University of Alberta

ANALYSIS OF PREDATION DATA FROM MOOSE-WOLF SYSTEMS

by

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fulfilment of the requirements for the degree of Master of Science

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ABSTRACT

I examine some assumptions researchers make about the nature of wolf numerical and functional responses to moose, and about the impact of wolf predation on moose populations. To address these assumptions, I assessed the fit of linear models to functional and numerical response data from wolf-moose systems. These predation data were described better by linear models than by hyperbolic models based on predator-prey theory. Linear functional and numerical response models produced a total predation model indicating that wolves might drive low-density populations to extinction. However, simulations using these linear models suggested that persistence of moose at low densities was possible if the population was divided into subpopulations, with some having high growth rates and densities, and wolves concentrating on only the high-density parts. These high-density subpopulations persist by remaining above densities where predation could cause extinction. The subpopulation idea is corroborated by low-density estimates reported in moose population surveys that are weighted averages of high- and low-density areas, where a majority of the survey is composed of low-density subpopulations that might experience little wolf predation.

If further research in wolf-moose ecology produces data showing that wolf functional responses follow a more conventional, theory-based shape, the next step is distinguishing between type II and type III functional responses. This ability is important because the shape of the functional response will indicate whether wolf predation on ungulate populations will have the potential to regulate those populations. I simulated data based on actual wolf-moose predation data to evaluate how easily a type II functional response can be distinguished from a type III functional response, and determined the

sample size necessary to attain a power of 80%. My ability to distinguish between functional response types was poor, due to the low sample sizes and high variance that usually accompany large mammal predation studies. Because of this low power, researchers should consider alternatives to functional response studies when trying to determine the effect of wolves on moose dynamics.

ACKNOWLEDGEMENTS

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I thank the 11th floor gang. All of the friendships I have made over the past three years have made grad school entirely worthwhile. How boring my world would have been if it weren't for cake day!

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CHAPTER 1. INTRODUCTION

In populations from which a sustained harvest of ungulates is a goal, an understanding of their predators and how they affect ungulate dynamics is important. If predation is an important influence, then predators will affect growth rates of ungulate populations, their densities, and ultimately the amount of harvest that can be sustained.

To improve the understanding and management of systems containing wolves (*Canis lupus*) and ungulates, researchers have recently begun to use predator-prey theory to explain their dynamics and interactions (Boutin 1992, Gasaway et al. 1992, Dale et al. 1994, Messier 1994). Much of this theory includes using functional response (change in rate of prey killed per wolf with prey density) and numerical response (change in wolf abundance with prey density) models to explain the predatory behaviour of wolf populations at different densities of ungulates. These responses for wolves are generally described by hyperbolic or sigmoid models (Dale et al. 1994, Messier 1994) that, when combined, produce a total predation response model.

This total response indicates whether wolf predation on ungulates is density-dependent (proportion killed increases with prey density), and if predation will have the potential to regulate ungulates at a low-density equilibrium. According to predator-prey theory, density-dependent predation occurs at low densities of prey if: 1) the functional and numerical response of predators are both hyperbolic, each having a rapid increase from zero at low prey densities; or, 2) the functional response is a sigmoid shape (Messier 1993). Because of their importance, researchers have been attempting to determine the shapes of functional and numerical response of wolves to understand the dynamics of wolves and their prey, and to have a better idea of how wolves and ungulates will respond to management actions, particularly ungulate harvest and wolf removal. However, data to support these model shapes and the dynamics that result are far from conclusive (Sinclair 1989, Skogland 1991, Boutin 1992, Van Ballenberghe and Ballard 1994).

A further suggestion from predator-prey theory involves the importance of sigmoid functional responses. If a functional response has a sigmoid shape, then total predation by wolves will be density-dependent regardless of the form of the numerical response (Oaten and Murdoch 1975). However, finding this functional response shape will probably be very difficult because of the inherent variability and small sample sizes that accompany large mammal studies. This means that using alternatives that do not rely on detecting small changes in a functional response curve would provide a more reliable method of determining if a functional response is sigmoid, or if predation is density-dependent. If the latter can be established by itself, then there would no longer be a need to directly measure functional response of wolves.

This thesis focuses on predator-prey systems involving wolves and moose (*Alces alces*). I analysed a previously-published data set containing wolf functional and numerical response information (Messier 1994). In chapter 2, I re-analysed this functional and numerical response data to see if simpler (linear) models fit given wolf-moose predation data. In chapter 3, I used these linear models in a population simulation to investigate the circumstances where linear models could produce persistence in a wolf-moose system. In chapter 4, I conducted a power analysis on the functional response data to investigate the probability of detecting a sigmoid functional response in wolf-moose predation data given that one truly exists.

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CHAPTER 2. DOES PREDATOR-PREY THEORY DESCRIBE THE DYNAMICS OF WOLVES AND MOOSE?[1]

While the direct causes of mortality in moose have been well documented (Messier 1994), there still exists a debate as to which factors ultimately drive the dynamics of moose populations in their environment (Skogland 1991, Boutin 1992, Van Ballenberghe and Ballard 1994). Several factors could limit moose populations including wolf predation (Fuller and Keith 1980, Keith 1983, Bergerud et al. 1983, Messier 1985, Messier and Crête 1985, Fuller 1989, Messier 1991, Gasaway et al. 1992, Messier 1994), bear predation (Ballard et al. 1981, Franzmann and Schwartz 1986, Boertje et al. 1988, Larsen et al. 1989, Schwartz and Franzmann 1989, Ballard and Miller 1990, Ballard et al. 1991, Ballard 1992), habitat quality (Messier and Crête 1984, Albright and Keith 1987, Crête 1989, Messier 1991) weather severity (Gasaway et al. 1983, Mech et al. 1987, Ballard et al. 1991), and harvest (Gasaway et al. 1983). Those commonly described as density-dependent are wolf predation and competition for food.

Researchers commonly analyse wolf predation using functional and numerical responses. The functional response of a predator reflects how individuals change their kill rates with changing prey densities (Solomon 1949, Holling 1959). There are two functional responses described for wolves: type II and type III (Dale et al. 1994, Messier 1994). Type II responses (Figure 2-1) follow a gradually decelerating hyperbolic function that eventually plateaus at high prey densities, presumably due to satiation of the predator

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[1] A version of this chapter has been submitted for publication in *Ecology*, December 1996 (Marshal and Boutin).

