to ecology which derive observed critical exponents based on the postulate that the systems are self-similar. We shall also examine some dynamical systems and mean field approaches to population probelms and we will see that these appraoches can apply to a wide variety of population problems.

2 What do Birds and Magnets have in Common?

A fundamental way to characterize the behavior in biological populations is that the mechanism is a *contact process*, i.e., an individual must interact locally with one of its nearst neighbors. In particular where the dynamics of the system can be considered to originate solely from contact processes, one might expect that the nature of the power law scaling could be similar to that of a nearest neighbor spin interaction, where near criticality the fluctuation of a single spin can propagate through the entire system (e.g. near T_c in a ferromagnetic transition), corresponding to a diverging correlation length in the thermodynamic limit. Looking at a variety of data such *long-range correlations* are in fact observed.

Keitt and Stanley et al. [15], noticed such behavior in studying the population variability of roughly 600 different species of birds. Figure 1 corresponds to the distribution in population growth rates where R_s is the growth rate calculated by

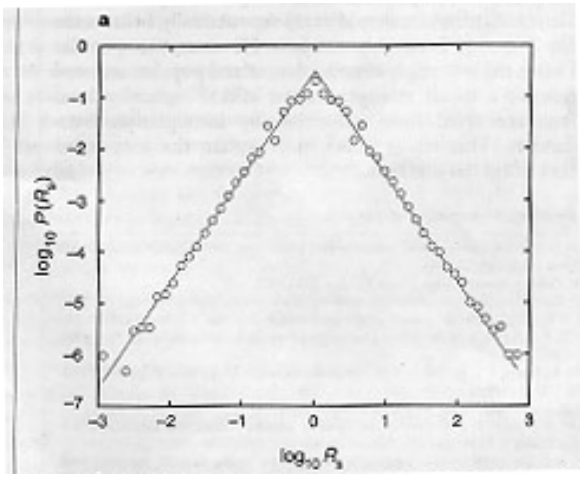

Figure 1: Scaling in the distribution of growth rates recorded in a given area from a North American bird survey.

dividing species abundances in successive years. Here we find that the distribution is symmetrical, as many species are increasing in abundance as decreasing[1], the distribution also follows a power law :

$$P(R_s) \propto \left\{ \begin{array}{ll} R_s^{\alpha} & \text{if } R_s \leq 1 \\ R_s^{-\alpha} & \text{if } R_s \geq 1 \end{array} \right\}$$

where $\alpha = -2$. Hence there is no characteristic scale of fluctuation in population size, instead we see a broad spectrum of growth rates.

The scaling of local species' lifetimes was measured as the time between colonization (when a species was absent the previous year) and extinction (absent, but recorded the previous year) from a local patch where data was taken. Their distribution yields a power law with an exponential cutoff :

$$P(\tau) = A\tau^{-\beta} e^{-\tau/\tau_{ch}}$$

where A is a constant and τ_{ch} sets the timescale at which the power-law scaling is no longer valid due to a finite data set.

The question now is: how does this type of behavior arise, what are the nature of the interactions that cause this type of correlated behavior? A key step in dealing with this question formally has been to simplify the microscopic interactions to a simple set of rules and assumptions which can still capture the large scale behavior. We will now discuss several such examples.

[1] one might be awed by this fact to think what kind of self-regulatory mechanism causes such a distribution

