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A NEW LOOK AT PREDATOR-PREY INTERACTIONS USING A SIMPLE ENZYME KINETIC MODEL

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[Author's note: In 1993 the Alaska Department of Fish and Game undertook a research planning effort designed to guide research for the next two decades. Integral to this process was the establishment of a predator-prey specialist group. I prepared the following essay to stimulate introspection and thought by this group. I offer it to this symposium for the same reasons.]

Abstract: Modern study of predator-prey interactions continues to produce increasingly sophisticated mathematical and conceptual models. Some of these models are purely descriptive, while some aspire to analytical and predictive functions. The obvious complexity of these models further mystifies the seldom-observed biological phenomenon we call predation. Further mystification increases the risk of distancing predator-prey biology from the practical mainstream of wildlife management resulting in compromised benefits for wildlife users. In an effort to demystify predator-prey biology, predator-prey systems may be considered in the framework of classic enzyme-catalyzed biochemical reactions. Approaching predator-prey study from this perspective suggests classic methods used by wildlife researchers, while productive in describing the process of predation, may be misapplied in attempts to study predator-prey interactions at the ecosystem level. If so, it follows that progressive development of increasingly complex iterative models may well be a distracting result of inappropriate research methodology. It may be time to reexamine methodology and seek a new perspective. This paper offers that opportunity.

I freely admit to being a relative 'outsider' to predator research. However, throughout the history of science, 'outsiders' have effected interdisciplinary transfers of perspective which have accelerated progress. To this end, I suggest review of the well-developed field of enzyme kinetics (to which I am also an 'outsider') may be helpful in predation research. The empirical findings and mathematical models commonly applied in the study of enzyme kinetics may satisfactorily quantify and explain much of what we have learned in past studies of predator-prey biology. In fact, much of the data recently cast as new predator-prey theory is more simply and readily understood in the terms of simple enzyme kinetic models than the complicated multiple equilibrium models which have attained preeminence in predator research.

METHODS

To establish a basis of understanding, I shall review some concepts from basic chemistry:

Basic Chemical Reaction Theory

In uncatalyzed reactions chemical equilibrium is determined by what chemists refer to as the "Law of Mass Action." That is, the direction and rate of a reaction under defined conditions of temperature and pressure is determined by the relative concentrations of the reactants and products. A numerical constant (called the equilibrium constant, K_{eq}) is defined, according the Law of Mass Action, as the ratio of reactant concentrations on the left side of the equation to the concentrations of products on the right side of the equation for the reaction,

$$A + B \rightleftharpoons C + D, \text{ the}$$

$$K_{eq} = \frac{[A]\,[B]}{[C]\,[D]}.$$

Life and Enzymatically-Catalyzed Reactions

Living organisms are highly organized enzymatically-catalyzed chemical systems which capture and channel energy into maintaining the organization and function required for life. Failure to capture and suitably channel this energy results in decreased order (increased entropy). As organized metabolic systems become increasingly entropic, their function progressively deteriorates. When an organism becomes sufficiently disorganized, that entropy is unchecked, we describe the condition of the organism as "dead." Death results from failure of separate, but integrated, enzymatic systems to capture and channel energy into organization. It results either from progressive deterioration of necessary enzyme systems or from traumatic

disruption of the organ systems necessary to support the enzymatic systems that process energy.

The point of all this is that biological systems are enzymatically based from the molecular level upward. Hence, I hope to establish some credibility for consideration of enzyme kinetic models at higher levels of organization. Life, at the molecular, cellular, tissue, organ, organism, population, community, and ecosystem levels may be linked by threads common to the tapestry of organization and catalysis. If so, predation may be profitably considered within this framework.

Predators live by disrupting the supporting organ systems of their prey and hastening its enzymatic death. Once prey are dead, the energy in their highly organized systems is appropriated by the predator to maintain its organ-supported enzyme-catalyzed systems. The quest of life may be thought of as an ongoing effort to appropriate energy to maintain the highly organized, but extremely delicate enzyme systems which support metabolism.

Metabolism requires the necessary chemical reactions to function at sufficiently low energy levels that the required energy transformations do not disrupt the delicate and fragile structure required for life. This is accomplished by enzymatic catalysis. Enzymes work by organizing life's chemical reactants, called substrates (by binding and orienting them to increase the chances of fruitful molecular interactions at life-supporting temperatures) so that metabolic reactions can proceed at lower energy levels. Chemists refer to this as lowering the activation energy required to initiate a reaction.

