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Food Signal Theory: Population Regulation and the Functional Response

by

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Food Signal Theory: Population Regulation and the Functional Response

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ABSTRACT

A fully stochastic food-signal model, a function of a patchy-prey encounter sequence, and a prey-processing function is described. The model shows how prey density and its second-order statistical properties can sequester prey from predators questioning the use of only numerical abundance of predator and prey organisms as a measure of prey-predator interactions. The model highlights the notion that patch structure can be generated by relative velocity of predator and prey as well as by their spatial distribution. The model extends ideas that include the "biological pump" and the downwelling of carbon from the upper ocean, the functional response, optimal-foraging theory, and the connections between population dynamics and variability in the physical environment.

INTRODUCTION

A mathematical model of the population-dynamics regulatory process accounting for density dependence, the interactions among populations, population age-structure, and the way that these phenomena are driven by the physical environment is difficult to

construct (for an early discussion see Paulik 1973; for the general level of the "state of the art" see Nisbet and Gurney 1982; and Getz and Haight 1989).

Conceptual models, however, are more easily articulated. A conceptual model that considers each life-history stage as a non-linear density-dependent module (Figure 1) evincing the population-stabilizing "struggle" between density-enhancing and density-depressing mechanisms can be found in Rothschild (1986: Chapter 8). The population stabilizing mechanisms are essentially trophodynamic. The mechanisms operate because in general high predation levels reduce prey abundance while high prey levels enhance predator abundance. (The terms "predator" and "prey" are intended to be taken generally, i.e., predators can include grazers, etc.)

That a component of predator-population stability owes to the effect of the predator population on prey density, means that prey-population abundance contains <u>information</u> on predator-population abundance. This information contributes to the predator-population regulation.

Because prey-predator interactions involve the transfer of both energy and information and because the information transfer can be quantified by the time points when individual prey are ingested, the temporal distribution of ingestions can be thought of as a <u>food signal</u> (Rothschild 1986, 1988; Rothschild et al. 1989). The idea of a food signal is closely analogous to the application of queuing theory to predator-prey relationships pioneered by Beyer

(e.g. 1980).

How food-signal accuracy (i.e. bias and precision) is affected by sources of variability external to the simple relative numerical abundance of predator and prey is a key consideration. For example, the functional response and physical forcing are well-known modifications of food-signal accuracy. The functional response (see general review by Taylor, 1954) where ingestion of prey declines as the density of prey increases has been studied primarily in grazers. Frost (1985) and Costello et al. (1990) demonstrate some of the problems in evaluating grazer functional response while Marasé et al (1990) show how physical forcing can affect food signals.

This paper develops the theory of food signals which in terms of population regulation, are defined by the arrows entering and leaving the rectangular box in Figure 1. The theory is developed by considering a food-signal model and the specification of an encounter signal and a processing function.

A FOOD SIGNAL MODEL

A food-signal model has three components: 1) the input or encounter signal, 2) the processing function, and 3) the output or food signal itself.

The model is based on the idea that prey are encountered by the predator as it "swims" along a search filament (see e.g. Blaxter and Staines, 1971). The three-dimensional existence of the predator and prey, can be considered in a single dimension (Figure

2; see Rothschild, 1988). Because both predator and prey are often motile, the points in time when the predator encounters prey can be recorded in terms of the relative velocity of predator and prey, as distinct from the absolute velocity of either (Gerritsen and Strickler 1977). However, the predator does not in general ingest every encountered prey because of functional-response phenomena such as predator "satiation" causing food-signal accuracy to deteriorate. The rules for whether or not a prey is ingested, transform the encounter signal into a food signal which conveys predator-abundance information to the predator.