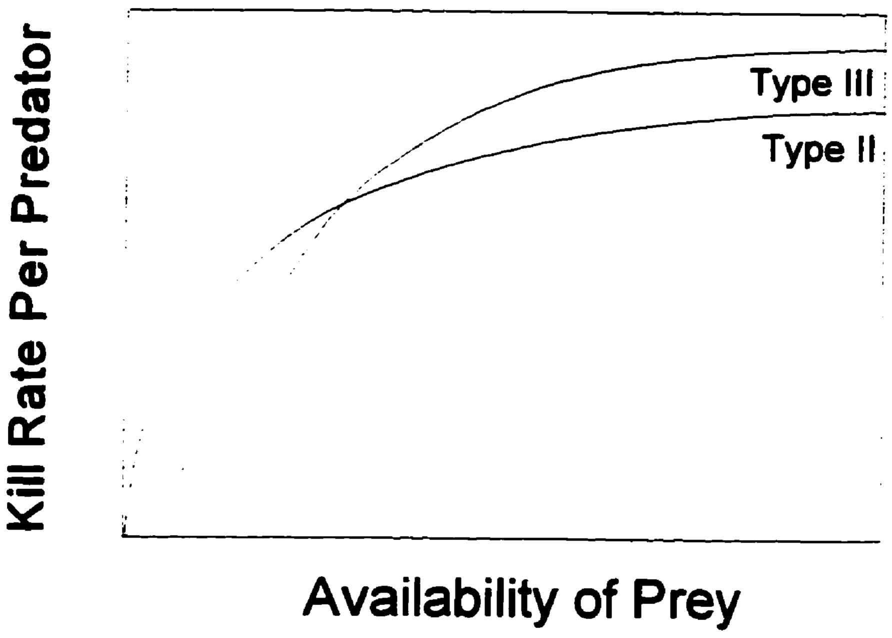

Figure 2-1. Model shapes for type II and type III functional responses.

(Holling 1959). Type II responses are usually described for systems where there is only one prey species available to a predator in a homogeneous environment. A type III response (Figure 2-1) follows a sigmoid curve, with a rapidly decelerating and plateauing portion at high prey densities. The accelerating slope over low to intermediate prey densities could be caused by an increase in predator efficiency as prey become more available, by prey refugia as prey become rare and difficult to find, or by switching from one prey source to another that is increasing and becoming more available (Messier 1993).

Numerical responses by predators also contribute to total predation rate. Many researchers believe that this response for wolves follows a curve similar to the type II functional response (Messier 1994). The numbers of predators increase rapidly at low prey densities, but become limited at high densities by social constraints. Others represent this response as a linear model that increases continually over prey and wolf densities (Keith 1983, Fuller 1989). Functional and numerical responses combine to determine total predation pressure by wolves (Messier 1993, 1994).

The shapes of these responses are important to the effects of wolf predation on moose populations. If these responses are modelled according to predator-prey theory, they each have a rapidly increasing region at low to intermediate densities of prey, and then become constant at high prey density. When responses are combined, the resulting total predation response also has a region of positive slope indicating a rapid increase in predation as moose density increases. If predation rate increases with moose density so that predation becomes greater than production of the moose population, wolf predation can regulate moose populations at low density (Figure 2-2). Without this region of

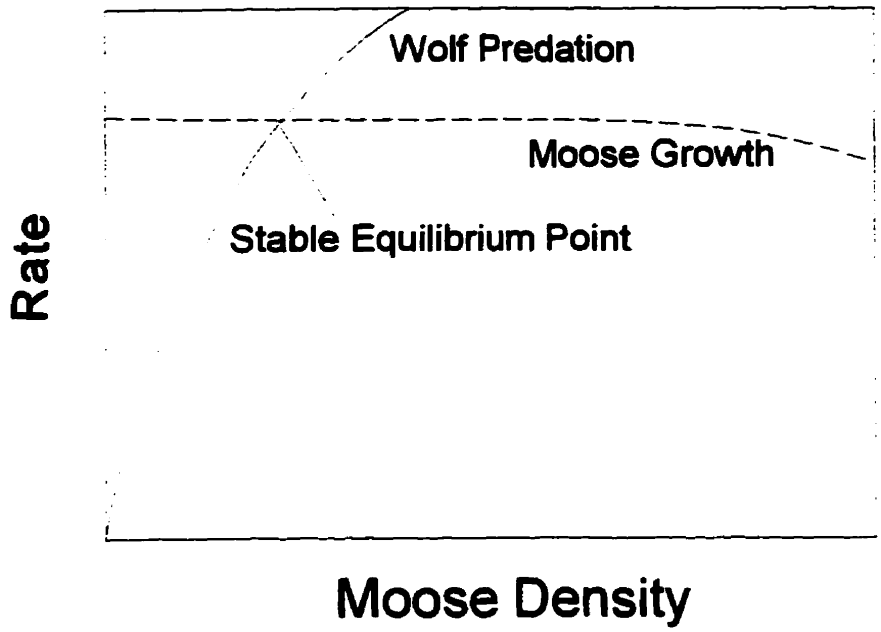

Figure 2-2. Predation model showing wolf predation rate (proportion of the moose population killed by wolves) and moose growth rate (proportion added through recruitment). These model shapes result in regulation of moose at a density coinciding with the stable equilibrium point.

positive slope and rapidly increasing predation rate, there is no low-density equilibrium, and regulation at this density does not occur. This density-dependent total predation model (Messier 1994) depends entirely on the shapes of the numerical and functional responses.

Messier (1994) fitted a hyperbolic model to functional and numerical response data of wolves preying on moose, and found that the model provided a reasonable statistical fit. In this chapter, I re-analysed these data to see if an alternate model (linear) could provide an equal or better fit to the data. My rationale for doing so is as follows. Hyperbolic models exist as predator-prey theory, and as such, should be tested by empirical data. The appropriate test of the model is not whether it provides a good statistical fit to the data but rather that it provides a better fit than do alternate models. I show that linear models actually provide a better fit to the data than do hyperbolic models. When these linear functional and numerical responses are used to produce total predation rates, I found that there was no density-dependent predation over a wide range of moose densities.

METHODS

The source of my information was moose densities, wolf densities, and wolf kill rates presented in Table 2 of Messier (1994). The linear models are of the standard form $y = a + bx$, where x is the density of moose (individuals/km^2), and y is either the kill rate by wolves (functional response, moose/wolf/100 days), or the density of wolves (numerical response, individuals/1000 km^2). Messier (1994) uses two models based on a modified Michaelis-Menton equation to produce a hyperbolic curve. For the functional response he uses $y = ax/(b + x)$, where a is the asymptote of the line, and b is the value of x when $y = a/2$, indicating how quickly the line rises to the asymptote or the efficiency of the response.

For the numerical response, he uses $y = a(x - c)/(b + x - c)$, where a and b are as above, and c is a parameter to allow the intercept to move away from the origin.

I evaluated the quality of fit by the number of parameter estimates that were significantly different ($\alpha = 0.05$) from zero for each model, and by the proportion of variation in the data that each model could explain (r^2). In addition, I used some regression diagnostics to address the assumptions of least-squares regression, the method used to fit both the linear and hyperbolic models. These assumptions are that the residuals are normally distributed with mean zero around the model, and that the error variance remains constant over all values of the independent variable. I used normal probability plots and histograms to evaluate the normality of the standardized residuals around the models, and used plots of the standardized residuals against moose density to look for departures of the standardized residuals from the shape of the model and changes in variance with changes in the independent variable.

RESULTS

Functional Response

The linear model was $y = 1.13 + 0.90x$ ($r^2 = 0.48$, df = 12, $P < 0.01$; Figure 2-3a). I compared this to a hyperbolic model (Figure 2-3b). The linear model explained variation in the data better, as seen by the improvement in r^2, and by the fact that both parameters for the linear model were significant, whereas only parameter a (indicating the asymptote of the hyperbola) for the hyperbolic model was significant (Table 2-1).