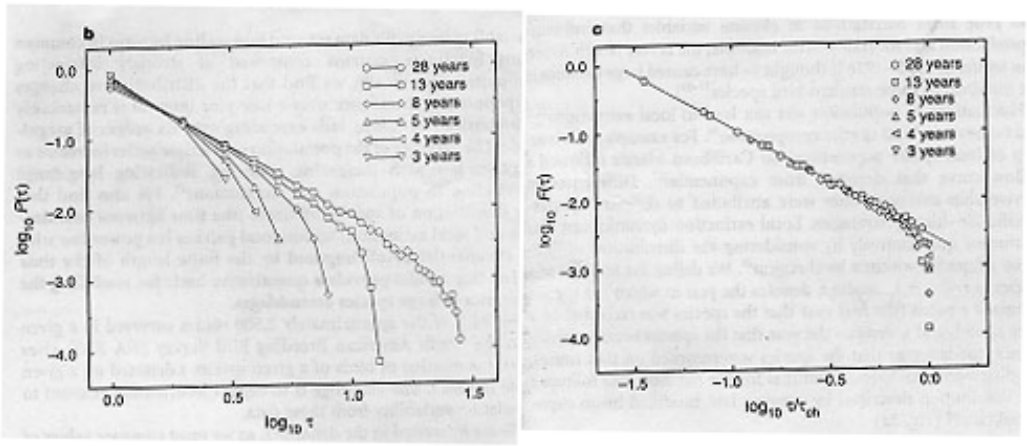

Figure 2: b shows finite size analysis of the species' lifetime distribution. The distributions are from nonoverlapping subsets of each time series, broken into increasingly shorter sequences. The number of years shown are the maximum possible species' lifetimes between the first and last year of the individual time series. c shows when the axes are rescaled to remove the finite scaling, the data collapses onto a single power-law curve. here $F(\tau) = P(\tau)/A\tau_{ch}^{-\alpha}e^{-\tau/\tau_{ch}}$ plotted against the rescaled time axis τ/τ_{ch}

3 Dynamical Systems and Self-Organized Criticality (SOC)

Dynamical systems are fundamentally different from equilibrium critical phenomena and cannot be described by thermodynamic variables which have no time dependance. In 1987 Bak, Tang and Weisenfeld (BTW) proposed the concept of *Self-Organized Criticality*[2] (SOC), to explain a wide variety of dynamical systems that evolve spontaneously into a critical state with no characteristic time or length scale. Like continuous phase transitions in equilibrium statistical mechanics, the systems are scale invariant and the response functions exhibit power law scaling. The defining feature of SOC is that no fine tuning of the system is needed to drive it to criticality (e.g., adjusting the temperature or external field) and so is said to "self-organize".

[2]The reader may further wish to examine the host of literature for greater detail on self-organized criticality and relation to 1/f noise, only some relevant aspects are mentionedhere

Various cellular automata models with a simple set of microscopic rules were created to understand this phenomena and the hope was that the critical exponents from these could group a wide range of dynamical systems into "universality classes" analagous to the Ising magnet for equilibrium systems. It turned out, however that the models were not truly "self-organized" critical, since the criticality did depend on certain relevant parameters.

Although there is not yet unequivocal proof of SOC occurring in nature, the models have proved to be powerful techniques in capturing the scaling behavior of population phenomena.

3.1 Epidemics, Duration and Size

We will now examine one such model [10], the Forest Fire Model (FFM) [3, 4, 5] in the study of measels epidemics in an isolated population. An epidemic can be defined as: a finite number of infected cases recorded in a sequence of consecutive months bounded by an absence [2]. Understanding the power laws that govern the occurence of these events can lead to very useful information in estimating e.g., both their duration and magnitude.

The study chose data recorded from the incidence of measels on one of the Faroe Islands. The isolated popualtion was chosen since external interactions of the given population are thereby minimized [2]. The two quantities of interest in this study are : $N(s)$, the number of epidemics with size s where s denotes the number of persons infected and $N(t)$, the number of epidemics which last for a duration of time: t $= \tau_{end} - \tau_{start}$. Both quantities were observed to obey a power law behavior given by:

$$N(s) \sim s^{-1-b}$$

$$N(t) \sim t^{-1-c}$$

with $b \simeq 0.28$ and $c \simeq 0.8$ (see Fig 1). The exponents were also found to be invariant under selection of truncated portions of the data. Using the FFM the same exponents were calculated and found to be in relatively good agreement with the results from the model giving: $b \simeq 0.29$ and $c \simeq 1.5$.