The first component, the encounter signal, is based on a record of encounter times. The initial encounter time ξ_0, occurs at the origin. The time of the initial encounter, and the times of subsequent encounters up until the N-th encounter are,

$$\Xi = (\xi_0, \xi_1, \ldots, \xi_i, \ldots \xi_N) \tag{1}$$

A sequence of inter-encounter time intervals is derived from Eq(1),

$$Z = (\xi_1-\xi_0=Z_1,\ \xi_2-\xi_1=Z_2, \ldots,\ \xi_i-\xi_{i-1}=Z_i, \ldots, \xi_N-\xi_{N-1}=Z_N), \tag{2}$$

where the Z_i's are random variables, and N represents the number of prey encountered during a particular realization, Z.

The transformation of the encounter signal Ξ into a food signal, is developed from the concept of the functional response. The idea is that once a predator ingests a prey, then the predator enters a <u>refractory period</u>; during the refractory period the predator cannot ingest another prey until the refractory period is

terminated.

The ingestion or food signal is,

$$\Xi' = (\xi'_0, \xi'_1, \ldots, \xi'_j, \ldots, \xi'_n), \tag{3}$$

where n is the number of prey ingested. The ingestion times, the ξ''s in Eq(3), are a subset of encounter times, the ξ's in Eq(1). The prime denotes only instants of time when prey are ingested given that these times are times when prey are encountered. Note, that for each ξ'_j; there must be an identical ξ_i, but for each ξ_i there is not necessarily an ξ'_j. Comparison of Eq(3) with Eq(1) shows that while N prey are encountered, only n prey are ingested. If there were no functional response, then the predator would ingest all N encountered prey (N=n). The functional response implies N>n.

Having identified the time points when each prey is ingested enables identification of the position and length of each refractory period following each ingestion. At each instant that the j-th prey is ingested, ξ'_j, the predator becomes refractory for a random period of time, Y_j. Thus, there is a sequence of time intervals when the predator cannot ingest a prey because the predator is in a refractory state. For the j-th ingestion, this period occupies the interval, $\xi'_j + Y_j$.

A sequence of actual inter-ingestion times can be constructed analogous to the sequence of inter-encounter times represented by Eq(2),

$$X = [\xi_1' - \xi_0' = X_1, \xi_2' - \xi_1' = X_2, \ldots, \xi_j' - \xi_{j-1}' = X_j, \ldots, \xi_n' - \xi_{n-1}' = X_n]. \qquad (4)$$

Hence, the food-signal model transforms an encounter signal into a food signal. The transformation involves three random sequences,

$$\begin{aligned} S_1 &= \{Z_i\} \\ S_2 &= \{Y_j\} \\ S_3 &= \{X_j\}, \end{aligned} \qquad (5)$$

where S_1 is the encounter or patchy input food signal sequence, S_2 is the processing sequence, and S_3 is the food signal sequence. In a way, the discrepancy between S_1 and S_3 must be thought of as statistical "error".

The linkage between S_3 and S_1 (in terms of time intervals) or N and n (in terms of a counting measure) is developed through renewal theory which requires that the time intervals in S_1 are independent and identically distributed (iid). Explicit expressions for the S_3 requires specifying the statistical form of S_1 and S_2.

The renewal-theory formulation is identical to that of electronic counter problems studied in the theory of point processes (e.g. Feller 1957). The examples in this paper are derivations, or based upon derivations, which can be found in Cox (1962) and in Cox and Isham (1980).

SPECIFYING THE ENCOUNTER SIGNAL: THE PATCHY INPUT SEQUENCE

The first problem is to specify the probability distribution of the encounter signal, S_1. There are several classes of probability distributions. One simple classification is that the encounter signal can be thought of as either "random" or "patchy". Empirical studies reflect that a random distribution of prey is unlikely, although in many instances the scales which have been studied are larger than the feeding scale.

While models of patchy distributions are more realistic, they are generally more difficult to specify and analyze than distributions associated with a Poisson or random process. Patchy probability density functions include generalized and compound distributions (see e.g. Pielou 1977:113-123). Particular applications have been reviewed by Fasham (1978). The basic point is that statistically indistinguishable patchy sequences S_1, can be generated by very different mechanisms (e.g. with-after-effect or without-after-effect; see Feller 1957). While the subject of the mechanisms that actually drive patchiness is critically important, it is beyond the scope of this paper, as we are essentially considering the behavior of the predator relative to a patchy distribution of prey, regardless of the mechanism that generates the patchiness.