A comparison of linear and hyperbolic models indicated that the residuals for the

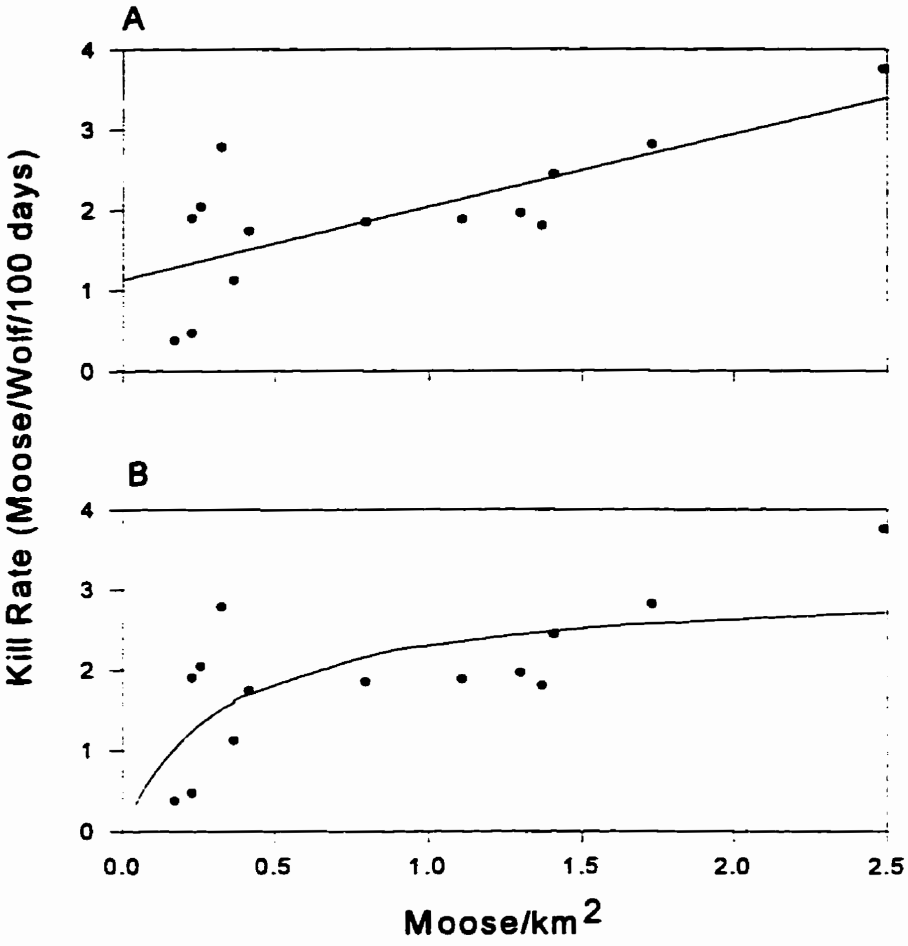

Figure 2-3. Functional response data for wolves preying on moose. A) Linear model fitted through these data ($y = 1.13 + 0.90x$; $r^2 = 0.48$, df = 12, $P < 0.01$). B) Hyperbolic model fitted through these data [$y = 3.08x/(0.34 + x)$; $r^2 = 0.43$, df = 12]. Data are from Table 2 of Messier (1994).

Table 2-1. Comparison of statistics for linear and hyperbolic models for functional and numerical response data from Messier (1994).

Response	Model	df	r^2	Parameters[1]	
Functional	Linear	12	0.48	*a*	Sig.[2]
				b	Sig.[2]
	Hyperbolic	12	0.43	*a*	Sig.[3]
				b	N.S.[3]
Numerical	Linear	30	0.70	*a*	Sig.[2]
				b	Sig.[2]
	Hyperbolic	29	0.63	*a*	Sig.[3]
				b	N.S.[3]
				c	N.S.[3]

[1] Sig. - significantly different than zero at $\alpha = 0.05$: N.S. - not significant.

[2] Statistical difference based on Student's *T*.

[3] Statistical difference based on asymptotic 95% confidence intervals.

linear model were more normally distributed around the model, as seen by the closer linear relationship on the normal probability plot (Pearson's correlation coefficient $r_{xy} = 0.99$ versus 0.95; Figure 2-4a), and by the more symmetrical distribution of residuals in a histogram (Figure 2-4b). A plot of the residuals against moose density (Figure 2-4c) should have shown no pattern or trend over the independent variable. For values above 0.5 moose/km^2, the residuals from the linear model fell more closely to zero, and were more symmetrical around zero as moose density increased. Residuals from the hyperbolic model fell farther from zero and were less symmetrical. For both the linear and the hyperbolic models, there was a much larger spread in the residuals at densities between 0 and 0.5 moose/km^2 (Figure 2-4c) than along any other part of the model. This indicated error variance increased with decreasing moose density, and this increase could not be accounted for by a change in model shape.

Numerical Response

The resulting linear relationship through log-transformed data [$\log_{10}y = 1.45 + 0.69(\log_{10}x)$; Figure 2-5a] was tighter than the hyperbolic relationship through the original, non-transformed values ($r^2 = 0.70$, df = 30, $P < 0.01$, Table 2-1, Figures 2-5c and d). In addition, only 1 of 3 parameter estimates in the hyperbolic model were significant (parameter *a*, the asymptote; Table 2-1). Messier (1994) presented the data on a plot with a logarithmic axis for wolf density but not for moose density (Figure 2-5c); however, when fitting the models he used non-transformed values. The range of values for both moose and wolf densities is over an order of magnitude, and the variance increases with moose and wolf density. For these reasons, I log-transformed both sets of values. The log-log