Forest Fire Model

The forest fire model is a cellular automata model performed on a d dimensional hypercubic lattice in this case an $L \times L$ with $2d = 4$ nearest neighbors. L is chosen large enough to avoid finite-size effects so power law scaling should be seen with minimal corrections. The following rules apply:[3]

[3]we here use the relevant terminology for the situation, a buring forest was oviously originally

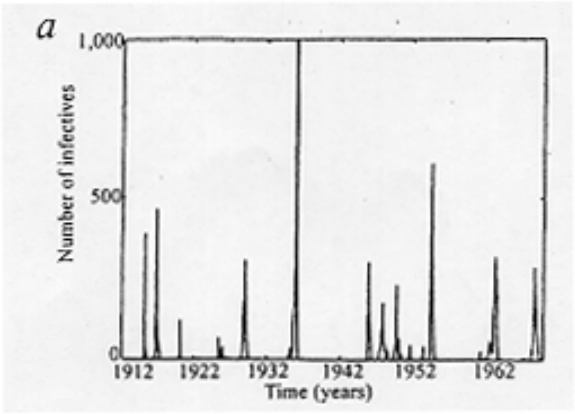

Figure 3: Raw data taken over a 58 year interval of the outbreak of measels on the Faroe islands. Adopted from Rhodes and Anderson [2]. Below we see the data plotted on a log-log scale with clear evidence of a power law behavior.

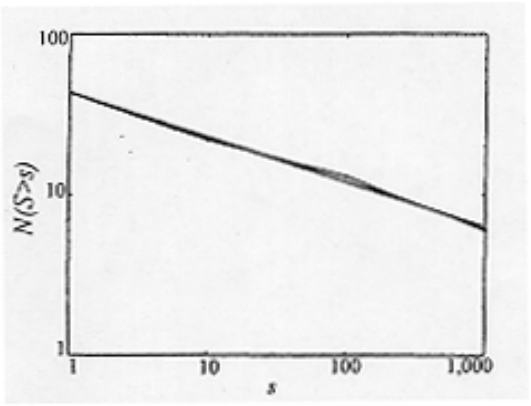

Figure 4: The y-axis is the number of epidemics with size greater than s and the x-axis s, we see a power law dependance of the form : $\log N(>s) = a - b\log(s)$.

In each time step the model is updated as follows:

1. Susceptibles who have at least one infective nearest neighbor become infected
2. Infectives, I become inactive and the site becomes empty, E
3. Susceptibles, S are introduced onto empty lattice sites with probability p
4. New infectives arise when a susceptible is spontaneously infected with probability f (corresponding to an immigrative infection from external sources).

used, the transformation to this system is: burning tree $\rightarrow$ infective, green tree $\rightarrow$ susceptible, growing tree $\rightarrow$ new susceptible, lightning struck tree $\rightarrow$ spontaneous infective

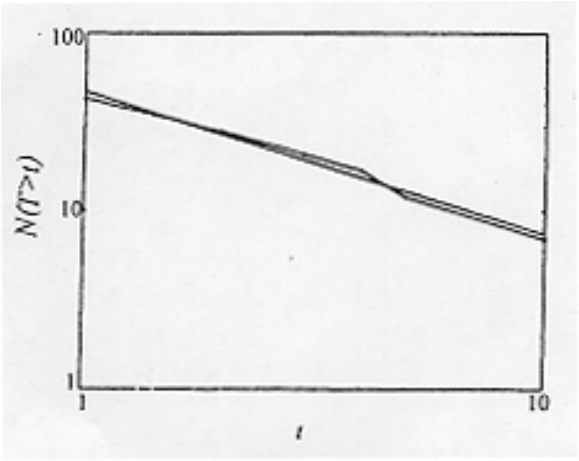

Figure 5: The y-axis is the number of epidemics time duration greater than t and the x-axis t, with a similar dependace as for s.

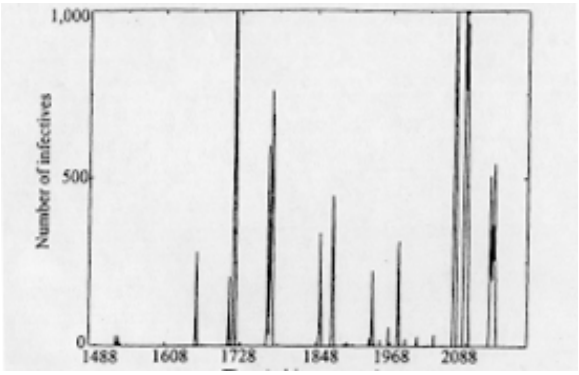

Figure 6: The results of the simulation using a 250×250 lattice. The horizontal axis is time in arbitrary years.