Keeping this in mind, we use the simplest patchy extension of the Poisson process, the mixed exponential distribution, with probability density function,

$$f_z(t) = \theta\rho_1 e^{-\rho_1 t} + \beta\rho_2 e^{-\rho_2 t}, \tag{6}$$

$$\beta = 1-\theta.$$

The mixed exponential distribution has a coefficient of variation which ranges from $1 \to +\infty$ and hence can represent extensive patchiness. Note that Eq(6) also contains the Poisson process as a special case (i.e. $\rho_1=\rho_2$ or $\theta=1$), and also that Eq (6) can be generalized to contain more than two exponential components.

With regard to the moments of Eq(6), its mean can be expressed as,

$$\begin{aligned} \mu_z = E(Z) &= \int_0^\infty t f_z(t)\,dt \\ &= \theta\frac{1}{\rho_1} + \beta\frac{1}{\rho_2}, \end{aligned} \tag{7}$$

Considering $\rho_1 \gg \rho_2$ implies that the inter-encounter times associated with ρ_1 are less than those associated with ρ_2. Hence there would be a greater number of encounters associated with ρ_1. The variance of Eq(6) is,

$$\begin{aligned} \sigma_z^2 &= E(Z^2) - \mu_z^2 \\ &= \int_0^\infty t^2 f_z(t)\,dt - \mu_z^2 \\ &= \frac{2\theta-\theta^2}{\rho_1^2} + \frac{2\beta-\beta^2}{\rho_2^2} - \frac{2\theta\beta}{\rho_1\rho_2} \end{aligned} \tag{8}$$

Note again that if $\theta=1$, Eq(6), Eq(7), and Eq(8) represent the density function, the mean, and the variance, respectively of the inter-arrival times of the exponential, random, or non-patchy

distribution of the Poisson process.

In contrast to the Poisson process or the random distribution case, the inter-encounter times of prey are not uniquely determined by a single parameter. In Eq (6) different values of the parameter set $\Omega=(\rho_1,\rho_2,\theta)$ can result in the same value for μ_z. In the patchy case, μ_z, contains a richer set of information than a corresponding parameter in the random case.

Because the <u>numbers</u> of prey rather than <u>time intervals</u> between prey are of interest, the renewal function is used to calculate the mean number of prey encountered up until a point in time t, from the inter-encounter times specified in Eq (2). The renewal function is defined as,

$$H_z(t) = E(N_t), \tag{9}$$

where H_z is the number of prey encountered between 0 and t, and E is the expectation operator. In other words the encounter signal can be viewed as a series of pulses. The renewal function provides the mean number of pulses that occur during a time interval.

For the ordinary renewal process, the counting measure associated with the renewal function, Eq (9), is derived from its Laplace transform which incorporates the Laplace transform of Eq (6). Cox incorporates the Laplace transform (denoted by an asterisk) of Eq (6),

$$f_z^*(s) = \frac{\theta\rho_1}{\rho_1+s} + \frac{\beta\rho_2}{\rho_2+s}, \tag{10}$$

into the Laplace transform of the counting measure,

$$H_z^*(s) = \frac{1}{s^2\mu} + \frac{1}{s} \cdot \frac{\sigma^2-\mu^2}{2\mu^2} - \frac{\theta\beta(\rho_1-\rho_2)^2}{[\beta\rho_1+\theta\rho_2]^2[s+\beta\rho_1+\theta\rho_2]}, \tag{11}$$

to yield upon inversion,

$$H_z(t) = \frac{t}{\mu_z} + \frac{\sigma_z^2-\mu_z^2}{2\mu_z^2} - \frac{\theta\beta(\rho_1-\rho_2)^2}{(\beta\rho_1+\theta\rho_2)^2}\exp\{[-\beta\rho_1-\theta\rho_2]\,t\}. \tag{12}$$

The mean number of prey encountered by the predator up until, t. For the Poisson process the second term disappears and for large t the last term disappears.