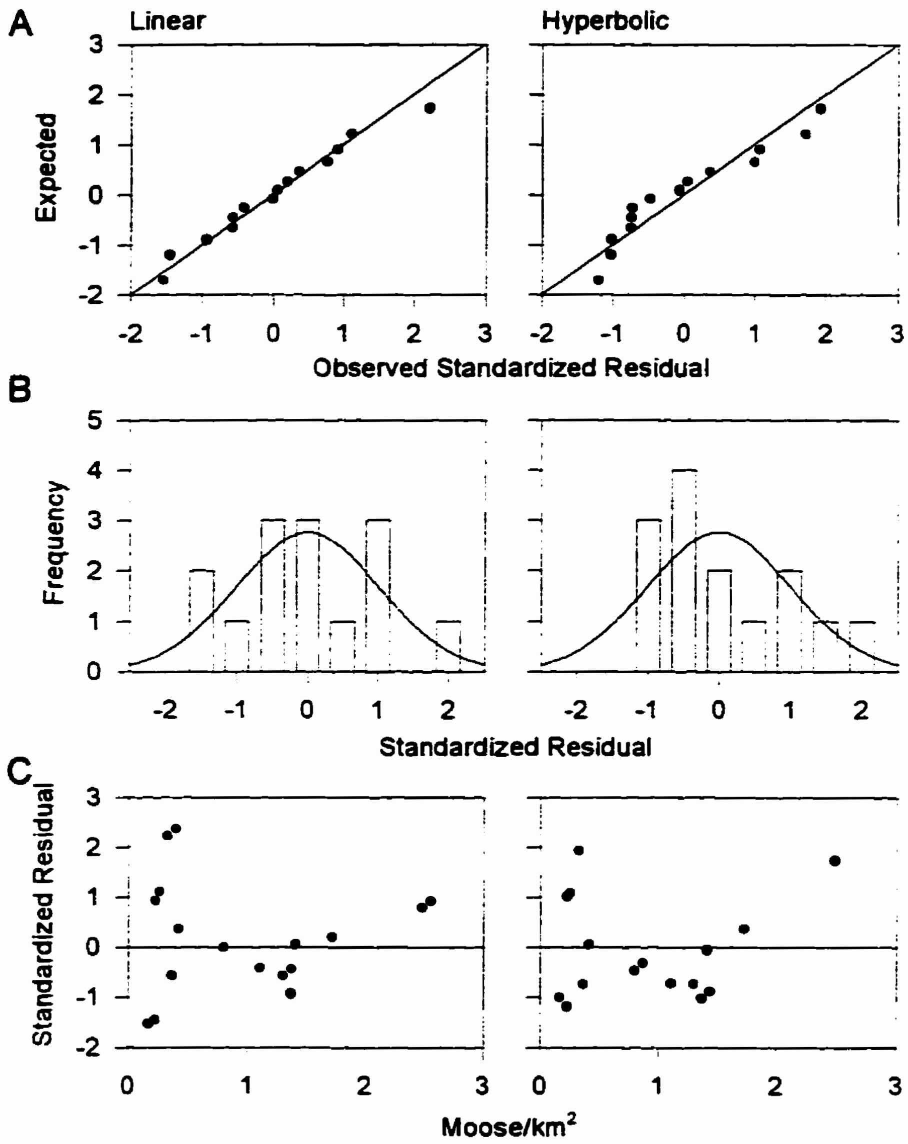

Figure 2-4. Regression diagnostics for linear and hyperbolic functional response models. A) Normal probability plot showing observed standardized residuals versus expected standardized residuals under normality; if the observed residuals are normally distributed then they fall on the diagonal line. B) Histogram showing distribution of standardized residuals, overlaid by a normal distribution curve. C) Plot of standardized residuals versus moose density; residuals should show no discernable pattern or trend with moose density.

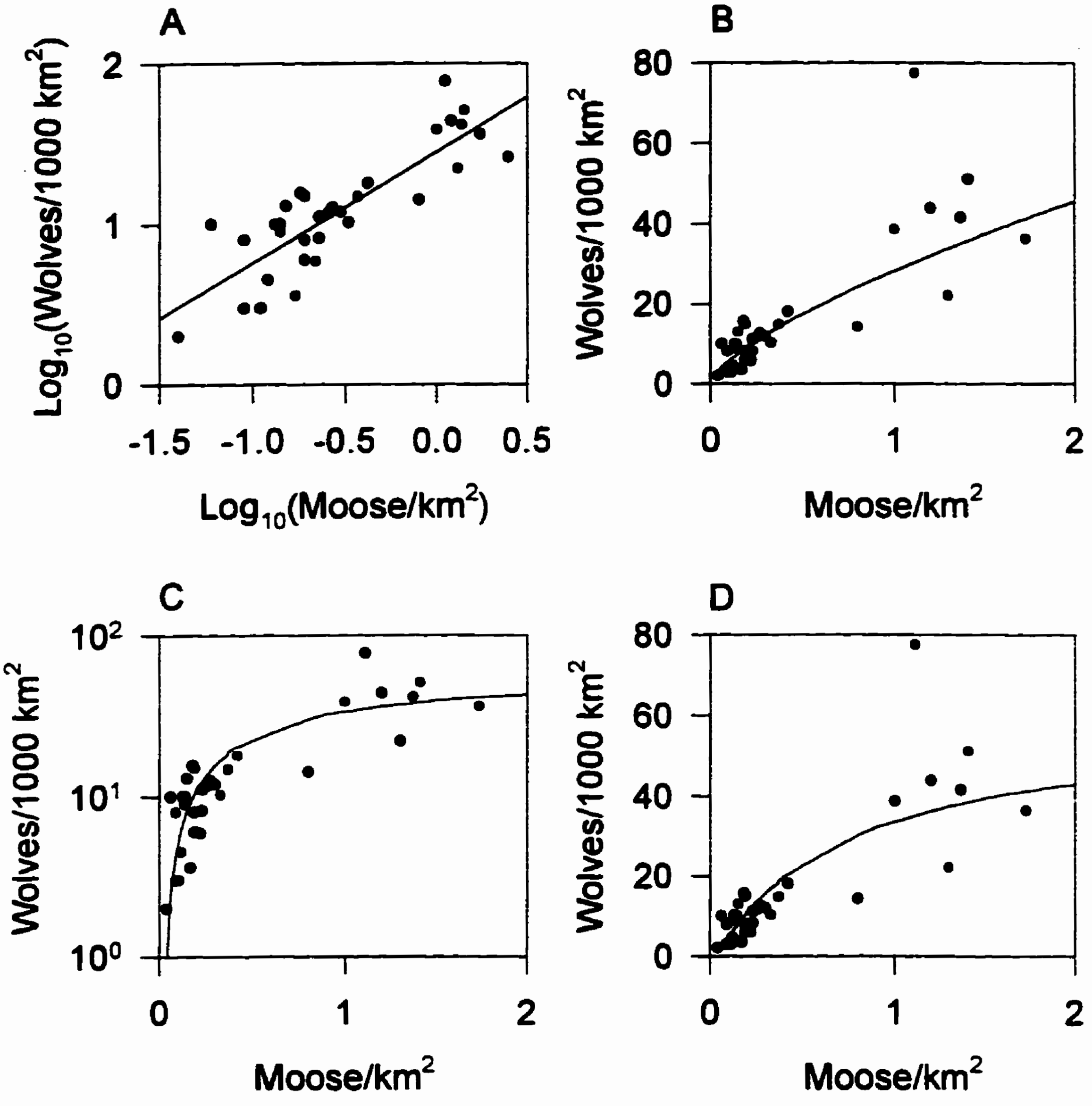

Figure 2-5. Numerical Response data for wolves preying on moose. A) Linear model fitted through log-transformed data ($\log_{10}y = 1.45 + 0.69\log_{10}x$; $r^2 = 0.70$, df = 30, $P < 0.01$). B) Data and model from (A) without transformation. C) Hyperbolic model fitted through non-transformed data [$y = 59.37(x - 0.03)/(0.72 + x)$; $r^2 = 0.63$, df = 29], with the wolf-density axis in log-scale [modified from Figure 4 of Messier (1994)]. D) Data and model from (C), with both axes in linear scale. Data are from Table 2 of Messier (1994).

relationship, when converted back to linear scale, is in Figure 2-5b.

When comparing models, I found that the linear numerical response had more normally distributed residuals ($r_{xy} = 0.99$ versus 0.87; Figure 2-6a). A histogram of the residuals (Figure 2-6b) showed that both models produced symmetrical distributions, although residuals from the hyperbolic model appeared to have a leptokurtic distribution, with more residuals falling closer to zero than they should for normality (kurtosis = 9.11, SE = 0.80, $P < 0.01$). When plotting residuals against moose density, both models showed symmetry in the residuals, but the hyperbolic model showed an increase in error variance with an increase in moose density. Because log-transformation corrected this, the linear model through transformed data had the appropriate patternless, symmetrical spread around zero.