Periodic boundary conditions are used. The critical exponents are derived from a steady state condition where we consider the mean number of new susceptibles = the mean number of infectives. In the following we sketch a derivation of the temporal exponent describing the distribution of epidemic lifetimes, the spatial one (i.e., the size of the epidemic at some time, resulting from infected clusters) follows analagously and results from that are used.

Let $N(T)$ be the portion of infectives that live for T time steps and $T(s)$ be the average time a cluster of size s needs to be infected. The exponents $\bar{b}$ and μ' are defined as :

$$s \propto T(s)^{\mu'} \; and \; N(T) \propto T^{-\bar{b}} \tag{1}$$

which is related to $n(s)$ the mean number of clusters with s susceptibles by :

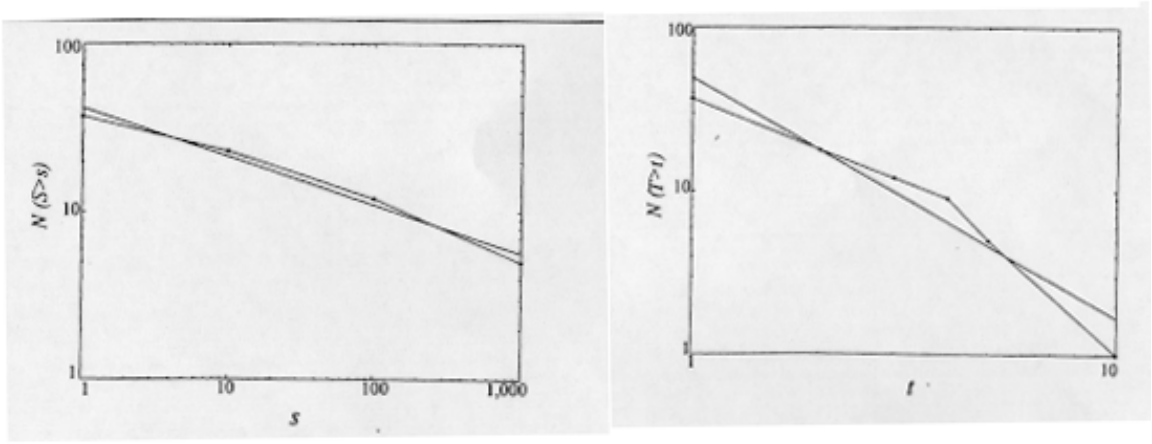

Figure 7: Clearly we see behavior that matches with that of the real data. The temporal exponent is severly affected by the poor prediction of long epidemics > 10 months, but comparison with data shows a good prediction for epidemics lasting up to 5 months [2].

$$N(T)dT \propto sn(s)ds. \tag{2}$$

the time scale of the system is set by $T_{max} \propto \left(\frac{f}{p}\right)^{-\nu'}$ and the correlation length goes as: $\xi = \left(\frac{f}{p}\right)^{-\nu}$ with the dynamical critical exponent z defined by: $T_{max} \propto \xi^z$. From this we can identify

$$z = \frac{\nu'}{\nu}$$

. The average lifetime of the infectives is:

$$\bar{T} = \frac{\sum_{s=1}^{\infty} sn(s)T(s)}{\sum_{s=1}^{\infty} sn(s)} \propto \left(\frac{f}{p}\right)^{-\tilde{\nu}'} \tag{3}$$

where $\tilde{\nu}'$ is an exponent whose relation depends on ν'. $N_s(t)$, the average number of sucseptibles that get infected in t time steps defines the temporal infective-infective correlation function $G(\tau)$ where :

$$G(\tau) \propto \sum_{s=1}^{\infty} n(s)s \sum_{t=0}^{\infty} N_s(t)N_s(t+\tau) \tag{4}$$

, criticality is reached in the limit that $\frac{f}{p} \to 0$ and $f \to 0$. With some more work the exponent can be read off, in the case of the measels simultation we then have $-\bar{b} = -1 - b$.