SPECIFYING THE ENCOUNTER-SIGNAL PROCESSING FUNCTION: THE REFRACTORY TIME

From a biological point of view, the mechanisms that cause the refractory time to be long or short are not well understood, and obviously relate to a complex of mechanisms (Costello et al. 1990) that extend beyond "satiation." The simplest first-approximation approach is to assume that the mean refractory time is exponentially distributed, an assumption analogous to those frequently made in queuing theory. Accordingly, consider the refractory-time probability density function to be,

$$f_y(t) = \alpha e^{-\alpha t}. \tag{13}$$

It is important to recognize that Eq(13) implies that the refractory time is "stationary" for all ingestions. Thus Eq(13) is taken to mean that the refractory time is related to the "processing" or "handling" of the prey which will not change as a

function of the number of prey "handlings". If on the other hand, the predator expedites prey handling time as a function of its experience in handling prey, or if the predator becomes satiated, such that the refractory-time depends on the number of ingestions then, Eq(13) will need to be generalized. Because the temporal distribution of arrivals at the point of ingestion varies with the mode of feeding (e.g. contrast grazing copepods and fish larvae) more research on the processing function is required.

DERIVATION OF THE FOOD SIGNAL: THE OUTPUT PROCESS

The food signal or the output process, S_3, is a function of the encounter signal or the input process, S_1, and the refractory time, S_2. The food signal is derived from the calculus explained by Cox and Isham (1980). Using their approach we can write the mean waiting time between ingestions, given a fixed refractory time (Y=y) as,

$$E(X|Y=y) = \mu_z\{1+H_z(y)\}, \qquad (14)$$

because $H_z(y)$ is the mean number of prey (cf Eq(9)) encountered in a refractory interval of specific length (o,y] that are not ingested (by definition), and we multiply this mean number by the mean inter-encounter time of those individuals not ingested, which then must be added to the mean inter-encounter time to obtain the inter-ingestion time.

Averaging Eq(14) over the specific refractory times yields,

$$\mu_x = E(X) = \mu_z\{1+\int_0^\infty H_z(y)\,[f_y(y)]\,dy\}. \tag{15}$$

The integral in Eq(15) using Eq(9) and Eq(13) is evaluated in three parts to obtain,

$$\mu_x = E(x) = \mu_z\{1+\{\frac{1}{\alpha\mu_z}+\frac{\sigma_z^2-\mu_z^2}{2\mu_z} - \frac{\alpha\theta\beta(\rho_1-\rho_2)^2}{(\beta\rho_1+\theta\rho_2)^2(\beta\rho_1+\theta\rho_2+\alpha)}\}\} \tag{16}$$

So now the mean time between ingestions, μ_x, can be linked with the mean time between encounters, μ_z, which is one way of defining a functional response. We can see that the longer the refractory time, the longer the waiting time between ingestions, and when the variance is much larger than the mean, then the waiting time becomes even longer, suggesting that the intensity of clustering sequesters the prey from predator.

We note that to develop the counting process explicitly we would not take the shortcut implied by our use of Eq(14), rather we would explicitly derive the probability density function $f_x(t)$, then use the Laplace transform of the density function in Eq(6) which would be inverted to obtain $H_x(t)$.

For the shortcut, we use the asymptotically normal version of the counting process,

$$H_x(t) = \frac{t}{\mu_x} \tag{17}$$

so for any t, and set of parameters ρ_1, ρ_2, θ, and α, we can compare the encounter rates given by $t\mu_z^{-1}$ and the ingestion rates $t\mu_x^{-1}$ which give the functional response for a fixed

parameter set Ω and α.