In both cases, linear models had a considerably better fit than hyperbolic models. Each linear model had a higher r^2 value than hyperbolic models, and for the hyperbolic models, at least one of the estimated parameters was not significantly different than zero. Regression diagnostics also suggested that the linear models agreed more closely with the assumptions of least-squares model fitting. Linear models appeared to be more statistically appropriate for describing these data sets.

A concern in this re-analysis was the influence of a very high kill rate value at 2.49 moose/km^2. This appeared to be an extreme value and including it could pull a line fitted by least-squares toward the outlying observation and result in a misleading model fit (Neter et al. 1990). Therefore, I omitted that value and re-fit the linear and hyperbolic models. Both models explained a small amount of variation (linear: $r^2 = 0.23$, hyperbolic: $r^2 = 0.34$).

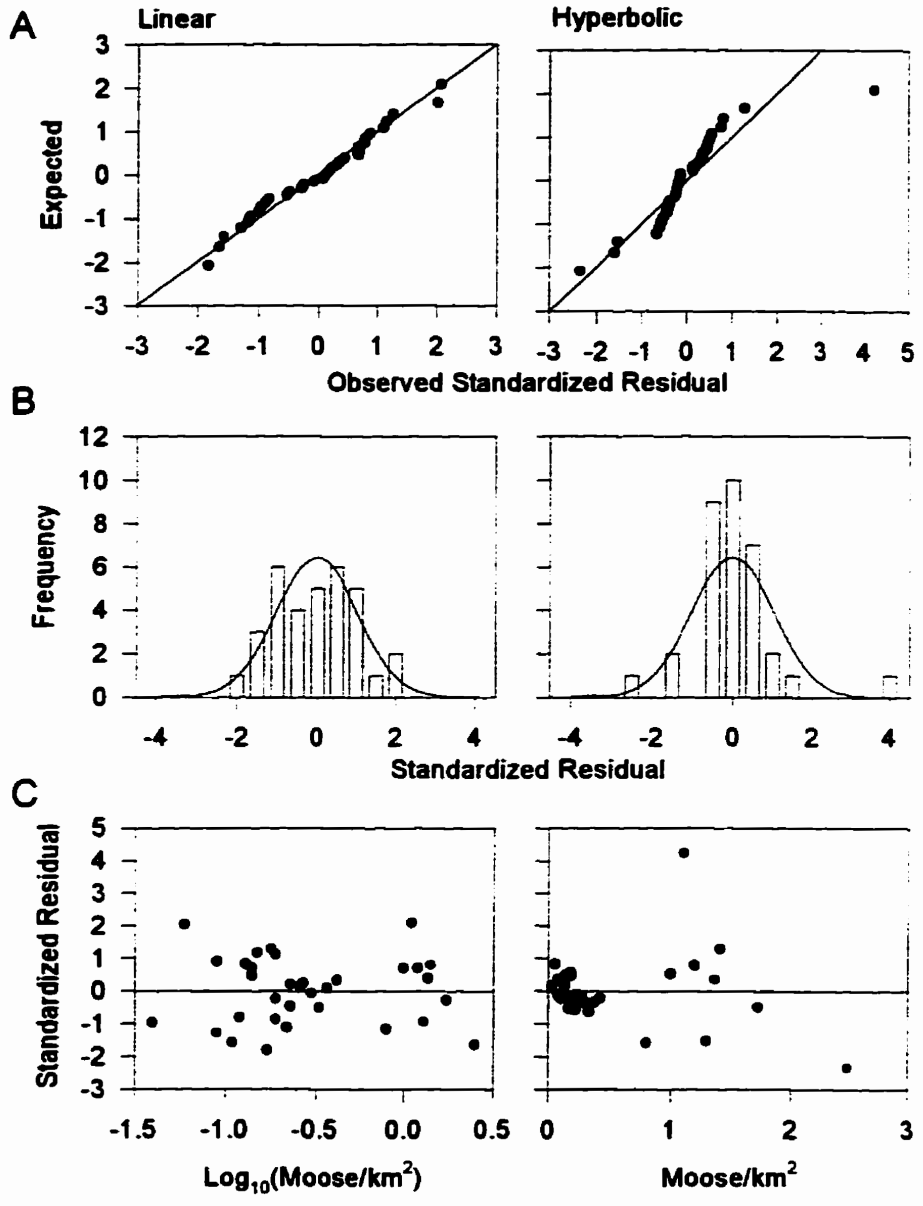

Figure 2-6. Regression diagnostics for linear and hyperbolic numerical response models. A) Normal probability plot showing observed standardized residuals versus expected standardized residuals under normality; if the observed residuals are normally distributed then they fall on the diagonal line. B) Histogram showing distribution of standardized residuals, overlaid by a normal distribution curve. C) Plot of standardized residuals versus moose density; residuals should show no discernable pattern or trend with moose density.

For the new linear model, both parameter estimates were significantly different from zero (a: $P = 0.05$, b: $P < 0.01$). For the new hyperbolic model only one parameter estimate was significantly different than zero (95% asymptotic confidence intervals a: 1.47 to 3.65, b: -0.11 to 0.54). Based on this analysis, neither model appeared to fit well to the functional response data set, as shown by low r^2 values or by parameter estimates that were not significantly different than zero. Because this data point appears important to both models, and because there were no a priori reasons to exclude this point, I chose to use the original model for the remainder of the re-analysis. However, this suggests that any analysis that relies heavily on a single data point could easily result in spurious conclusions.

Do these new models allow for a predation effect that is consistent with observations of wolves and moose in the real world? To address this, I determined the total annual predation by multiplying the functional response by the numerical response, as determined by linear models, and then multiplying again by 3.65 (predation was over a 100-day interval during the winter). Then I divided by moose density to calculate predation rate. The resulting total predation model was plotted over moose population growth rate to look for regions of net growth. The resulting graph (Figure 2-7a) indicated that predation rate was very high at low densities, decreased at intermediate densities, and then began to increase again at higher densities. When the finite rate of increase of moose at low densities was 1.25 (25% per year), net growth of moose occurred between 0.15 moose/km^2 (an unstable equilibrium point) and 1.15 moose/km^2 (a stable equilibrium point). However, at intrinsic rates of increase lower than 1.20, this net growth vanished.