Once the reaction is completed, the substrate has become the product; but the enzyme is unaltered. This produces the common generalized reaction for enzymatic catalysis:

$$E + S \rightleftharpoons \text{Enzyme-Substrate Complex} \rightleftharpoons E + P$$

(Where "E" is enzyme, "S" is substrate, and "P" is product)

Carnivores and ungulates (where our interest in predators and prey usually centers) are both homeothermic species groups. Consequently, I shall assume constant temperature conditions for all catalyzed reactions. At constant temperature conditions, and enzyme works at a given efficiency or rate. That is, the rate at which each enzyme molecule attaches to (or binds) substrate, facilitates conversion to product, and releases the product is constant. Hence, unless there is more substrate than each enzyme molecule can process, any changes of substrate concentration in an enzymatically catalyzed reaction will change the rate of the reaction. Put another way, until an enzyme's 'environment' is 'saturated' with substrate, the rate of the reaction it catalyzes will increase with increases in substrate concentration.

Because holding the amount of enzyme catalyzing a reaction constant and varying the amount of substrate available to it affects the rate of the reaction, reaction rates are slow at very low substrate concentrations. There are more enzyme molecules available than substrate molecules to convert to product. As the amount of substrate is increased, more of the available enzyme molecules are supplied with substrate and the rate of the reaction increases. If substrate concentration is further increased, eventually each enzyme molecular will be "substrate-saturated," and the reaction rate will plateau at maximum velocity. Enzymologists call this rate, V_{max}. This velocity will be limited only by the inherent "speed" of the enzyme itself.

Through the application of the Law of Mass Action and mathematical manipulation, a formula defining the numerical constant describing the inherent ability of an enzyme to work at standard conditions can be derived. I shall not go into the mathematical exercise here. It can be found in any elementary biochemistry text. This constant, called the Michaelis-Menten constant (K_m) after the biochemists who pioneered enzyme kinetics, has units of substrate concentration and is, as a general rule, half of V_{max} (Fig. 1). It is similar in concept to maximum sustainable yield being half of carrying capacity in wildlife carrying capacity theory.

Once there is enough substrate that every enzyme molecule is functioning at V_{max}, the rate of reaction can only be increased by increasing the number of enzyme molecules. A plot of reaction rate as a function of enzyme concentration when substrate is in excess is represented in Fig. 2.

In this situation, the rate of reaction can be controlled by altering the effective concentration of enzyme. Adding more enzyme molecules increases the rate; reducing the number of functional enzyme molecules reduces the rate. Because it is difficult to take enzyme molecules out of a reaction system, enzymologists interested in the kinetics of catalysis (analogous to population dynamics in wildlife biology) found ways to decrease the effective concentration of enzyme without varying the actual amount of enzyme present. This was done through introducing substances called enzyme inhibitors into the reaction medium.

Enzyme inhibitors come in two basic varieties, called "noncompetitive" and "competitive" inhibitors. Noncompetitive inhibitors reduce effective enzyme concentration by irreversibly "tying up" the reactive sites of individual enzyme molecules. Once these reactive sites are tied up, the enzyme no longer works. It is functionally "dead." Hence, the effective concentration

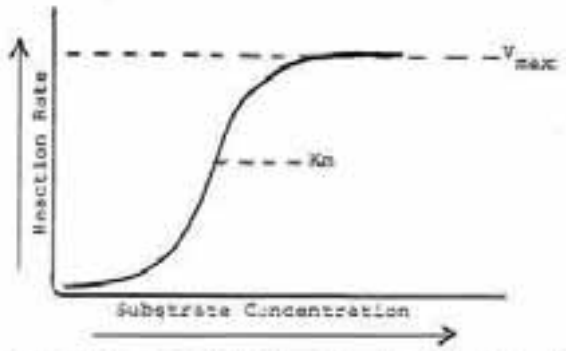

Figure 1. Enzyme catalyzed reaction rate as a function of substrate concentration with amount of enzyme held constant.