Put another way we could derive the instantaneous mortality rate, M, per unit time (valid for large t), for any fixed set of the above parameter values by writing the mean ingestions per contact,

$$\frac{H_x(t)}{H_z(t)} = \frac{\mu_x}{\mu_z} = 1-e^{-Mt} \tag{18}$$

Hence for a specified unit time we have,

$$M = -\log\left(1-\frac{\mu_x}{\mu_z}\right) \tag{19}$$

This component of the mortality rate is then a function of prey patchiness and predator refractory time. Therefore, we can consider the mortality rate of prey owing to predation as a function of not only the mean abundance of prey, but as a function of the mean abundance of prey, prey patchiness, and predator refractory time. This provides an opportunity to link this complex of trophodynamic relationships with the physical environment.

The food signal model represented by Eq(16) is more general than those presented previously. It can be seen that μ_x is particularly sensitive to the magnitude of α. Also patchiness given some μ_z and α causes a decrease in ingestion rate as σ_z^2 increases. In fact, special cases of the relationship can be cast in a format that is analogous to the classic functional response calculations. To do this it is necessary to constrain the parameter set. One constraining convention would be to

compare the approximate ingestion rate in Eq(17) with the approximate mean encounter rate in Eq(12), where some of the parameters are fixed. For example we might wish to examine how the ingestion rate varies with encounter rate given a fixed ratio between ρ_1 and ρ_2 for a fixed θ. An example of these calculations are shown in Figure 3 for two cases:

$$\begin{aligned} &\textit{Case I;}\ \rho_1^{-1} = \rho_2^{-1}, \theta = .1 \\ &\textit{Case II;}\ \rho_1^{-1} = 10\rho_2^{-1}, \theta = .1 \end{aligned} \tag{20}$$

Case I is identical to the random Poisson process while in Case II ρ_2^{-1} is "large" while θ is "small", a condition generating a relatively large variance or patchiness. We can see that in both cases, we have a typical functional response, but in the patchy case, ingestion is reduced by a factor of roughly 25 percent per unit time. Of course some parameter sets will minimize the patchy effect while others will increase it.

Comparison of plots of H_x versus H_z with functional responses reported in the literature is interesting. For example Frost has reported in a series of papers (e.g. 1985) the relation between the ingestion of phytoplanktonic unicells by Pseudocalanus sp. as a function of food concentration. His experimental results are partially comparable to our theory in the sense that his Ivlev curve has an asymptote of 1348 cells ingested per hour. This corresponds to α=1348. Accordingly we can compute the functional response $H_x=f(H_z)$ for Ω and α=1348 for the Poisson process case. Comparison of Frost's experiments based upon food concentration

(cells ml^{-1}) with the food signal theory based upon number of "cells" encountered can be enlightening.

DISCUSSION

The variability and stability conferred upon populations by the population-dynamics process can be placed in the context of food signals which have the dual function of transmitting biological energy and information on the abundance of predator populations. Food signal theory also ties together and extends ideas on the functional response, optimal foraging theory, physical-biological interactions, and the "biological pump".

The primary idea that results from food-signal theory is that the statistical nature of the input sequence (as summarized by Ω) and the processing function contribute to regulation in the population-dynamics process. The accuracy of this regulation can be measured in terms of food signals which reflect both patch structure <u>and</u> relative velocity of predator and prey as induced by motility and turbulent flow.