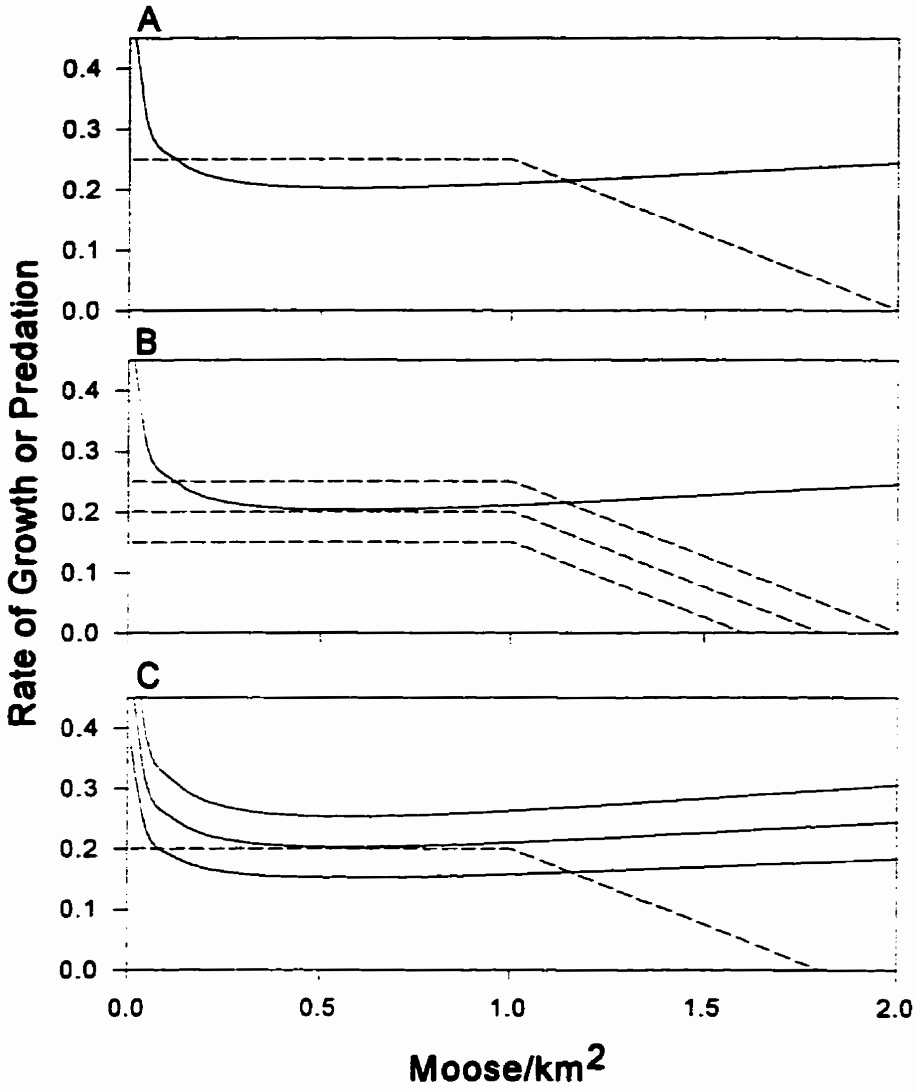

Figure 2-7. A) Low-density extinction model based on linear models of functional and numerical response data. Predation rate is the solid line, and growth rate of moose is the dashed line. B) Effects of changing growth rate on net moose population growth. Changes in growth could result from changes in habitat quality, weather severity, bear predation, or harvest. C) Effect of changing wolf predation rate on net moose population growth. Changes in predation could result from changes in snow depth, and consequently ability to capture prey. For A, B, and C, net growth is represented by area above the predation line and below the growth line.

DISCUSSION

Because of the very high predation rates at low moose densities, I called this the low-density extinction (LDE) model. This model was based on a statistical analysis of wolf predation data, and not on the theoretical shapes of numerical and functional responses for wolves. Considering the improvement in fit that the functional and numerical response data showed to linear models, predation data provide better support for the LDE model than for the density-dependent model. Existing data do not conclusively support density-dependent predation models of wolf predation over other possibilities.

According to this model, predation rate is very high at low moose densities, drops with an increase in moose density, and then increases again as density continues to rise. This relationship is opposite to the predation model suggested by Messier (1994), but it is similar to the predation curve generated for wolves preying on caribou (*Rangifer tarandus*) in Alaska (Dale et al. 1994). Dale et al. (1994) and Messier (1994) are the only two studies that measured wolf predation over a wide range of prey densities. Dale et al. (1994) concluded an anti-regulatory predation model was most appropriate, and my re-analysis of Messier's (1994) predation data support their conclusion.

What does the LDE model suggest for dynamics of wolves and moose in natural systems? The first outcome is that there is a wide range of moose densities where there is a net increase in the moose population (0.15-1.15 moose/km^2). Second, only at the high end of this range will wolves begin to show density-dependent predation on moose, but this density dependence is very weak. Through most of the range of densities where net moose growth occurs, wolf predation removes a constant proportion of moose from the population.

What happens if moose populations have densities lower than 0.15 moose/km^2, or growth rates below 20%per year? There are several environmental factors that could change the height of the predation and growth lines, and change the net growth of moose from year to year (Figure 2-7b and 2-7c). During good years, when there is little snow, abundant food, and low bear predation, moose growth rate would remain high. Also, the elevation of the predation curve might decrease because environmental factors hinder a wolf's ability to capture moose [e.g., Messier's (1994) correction for snow-free periods that decreases the total predation response]. In bad years with high snowfall, severe weather, low food availability, or high bear predation, the moose growth line could decrease to 10-20% per year (Gasaway et al. 1992, Messier 1994, Larsen and Ward 1995). During these years, growth rate is lower than predation rate, and moose populations decrease. Over several years, the density appears to fluctuate between higher and lower densities caused by several non-equilibrium factors. If one year is particularly bad, there are several bad years in succession, or harvest is too high, then moose populations could drop to a density where anti-regulatory predation of wolves could cause an extinction.

Of the existing models explaining wolf-moose dynamics, the model presented above is most similar to the recurrent fluctuations model (Van Ballenberghe 1987), or the predator limitation hypothesis (Boutin 1992). According to both models, moose populations fluctuate over time around a mean density, but do not tend to an equilibrium density. Such a model has been described for highly manipulated systems (Gasaway et al. 1983), where growth rates and densities of moose are primarily limited by wolf predation, but where interactions between moose populations and severe weather events, habitat quality, bear predation, and harvest cause varying densities of moose over time. In addition, time lags could occur between wolves and moose (Keith 1983). These time lags plus the effects of habitat, weather, and harvest could result in these recurrent fluctuations.

Are there biological explanations for linear functional and numerical relationships? According to theory, the functional response is zero at zero prey density and plateaus at some high availability of food. Although I cannot argue that wolves could eat moose when there are none around, kill rates could jump very quickly to some amount even if there are very low densities of moose. In truth, such a functional response would intersect the origin, but statistically this could never be detected because the lowest measured values would be at some value greater than zero. A true positive intercept and a immediate increase from zero to a positive value would not greatly alter the dynamics of the system. The result of a positive intercept would be anti-regulation of moose, while an immediate increase from zero would produce an extremely low-density equilibrium caused by a very rapid increase in predation rates. Regardless, I believe the data cannot be used to determine the shape of the functional response at low densities. Figures 2-4a and 2-4b show that there is far too much variance below 0.5 moose/km^2 to be able to adequately conclude that the functional response shows any specific behaviour, whether it be type II, type III, or linear.

Boutin (1995) gives examples of how surplus killing and partial consumption of small mammal prey by predators could result in a functional response that fails to plateau. These behaviours have also been described in wolves (Peterson 1977, Keith 1983). If moose are very abundant and easily available, wolves would benefit more by consuming only the most digestible parts of a kill and then killing another moose before completely consuming all of the edible biomass from the first carcass. This means that even though wolves might become satiated according to the biomass they consume, they are obtaining that biomass from many more moose than they would at low availabilities of moose. As a result, kill rates of moose continue to increase even at high densities. This is not to suggest wolves would never reach a point where they can no longer increase their kill rates, only that those densities of moose might not occur in a natural system.