The exponents derived from the model are also universal in the sense that they do not depend on the microscopic details of the model. The same results were obtained for the forest fire model when using a triangular lattice or switching from nearest to next nearest neighbor interactions. Another modification was made by including an immunity parameter and still the exponents did not change [4]. The social networks of the community are known to be much more complex than the rules prescribed, yet the large-scale behavior seems to be independant, and we see that this formalism can applied to a wide variety of population probelms which depend on contact processes.

Other examples where SOC models have been applied are in the Hawaiian Avifauna where the extinction rate goes as a power law with the number of species. The behavior is thought to be minimally stable around a critical point in accordance with the Sanpile Model [5, 7] of SOC. Order-disorder transitions in ant societies are also given a similar treatment, where when a critical density is achieved in a given area, the food carrying capacity exhibits power-law scaling [9].

4 Mean Field Model for Contact Processes

This particular approach, albeit a rather simple version is characteristic of the features needed to describe the spread of an epidemic. Consider a d-dimensional lattice with $2d$ nearest neighbors. The following rules apply:

- Two types of objects on each lattice site: *active* (A) and *inactive* (I)
- A, can "die" with probability γ and then A $\to$ I
- If A survives each I nearest $\to$ A (infected) with probability β

In the mean field approximation, the average number of sites $< n_a >$ which can be infected by a single A is given by:

$$< n_a >= (1 - \gamma)\,(2d\beta + 1) \tag{5}$$

In order for the epidemic to spread the requirement is that $< n_a > \geq 1$, therefore the condition for critical threshol is:

$$\gamma = \frac{2d\beta}{1 + 2d\beta} \tag{6}$$

.

Such a generic cellular automata model [11] simulates some of the observed behavior in the spreading of infectious diseases, showing a sharp transition in the

number of "active elements" (infectives) when a certain probability of infection is reached. An example of this type of "phase transition" having a sharp breakpoint after a certain critical number is shown in the data inset. We can hence define an order parameter, where after a critical probability or critical size in population as the case may be for the data, we get an epidemic.

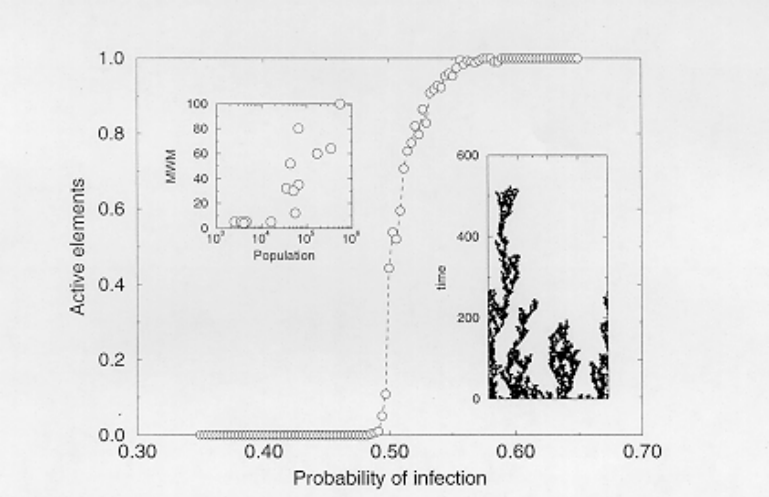

Figure 8: A one dimensional lattice of N=150 automata is used with a random initial condition. The decay probability of an active is 0.5. After T=250 times steps the propagation is checked. By repeating this process the probability of propagation is calculated for different infection rates. At the critical point, long transients are observed and the left inset shows fractal-like trees of propagating particles [11].

5 Self-Similarity and The Species Area Relationship (SAR)

The species area relationship (SAR) is of significant interest in ecology. Understanding how species are distributed could give information on the richness of species diversity and also discover the danger of extinction [14]. There has been debate in particular on weather SAR is log-normal or follows a power law. Much recent evidence shows that the power law Arrhenius [8] discovered[4] discovered is currently the more likely candidate. This law has been derived through a very general means simply by assuming scale invariance in the SAR. This assumption turns out to predict a number of interesting observed ecological patterns much different from a log-normal distribution [16].

The so called Harte [16] derivation of the SAR makes first the fundamental assumption that the fraction of species found in a patch of area A, that are also

[4] $S \sim A^z$, as previously introduced

found on a patch $A/2$ are independant of A. Let us now examine this derivation to make more explicit how the power law form is obtained.