The food-signal framework allows consideration of "new" kinds of non-spatial patch generating mechanisms which may exhibit trophodynamic effects indistinguishable from spatial patchiness. For example temporal patchiness can be induced by patchy relative velocities of prey relative to predators, and as a consequence, the mean density of prey perceived by a predator has components which owe not only to prey relative density, but to the relative velocity of the prey and possibly the effect of

small scale turbulence as well (Gerritsen and Strickler, 1977; Rothschild and Osborn, 1988; Costello et al. 1990; and Marasé et al. 1990). The velocity patches which can have effects indistinguishable from the conventional spatial patches, and are easily parameterized in terms of ρ_i in Eq(6), for example, are generally not measured or taken into account. As a second example, non-spatial patchiness may arise from the "mix" of prey. A predator might be confronted with two species of prey with relative densities or velocities measured in terms of ρ_1/ρ_2 and relative abundances measured by θ (a situation which could be generalized to a large number of species). Inasmuch as it seems unlikely that "real" prey of any life-history stage are likely to be homogeneous with respect to ρ and θ, patchiness as the result of multiple-species prey almost seems guaranteed.

Relative to various theories, food-signal theory is intimately related to functional-response and optimal-foraging theories. With respect to functional-response theory, most of its articulations, implicitly or explicitly assume that prey are distributed at random with respect to the predator (e.g. Holling's (1965) classic work). However, most distributions of organisms in the ocean are not random but, "patchy"; ("patchy" means that the spatial distribution of organisms cannot be described by either a uniform or a Poisson statistical process). The patchiness of organisms at small scales is well known (e.g. Cassie 1959; Owen 1989). The notions of the functional response are extended from a random to patchy setting by food-signal

theory. Holling's result is just a special case of the more general food-signal theory (cf. Mangel and Clark, 1988). With respect to optimal-foraging theory, it tends to concentrate on predatory, rather than prey behaviour, failing to take into account the schizoid predator-prey nature of every organism. The food-signal notion shows how patchiness sequesters prey from predation. Since prey want to minimize their "nearest neighbor distance" (to avoid predation) and predators want to maximize their "nearest neighbor distance" (to minimize competition for food) and since all organisms are both predator and prey; an optimal or equilibrium spacing among organisms must exist. This spacing must change as a function of the physical environment, showing directly how the physical environment modulates the metabolism of the marine community. The patchy "behavior" of prey is clearly a mechanism that serves to avoid predation but is often not considered (see Taylor 1984,; also papers by Milinski and Heller 1978 and discussion by Mangel and Clark 1988).

The food signal can also facilitate the detailed study of how the physical environment affects population regulation. This is because the food signal definition is cast in fundamental terms (e.g. the waiting times between prey) so in principle it should be easier to show how the food signal is affected by temperature, than to show a similar effect with a more highly aggregated variable such as mortality rate. The study by Rothschild and Osborn showed that encounter rate is a function of the relative velocity of predator and prey where components of

velocity owed to predator/prey mobility and homogeneous-and-isotropic turbulence. The food signal provides a linkage between the population-dynamics process and the Rothschild-Osborn conjecture in that it shows how the variance in the encounter signal affects ingestion rate and thereby sets the stage for further study of the specific ways that the physical environment affects the parameters of the food signal. We can see how the physical environment might affect the signal in terms of θ, ρ, and α and thereby affect many aspects of the relation between predator and prey. This effect may be expressed in two modalities; the first (Type I effect) involves factors in the physical environment that primarily affect the physiology of the predator and the prey, the second involves factors in the physical environment that do not particularly involve the physiology or behaviour of the predator and prey, but rather affect the transmission medium of the food signal. Temperature is an example of a factor that affects the physiology or behaviour of predator and prey. Any change in temperature relative to a reference level may cause aspects of predator and prey physiology to move in the same or in opposite directions thereby causing variability in the food signal. The second (Type II effect) involves factors that affect the transmission medium. The extent to which turbulent flow affects patchiness depends of course on the scale of turbulent eddies or residual shear relative to parameter such as ρ and θ.

Finally, the notion of food signals is important in viewing

basin-scale biological variability that might be associated with "global changes." For example the "biological pump" (Longhurst and Harrison, 1989) is thought to account for significant variability in downward carbon flux. The magnitude of the flux is dependent upon the extent of grazing. But the extent of grazing is generally thought of in terms of the relative abundance of grazers and phytoplankton cells. As can be seen from Figure 3, phenomena that control spatial and velocity patchiness can be an important source of variability in the downwelling of carbon even if the number of grazers and algal cells is fixed.