According to theory, numerical responses for wolves are primarily determined by prey availability, and wolf territorial behaviour limits growth rates of wolf populations (Zimen 1976, Packard and Mech 1980, Keith 1983, Messier 1985, Peterson and Page 1988). At very high wolf densities, these limits could result in an upper bound in wolf densities. This upper bound is represented as an asymptote in a numerical response model at high availabilities of prey (Messier 1994). I see several arguments suggesting this upper asymptote might not occur, at least at densities of moose observed in natural systems.

Several studies examining wolf pack dynamics reported an increase in intra-pack aggression at high wolf densities (Peterson and Page 1988, Thurber and Peterson 1993). Each of these studies acknowledge that food supply plays the ultimate role in determining wolf densities. However, I suggest that it is not wolf density or food per se that produces the aggressive behaviours within packs, but decreases in per capita food availability that cause these behaviours. Intra-pack aggression could manifest where absolute food availability remains constant while wolf densities increase, or where food availability decreases while wolf densities remain constant. Peterson and Page (1988) report high levels of intraspecific aggression on Isle Royale and attributed this to high wolf densities and a lack of opportunities to disperse. However, during the time that these behaviours occurred, moose densities had decreased. I believe that this decrease, not the high densities of wolves themselves, is the direct cause of aggression. Likewise, Messier (1985) reported high rates of wolf deaths due to strife in an area with low moose density as compared to an area with higher moose density nearby. However, Messier (1985) also reported a decrease in moose density in this same low moose density area (0.25 to 0.21 moose/km^2). While the density change is not of the same magnitude as the moose density changes of Isle Royale during a moose crash, perhaps the change in food was enough to see more aggressive behaviours in the low-density area.

Thurber and Peterson (1993) reported an inverse relationship between wolf density and percent lone wolves in the population on Isle Royale. Wolf population density decreased as a result of a moose crash. The decrease in moose caused an increase in intra-pack aggression, and in turn caused greater egress of transients from established packs, and an increase in lone wolves in the population. With these relationships in mind, I propose that if wolves are bounded at an upper density, as shown by a plateauing numerical response, then Thurber and Peterson (1993) should have found the opposite relationship, that percent lone wolves should increase with wolf density, because of higher aggression at high wolf densities. I expect that if available prey had continued to rise, and if the amount of food available per wolf did not decrease, wolf density would have also continued to rise without an increase in the percent of lone wolves. There are other published relationships that show no suggestion of a behaviourally-imposed upper density of wolves. Keith (1983) and Fuller (1989) each produce relationships showing a linear increase in wolf density with an increase in available prey.

CONCLUSION

I began this re-analysis by using parsimony in choosing models to explain the data, and by trying not to rely upon assumptions about the underlying relationships: that functional or numerical responses are hyperbolic or sigmoid, or that wolf predation on moose is density-dependent. I found linear models showed a better statistical fit than curvilinear theoretical models. Given these finding, I concluded that wolf-moose predation data were not adequate to support one theory of population dynamics to the exclusion of other possible theories. This means more studies must be conducted in an appropriate experimental manner to provide additional information until more definite conclusions can be reached. It also means wildlife managers must carefully consider the implications of several possible theories when making management decisions involving moose populations.

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CHAPTER 3. PERSISTENCE OF MOOSE AT LOW DENSITIES WITHOUT DENSITY-DEPENDENT WOLF PREDATION

In chapter 2, I presented a model of wolf-moose dynamics showing strong anti-regulatory predation and having the potential to cause extinctions at low densities of moose [the low-density extinction (LDE) model; Figure 2-7a]. I further explain that variation in moose densities over time could be the result of annual changes in the moose growth and wolf predation curves. At times wolf predation might be high and the moose growth might be low because of deeper snow improving capture rates by wolves, and also because of higher bear predation, greater difficulty in foraging, mortality during severe winters, or harvest. These, in turn, could decrease moose densities. Alternatively, during mild winters with shallow snow, wolf predation drops, while growth rates of moose increase, resulting in a net increase in moose densities. The sum of these dynamics is a moose population that fluctuates around a mean density (Van Ballenberghe 1987), but never reaches a regulated stable equilibrium.

One question that arises is what happens after a particularly severe winter, or during several consecutive severe winters? Moose densities could be driven low enough so that the anti-regulatory portion of LDE predation causes extinction of moose. Wolf-moose systems in Northern Canada and Alaska continue to persist, even with low productivity, low growth rates, and low moose densities. The purpose of this chapter, therefore, is to investigate the LDE model using population simulations drawing information on moose and wolf population dynamics from the literature, to look for the condition that would allow an

LDE model to occur in nature, and to evaluate whether these conditions are realistic.

Messier (1994) presents a summary of existing wolf-moose predation data spanning a wide range of densities. From this summary, he produces numerical and functional response models and uses these to determine how wolf predation on moose changed as moose density changes. The response models Messier (1994) uses are both hyperbolic (type II). The resulting predation model has an increasing (density-dependent) predation rate over low to intermediate densities, and a decreasing (depensatory) predation rate over intermediate to high densities of moose. This total predation model, when compared to moose population growth rate, results in moose populations that are regulated by wolf predation to a low-density equilibrium when population growth rates in the absence of wolf predation are 15-20% per year, and results in a high density equilibrium primarily regulated by competition for food when population growth rates are >20% per year.

In chapter 2, I argued that the functional and numerical response models fit by Messier (1994) are based on theoretical expectations of what these responses should look like, not on which models would be best supported by the data used in the analysis. I presented linear functional and numerical response models, and used these to produce a total predation model with high predation rates at low densities of moose, and predation rates low enough at intermediate densities to allow for a net moose population growth at low moose densities, given that the densities were > 0.15 moose/km^2, and the population growth rates were > 20% per year (Figure 2-7a). Otherwise, wolf predation would cause a net decrease in moose density, perhaps to the point where the anti-regulatory predation at low densities would cause extinction of the moose population.

One problem that the LDE model does not allow for is the very low densities and population growth rates of moose in northern systems. Here, I extend the LDE model by suggesting that a spatial structure to moose populations and concentration of wolf predation on only part of the total population could produce persistence of moose at low densities. This is based on the premise that low moose densities reported in the literature are actually averages of higher and lower density areas within a study area, and that if the high-density areas are high enough, and wolves concentrate on these areas, then that will allow moose to persist over time.

THE MODEL

I designed this simulation after patterns found in stratified moose surveys, where there are small areas with many moose (high-density strata) and large areas with few moose (low-density strata), and the resulting density estimate is an average of several widely differing strata. I based the starting values in our simulation on moose survey results from several areas in the Yukon (Jingfors 1988, Ward and Larsen 1994, Larsen and Ward 1995). The total study areas in these reports were 2744-4936 km^2, and the high-density strata were 5-10% of the total study area. Moose density in the high-density strata were 0.51-0.97 moose/km^2 where wolves were not actively managed. The remaining low- and medium-density strata were 0.05-0.37 moose/km^2. Most moose surveys have 3 and sometimes 4 strata in their sampling designs. Rather than simulate 3 or 4 moose subpopulations, I used information from high-density strata to model a high-density subpopulation (HDS), and information from the remaining strata to model a low-density subpopulation (LDS).