First we start with an area A_0 in which there are S_0 species. The number of individuals in each species is given by the probability distribution $P_0(n)$ where $P_0(n)S_0$ is then the number of species with n individuals. A_0 is taken such that its $length/width = \sqrt{2}$, so that repetitive bisections of the long side leave similar rectangles. Let $A_i = A_0/2^i$, the area of each rectangle after the ith bisection and S_i, the number of species found, on average, in A_i. The self-similarity is imposed as follows:

Consider if a species is known to be in A_i

- the probability of it being found in one of the two resulting A_{i+1} recatangles is a constant, **a**, independant of i and therefore independant of spatial scale. One can equally think of 1- **a** as being the probability of not being in the right or left halves.
- This then results in the three possibilities
probability	**rectangle**
1. 1 - **a** :P_r	only "right" half
2. 1 - **a** :P_l	only "left" half
3. 1- P_r - P_l = 2**a** - 1	both halves
4. (where P_{lr} denotes the probability of left,right halves respectively)	

and we require that $0.5 \leq \mathbf{a} \geq 1$, since 0.5 would correspond to every individual belonging to a different species and 1 corresponds to one species being found everywhere. Repeating the bisections, the average number or species found in any particular A_i is :

$$S_i = \mathbf{a}^i S_0 \tag{7}$$

and we define the exponent z by $\mathbf{a} = 2^{-z}$ and by equation (7) it follows that $S_i/Sj = \mathbf{a}^{i-j}$. After this we can easily write $(S_i/S_j) = (A_i/A_j)^z$, from which follows $S_i = cA^i$, the power-law form of the SAR. Now we seek to relate the exponet to observables, namely the probability distribution $P_0(n)$. By introducing the notion of the smallest patch size A_m, which is to contain on average one individual, so that $A_m = A_0/2^m$, where the mean number of individuals in A_0 is $N_0 = 2^m$. From this the following recursion relationship is obtained, where we identify $x = 2(1 - \mathbf{a})$:

$$P_i(n) = xP_{i+1}(n) + (1-x)\sum_{k=1}^{n-1} P_{i+1}(n-k)P_{k+1}(k)$$

where solutions must be obtained numerically for high values of i, n and x. When plotted for high values we see that $P_0(n) \sim n^{c(x)}$, where now we have an $x = 2(1-\mathbf{a})$

dependance in $c(x)$ which we can compare to z since $\mathbf{a} = 2^{-z}$. The predicted species abundance distribution should then have a form as we see in the following graph:

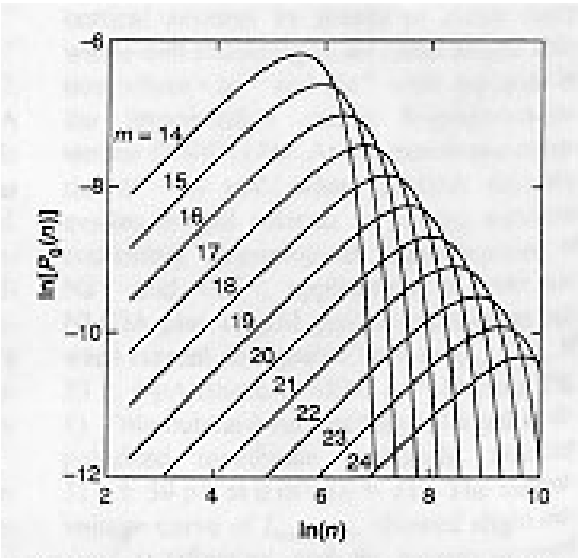

Figure 9: Species-abundance distributions $\log P_0(n)$ vs $\log n$ for a particular x value and varying m , corresponding to variation in the total number of individuals.