What is the significance of this theoretical analysis vis à vis views on ecosystem structure? The analysis of this question really does not depend upon the food signals, per se, rather it depends upon the way that the signals influence both predator and prey population dynamics and hence the ecosystem as a whole. Empirical results may seem counterintuitive because of the inherent nonlinearity of the population-dynamics process; small trophodynamic effects could have large population-dynamic effects, while large trophodynamic effects could have small population-dynamic effects (are θ=.1 or ρ_1/ρ_2=10 large or small?). Empirical results may also be surprising in the sense that seemingly minuscule changes in the average food signal could generate observable changes in the dynamical or statistical properties of population abundance. In addition it may be that in particular instances, concern is neither with average values,

nor statistical distributions, particularly of past events, but with the establishment of conditions that generate unusual population fluctuations.

The food signal in the context of the population-dynamics process, provides an alternative way to think about marine ecosystems. The traditional approach is to consider the relations among highly aggregated taxa or size groups. The traditional approach has the advantage of being a rather simplistic metaphor of the structure of the marine ecosystem. On the other hand, these metaphors constrain the enhancement of fundamental understanding because they obliterate the very factors that are so important in population regulation: the factors that generate variability, and the factors that maintain stability. The notion of the food signal implies that general knowledge of ecosystem behaviour can be enhanced by the study of a small subset of related populations and their food-signal interconnections. It would of course be difficult to study the "entire" ecosystem in any detail, rather generalities would emerge that reflected upon the nature of system variability in that this alternative approach would lend itself to studying the extent to which perturbations are either propagated or damped among interconnected population-dynamics process models.

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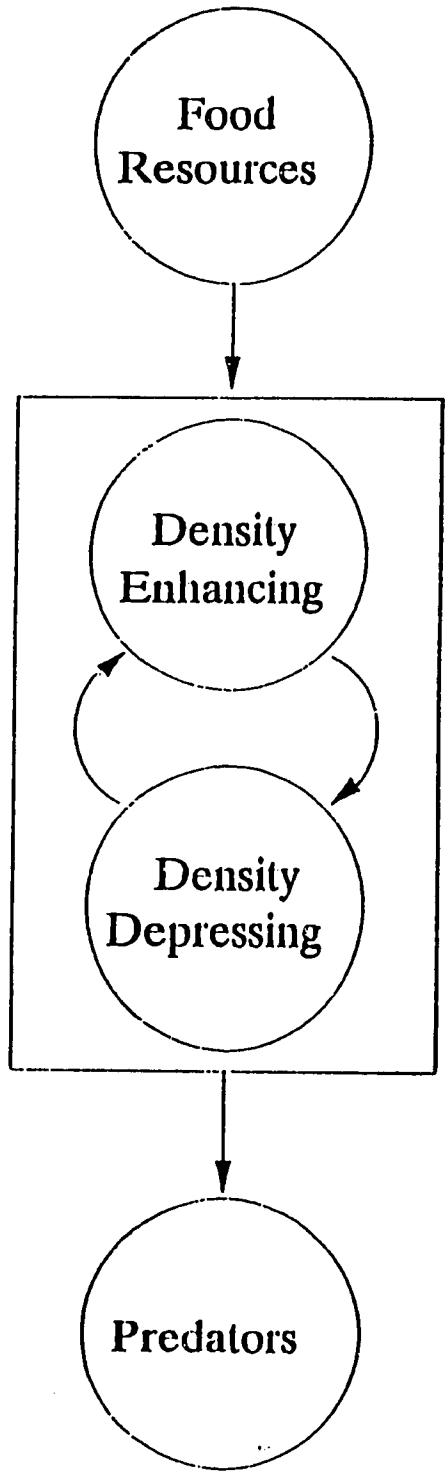

Figure 1. Nonlinear population regulatory module. The arrows exiting and entering the rectangular box are defined as signals (based on Rothschild, 1986).