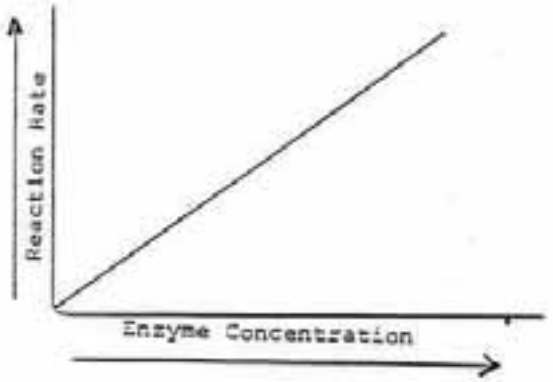

Figure 2. Enzyme catalyzed reaction rate as a function of enzyme concentration with substrate in excess.

is diminished; and the conversion of substrate to product slows.

Competitive inhibitors slow the rate of reaction by temporarily binding enzyme active sites in a reversible manner. The wildlife immobilizing agent, succinyl choline (used in early chemical wildlife capture) was a competitive inhibitor of sorts. It competed with the natural neural transmitter, acetyl choline, for enzymatic "space" at neural receptor sites. Acetyl and succinyl choline have the same chemical structure except that succinic acid is two carbon molecules longer than the acetic acid molecule in acetyl choline. Still, succinyl choline was structurally similar enough that it attached to neurotransmitter sites; it just didn't work as a neurotransmitter. Hence, nerve (and muscle) function were interrupted and animals couldn't use their muscles until the succinyl choline was metabolized and nerves (and muscles) returned to normal, acetyl choline mediated, function.

The kinetic mathematics of inhibited enzymatic systems have been well worked out in general, and for many specific competitive and noncompetitive enzyme inhibitors. I suggest these kinetics may be relevant to predator-prey dynamics.

RESULTS

What Does This Have to do With Predator-Prey Systems and Wildlife Management?

If we think of enzyme systems as analogs of predator-prey systems, some interesting comparison arise. Let us consider a simple wolf/moose system as a beginning. Remember that an enzyme mediates the rate of a reaction without being permanently altered. I suggest we think of wolf predation as an enzymatic system where moose are the substrate, wolves are the enzyme, and the product is wolf feces. That is, wolves may be thought of as catalyzing the conversion of moose to wolf scat.

Predators must defecate to live, and they must do so reasonably frequently. Hence, they must be pretty good at killing prey and converting it to feces. This means that unless predators are on the bitter edge of starvation they will pretty much "get theirs" from a prey population.

If wolves are, in fact, good at killing, eating, and fecal production, it follows that, in enzymatic terms, substrate (prey) is in effective excess as long as wolves continue to produce moose-generated scats. Hence, there is no reason to expect moose at low densities to recover from wolf predation without lowering the conversion rate of moose to wolf feces. In terms of our more complicated, and more alliteratively appealing multiple equilibrium model, moose are in a "predator pit."

Typically, the rate of conversion of moose to wolf scat will be a direct function of the number of wolves (analogous to enzyme molecules) catalyzing the conversion of moose to wolf scat (Fig. 2). Furthermore, if we know substrate (moose) concentration is not limiting the ability of wolves to convert them to wolf scat, the "enzyme rate constant (K_m)" for wolves, and the concentration of wolves, we will be able to calculate the equilibrium direction and rate of moose conversion to wolf scat. If we know these things, and can determine moose concentrations, wolf/moose dynamics can be described.

We can determine the "wolf rate constant" (K_m) for wolves in two ways. If we could find an ecosystem where the concentration of moose limited the production of moose-generated wolf scat, and could experimentally increase the concentration of moose until moose-generated wolf scat production reached V_{max},

we could then read the concentration of moose (moose density) required to produce half of maximum velocity from Fig. 1. More practically, we could study the activity of "the enzyme" (wolves) directly to determine their kill rate on moose because K_m is homologous to this kill rate.

An enzymologist would consider definition of the "enzyme rate constant" (K_m, moose predation rate for wolves) basic to understanding the system. I suggest predator-prey research might profit from the same approach in studying wolf kill rates.

The Management Issue

Once we assess the kinetics (or as wildlifers call them, the dynamics) of a predator-prey system and find that moose are being converted to wolf scat faster than they are being produced, we may decide the equilibrium should be shifted to favor moose (substrate) concentration. At that point, the question of how to lower the conversion of moose to wolf scat becomes relevant.