This gives then a powerful framework with which to work where these problems can be treated with such generality. More work has been done on this and in particlar finite size scaling techniques have been applied by Banavar and Maritan et al [17] where the probability distribution is taken to be of the form:

$$P_i(n) = \frac{1}{n} f\left(\frac{n}{N_i^{\phi}}\right) \tag{9}$$

where n is again the number of inividuals in a given species in A_i and N_i is the finite size of the population, f is the same scaling function within a universality class and ϕ is the crossover exponent which changes the function from power-law to rapidly decreasing at $f(1)$. This is very useful since all populations are of course finite and would enable their comparison and test for data collapse when finite size is corrected. This could then characterize them all in the same universality class. As mentioned before this approach has had some success in predicting the expected exponents for certain mainland species popualtions , but has deviated significantly for others. M. Rosenzweig [14] suggests that this may be due to fundamentally different mechanisms for diversity on different scales contrasting island speciation from mainland, where the processes are different and the Harte model has appealed to mostly mainland speciation. Nevertheless this approach presents an interesting framework, developed in the study of critical phenomena, which shows promise to deal with many problems in ecology.

6 Conclusion

We have seen that many otherwise perhaps complex problems in population biology can be approached and to some extent understood from the formalism developed in critical phenomena and extended to dynamical systems. The properties of these systems which exhibit such power law scaling and display order may show that these systems are not so different on the appropriate scale.

What is also evidenced are the shortcomings of these approaches or lack or others from the problems that remain to be understood and the amount of inconsistency. The SAR power laws have been good for many situations, but perhaps can find improvement by appreciating their limitation sand being able to appropriate them as Rosenzweig suggests [14]. The SOC have also the limitation in that we cannot with confidence yet say that any system is trult self organized critical since the models themselves have parameters pretuned to allow criticality. In fact, Sole and Bak et al [11] mention that measels are known to display low-dimensional chaos in large popualtions, therefore suggesting that in the isolated population studied by Rhodes and Anderson [2], a relevant parameter to allow critical behavior must have been tuned.

This then becomes a fundamental question in dealing with population problems : what are the relevant parameters that cause the system to be critical? What is it that creates systems like this with long range correlations displaying power-law behavior. One might humbly opine that this is what needs to be understood in order to predict such behavior, although it may be a daunting task to do so. From a biological point of view one might be interested in investigating if all biological systems are in such a detailed balance where many external parameters may tune them to criticality.

In this sense the tools of critical phenomena may provide a promising way, in order to understand the systems on the appropriate scale which may be independant of the microscopic properies. It may even be in our future to find complementary answers from these systems and inanimate ones in being able to "understand" universality [13].

References

[1] Anderson, R.M. and May, R.M. 1991. *Infectious diseases and control.* Oxford University Press, Oxford.

[2] Rhodes, C.J. and Anderson, R.M. *Power laws governing epidemics in isolated populations.* Nature **381**,600-602 (1996).

[3] B. Drossel and F. Schwabl. Phys. Rev Lett. **69**,1629 (1992).

[4] S. Clar, B. Drossel and F.Schwabl. Phys. Rev E **50**, 1009(1994)

[5] P. Bak, C. Tang, K. Wiesenfeld. Phys. Rev. A **38**,3645 (1988)

[6] J.D. Murray, *Mathematical Biology*, (Springer-Verlag, N.Y., 1993).

[7] T.H. Keitt and P.A. Marquet. J. Theor. Biology (1996) **182**, 161-167.

[8] O. Arrhenius. J. Ecology **9**,95 (1921).

[9] O. Miramontes, *Order-Disorder Transitions in the Behavior of Ant Societies*, with the accompanying website: http://scifunam.ifisicacu.unam.mx/mir/ord-diso.html.

[10] The Sandpile Model, Forrest Fire Model and Earthquake model,(references)

[11] R.V. Sole, Per Bak et al. *Criticality and Scaling in Evolutionary ecology.* submitted to Trends in Ecology and Evolution.

[12] Goldenfeld, N.D.. *Phase Transitions and The Renormalization Group.* Pereus Books, 1992.

[13] H.E. Stanley. Rev. Mod. Phys., **71**, No.2, 1999.

[14] M. Rosenzweig. Science, **284**, 5412, 2000.

[15] T.H. Keitt and H.E. Stanley. *Dynamics of North American Breeding Bird Populations*, December 12, 1997.

[16] J. Harte, A. Kinzig and J.L. Green. Science, **284**, 334, 1999.

[17] J.R. Banavar, Amos Maritan et al. Phys. Rev. Lett., **83**, 4212, 1999.