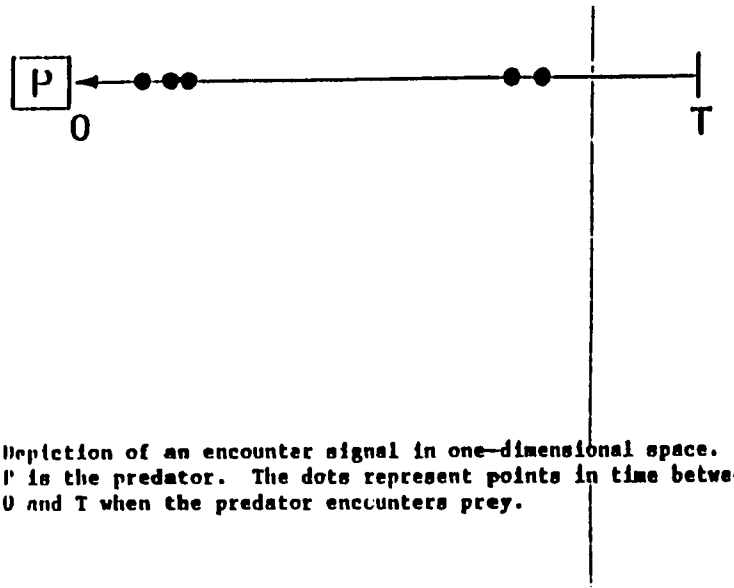

Figure 2. Depiction of an encounter signal in one-dimensional space. P is the predator. The dots represent points in time between 0 and T when the predator encounters prey.

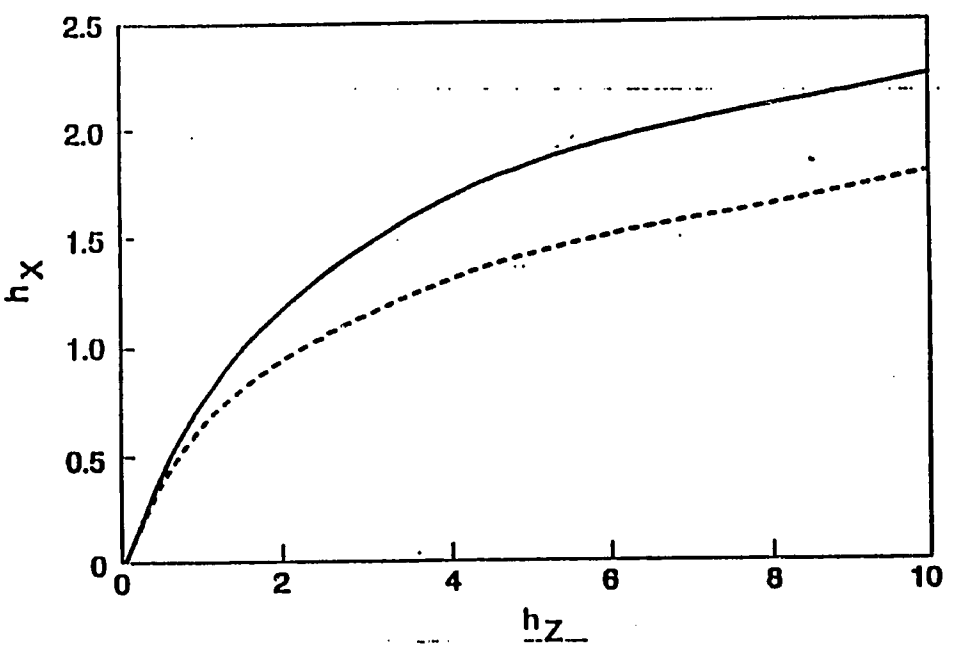

Figure 3. An example of the food-signal renewal function plotted as a function of the encounter-signal renewal function. The solid line is Case I and the dashed line is Case II.