From the above, it is obvious that decreasing the number of active enzyme sites is the only way to cause a reduction in reaction rate. This can be done by reducing the number of enzyme molecules (wolf population reduction) or making them less effective through the use of inhibitors. Diversionary feeding of predators would be one way to do this. *[Author's note: Alaska Department of Fish and Game was considering diversionary feeding of wolves and bears, using railroad-killed moose, in an effort to increase caribou herd numbers in Interior Alaska when this essay was written.]*

Within the framework of this discussion, the relevant question relating to diversionary predator feeding is not, "Can we make more caribou or moose using this method?" Instead, it is, "Is carrion an effective competitive inhibitor of wolf (or bear) catalysis of caribou (or moose) conversion from living animals to wolf or bear scat?"

In the same frame of reference, the questions about socially acceptable methods of wolf control should not only address the issue of whether the public will tolerate them, but whether or not they will affect the overall rate of prey conversion to wolf product. *[Author's note: Alaska Department of Fish and Game is now involved in sterilization of alpha pairs of wolves in an attempt to increase caribou calf survival in the Forty-mile Caribou herd in Interior Alaska.]*

DISCUSSION

Here it is important that we not confuse "rate" and "dynamics." As developed above, "rate" is a function of concentration, however "dynamics" are the cumulative results of associated reaction "rates." Obviously managers are more interested in "dynamics" than "rate" because altering the dynamics of the system to produce more moose for human consumption is the traditional management goal. To approach defining "dynamics" using "rates," I suggest solving a system of simultaneous equations describing the plots of Fig. 1 and Fig. 2 (where the wolf/moose K_m is a common constant) would be a beginning.

What About Research Methods?

As long as substrate is in excess, it is impractical to monitor disappearance of substrate as a measure of the reaction rate. That is, it is semantically (and scientifically) inappropriate for us to define our monitoring of the disappearance (a rate study) of marked moose (or other substrates) as "predator/prey" research. If substrate is in excess, monitoring its disappearance would be correctly termed "prey" research.

If we are primarily interested in prey research, and what we really want to know is the rate of radio-collared moose disappearance, moose mortality studies are appropriate. Studies of this type serve primarily to confirm existing knowledge. They demonstrate conclusively that predators mediate (by accelerating) the recycling of moose in the ecosystem. However, studying substrate conversion where substrate is not limiting cannot elucidate the dynamics of the system, it can only document the rate of prey disappearance. When biologists engage in studies of this type, they may also infer the cause of disappearance (which predator did the killing) from study of carcass remains. Only on rare occasions is predation actually observed. Within this frame of reference, an enzymologist would ask:

> If you are interested in dynamics, why do you study the disappearance of moose to measure its conversion to wolf scat in systems where you don't think moose concentration actually limits wolf predation. Would it not be better to study wolves and see how often they accomplish the conversion, or alternately, why not study the kinetics of moose-generated wolf scat appearance? After all, how often does a moose turn into wolf scat in the absence of wolves?

If this frame of reference is relevant (obviously I think so I wouldn't be writing this), we have inappropriately labeled our research into predator-prey dynamics for as long as I can remember us having identified it as such.

Earlier investigators studied predators more-or-less separately from prey. This approach was productive to the extent that it documented the now-obvious fact that it is no problem for wolves to kill enough prey to survive and thrive. This was not a trivial finding

because, at the beginning of modern predation studies, the popular scientific perception of predation (as a sort of biological toothbrush taking only "the unfit") was so far from reality it was both necessary and expedient to document the basic facts of life and death. *[Author's note: In Alaska this documentation was more necessary for the scientific community than the experienced Alaskan public. Unfortunately, part of the credibility cost managers have born in "convincing ourselves" has been loss of respect from folks who already knew it from empirical observation over long periods...but that's another story.]*

Once an adequate data set demonstrated that wolves were efficient predators without a compelling altruistic evolutionary rationale driving their prey selection, research biologists decided to address the problem of predation research more inclusively. They began to think of predator-prey research as a "systems analysis" problem, and attempted to describe the larger picture (presumably with an eye toward managing the system).

In retrospect, I suggest predator-prey specialists began to ask "systems-type" questions without making appropriate changes in methodology. That is, they began to ask "dynamics" questions based first on qualitative and then "rate" methodology. This methodology had been effective in answering the most basic questions relating to predator biology, but was extremely difficult to "marry" to systems analysis. As a result, the data gathered didn't directly answer the questions being asked. This is not an uncommon situation as research questions become more complex. Neither is it unique to wildlife research. Nevertheless, we were quite resourceful in our efforts to rationalize the disparity between what we hoped to know and what our methodology revealed. We turned to modeling.

In an effort to make the data obtained using traditional, natural history-type methodology relevant to the complex management situations involved, researchers pursued modeling. After all, we've all been assured that modeling is the way to identify strengths and weaknesses in knowledge. Through experience, we eventually discovered the obvious (within the traditional wildlife management frame of reference)...that managers need to know how all the variables in as system will affect each other before they can, with surety, predict an outcome within the narrow range of probable results demanded in programs as controversial as wolf control. This was not a particularly satisfying prospect because knowing how unpredictable systems will react is impossible. Still, we kept at modeling, and our models became increasingly sophisticated. These complex models soon so dominant our thinking, that we began to focus more on their refinement than on the management task at hand. The result has been that our models have become so complex and esoteric that they are beyond the reach of most managers.

I think this is risky for two reasons. First, management is most often justified to the public and implemented by area management biologists. These biologists are typically too busy with "real life" to focus on the esoteric details of models which, to date, have functioned primarily as research tools. Hence, managers are often in a difficult position when it comes to justifying a management action derived from a research model. As a result, managers may be more comfortable managing on the basis of local knowledge than by the uncertain or probability-defined model outputs. This is particularly true when model outputs are based on questionable, assumed, or extrapolated inputs.

Second, the ephemeral nature of models invites criticism from others. This is an interesting and perhaps edifying exercise for those who are "into modeling," but generates public confusion and endless debate over alleged points of biology which may not be vitally relevant to the big picture. Public confusion compromises management effectiveness, particularly when it is easy to find "expert wildlife scientists" with competing views or philosophies who can always find something to criticize in a complex model.

What Does All of This Mean?

Perhaps nothing. However, I think it points to several relevant questions about the way we plan to conduct predator-prey research in the future. First, we need to decide what questions we are asking, and identify the most productive methodology to apply in seeking answers. If we really want to study predator-prey systems, I suggest it will be productive to move beyond the standard methodology, which focuses on neonate mortality studies. If we would understand "the system" with an eye to regulating it, we must first understand "the predatory enzyme" itself.

Appropriate questions include:

1. Is prey actually in excess to predator food requirements?
2. What is the per unit rate of conversion of prey to wolf products? What is K_m for wolf conversion of the species they utilize?
3. What are the concentrations of "enzyme" (predators) and "substrate" (prey)?

[Author's note: Alaska came close to addressing these kinetic/ dynamic relationship questions in the predator reduction programs of the late 1970s and early 1980s. This almost happened as we arbitrarily selected specific predator: prey ratios as defined end points of predator reductions of that period. However, once changes in the political climate pre-

cluded predator reductions, research remained the only predator-related activity, and research methodology reverted to prey mortality studies (primarily among neonates).]

4. What can be done to decrease the rate of conversion?

SUMMARY

Sociopolitical forces and a systems-approach based on adherence to established, but questionably relevant, methodology have driven predator research toward increasingly esoteric models which have yet to yield a management benefit. It might be productive to returned to a simpler, generalized approach if we hope to understand the actual dynamics of predator-prey systems. After all, concentrations of wolves and prey and the species-specific kill rates by wolves are the keys to understanding dynamics.

Additionally, it is important for us to continually remind ourselves of the impact our models have on our perceptions and how we convey them to others. For example, consider the well-worn term, "predator pit." This term attractively describes the lower level equilibrium predicted from spruce tree/ insect population dynamics that has been applied (or perhaps misapplied) to ungulate/carnivore systems. In this case, our terminology affects public perception.

Our use of the term (and concept) of a "predator pit" (which has its basis in insect fecundity) invites the public to infer that once wolf reduction has boosted a moose population from "the pit," no further reductions in environmental resistance are required to assure moose (and wolf) abundance. When we cause or allow this to happen, we fail to remind everyone involved that maintenance of high moose abundance requires continuous low environmental resistance. Hence, we should not lead or allow the public to assume that wolf control is a "one-shot" proposition.

When we allow facilitate public consideration of complex theoretical models, the cultured skepticism about wolf reductions will seize the notion that environmental resistance (of which wolf control is the major manageable component) can be effectively assured by intermittent and infrequent wolf control. If so, the public may resist continuous manipulation of predator populations on the assertion that "once in a while" is adequate. Adopting a simplified model (the enzyme kinetic model, despite its unfamiliarity to wildlife biologists, is much less complicated than the multiple equilibrium spruce/ insect model) would logically preclude the opportunity for complicating erroneous assumptions of this type.