The simulated study area was a total of 3000 km^2. The initial number of moose in the HDS was 100, and they were in an area of 200 km^2 (7% of the total area) resulting in a starting density of 0.5 moose/km^2. In the low-density area, 200 moose were in 2800 km^2 (93% of total area) resulting in a density of 0.07 moose/km^2. The average density over the entire study area was 0.10 moose/km^2. Growth of each subpopulation was based on the logistic population model, calculated by

$$\text{growth} = rN(1 - N / K),$$

where r is the population growth rate, N is the number of moose, and K is the food-imposed upper limit of moose that the habitat can support. Population growth was calculated with separate parameters for the HDS and the LDS. The parameter r was a random variable with a normal distribution, from which I chose a new growth rate each year to represent changes in growth rate due to factors other than predation by wolves, such as changes in habitat quality, effects of severe winters, and changes in bear predation. For the HDS, the distribution had a mean of 0.25 and standard deviation of 0.10. Fryxell et al. (1988) and Messier (1994) judged that moose populations in ideal conditions would increase at a rate of 25% per year. The LDS had a mean of 0.00 and standard deviation of 0.10. The low average growth rates represent the effects of poor environmental conditions due to sub-optimal habitats and food availability, and the resulting increased susceptibility of individuals to mortality factors. The assumption is that moose in poor habitats are kept at a constant, low density because of these factors and will not increase without immigration. The standard deviation was based on a sample of the literature that reported growth rates for moose (Table 3-1) during wolf removal experiments in several study areas. The mean growth rate, however, was not based on this sample of the literature. Several of the studies either had inadequate wolf removal (leaving too many wolves, not removing wolves for enough time), or moose populations continued to decline even after wolf removal, suggesting exceptional circumstances resulting in high moose mortality.

The upper limit (K) was 800 moose (4.0 moose/km^2) for the HDS, and 400 moose (0.14 moose/km^2) for the LDS. The HDS value was based on the findings of Schwartz and Franzmann (1989) who studied a moose population that increased in response to a large-scale fire disturbance on the Kenai Peninsula, Alaska. Densities rose to 3.7 moose/km^2 because of the availability of high-quality forage and habitat. I chose to use this as a model of high-quality habitat pockets in a heterogeneous landscape. I assumed that the LDS upper limit was very low and that the densities of moose would not in general increase. That is, I assumed that in poor-quality habitats, moose populations were already at their maximum density.

Migration out of each moose subpopulation was calculated as

$$\text{emigration} = EN,$$

where E is the emigration rate, and N is the number of moose of the source subpopulation. Migration rate between the two subpopulations was a function of population density. I based these rates on relationships determined for moose at Rochester, Alberta (see Figure 3 in Rolley and Keith 1980). These authors calculated an expected λ based on survival

Table 3-1. A survey of moose population finite rates of growth (λ) from several moose predation studies.

Study	λ	Density (moose/km^2)[a]	Cause of increase
Ballard et al. 1991	1.07	0.688 to 0.848 (3)	wolf and bear removal
	1.01	0.877 to 0.892 (3)	wolf and bear removal
	1.06	0.710 to 0.844 (3)	wolf and bear removal
Fryxell et al. 1988	1.25		moose harvest[b]
Gasaway et al. 1983. Gasaway et al. 1992	1.18	1.5	wolf removal
Larsen et al. 1989	0.96	0.151 to 0.145 (1)	wolf and bear removal
	0.97	0.145 to 0.141 (1)	wolf and bear removal
	0.96	0.232 to 0.223 (1)	wolf and bear removal
	1.17	0.223 to 0.249 (1)	wolf and bear removal
	1.03	0.249 to 0.274 (3)	wolf and bear removal
	0.86	0.443 to 0.328 (2)	wolf and bear removal
Larsen and Ward 1995	1.12	0.299[c]	wolf removal
	1.18	0.299[d]	wolf removal
	1.11	0.339[c]	wolf removal
	1.16	0.339[d]	wolf removal
Schwartz and Franzmann 1989	1.24	0.3 to 3.3 (11)	high quality habitat
	1.02	3.3 to 3.7 (5)	high quality habitat
Ward and Larsen 1994	1.07	0.513	wolf removal
	1.03	0.717	wolf removal
Mean	1.08		
SD	0.1		

[a] Presented moose densities for a measured λ from cited paper. Where a range is displayed, we determined λ from moose densities using the equation from [c]. Number in parentheses are the years between estimates.

[b] Growth rate determined from harvest rates. A harvest of greater than 25% of the population caused a decline in this moose population.

[c] Cited growth rate calculated as : $r = (\log_e \text{estimate 2} - \log_e \text{estimate 1}) \div \text{years between estimates}$, and $\lambda = e^r$ (Gasaway et al. 1986).

[d] Growth rate calculated as: $\lambda = (1 - M) \div (1 - R)$, where M is the adult mortality rate and R is the recruitment rate (yearlings ÷ yearlings and adults) (Bergerud and Elliot 1986).

and fecundity, and compared that to an observed λ. The differences between them they attributed to migration. I took the observed λ-values and subtracted them from 1.12 (the calculated λ for a population with a stable age structure) to determine changes in net migration rate over time (Table 3-2). I also used a relationship of moose density over time (Table 3-2) and used these two relationships to determine net migration rate changes as a function of moose density. The relationship was $E = 0.084 - 0.253x$ ($r^2 = 0.82$, df = 10, $P < 0.01$), where x was the density of moose (moose/km^2) (Figure 3-1). For the HDS, x was the density of moose remaining after moose were removed by wolf predation. Because Rolley and Keith (1980) did not study surrounding populations to determine their influences on migration into Rochester, I used this relationship to estimate rate of movement out of a subpopulation. Movement into a subpopulation depended on the density of the source subpopulation.

Wolf predation only removed moose from the HDS. Predation was based on the LDE model, and simulated wolves selecting moose only in high-density areas. The functional response was $FR = 0.90x + 1.13$, where FR was the kill rate of wolves on moose (moose/wolf/100 days), and x is the density of moose in the HDS (moose/km^2). The numerical response was $\text{Log}_{10}NR = 0.69\text{Log}_{10}x + 1.45$, where NR was the density of wolves (wolves/1000 km^2), and x was as for the functional response. To determine the number of wolves in the 200-km^2 high-density area, $W = NR \times 200 / 1000$. Total annual predation on the HDS was $FR \times W \times 3.65$ (moose killed/year), and this number of moose was removed from the HDS each year.

I evaluated the importance of having two subpopulations by comparing the model

Table 3-2. Rate of increase information for the Rochester moose population from which we estimated net migration rates as a function of population density.

Year	Density (moose/km^2)[a]	Calculated rate of increase	Observed rate of increase[c]	Immigration rate[d]
1965	0.02	1.12[b]	1.24	0.12
1967	0.06	1.12[b]	1.21	0.09
1968	0.05	1.12[b]	1.19	0.07
1969	0.09	1.12	1.18	0.06
1970	0.15	1.12	1.16	0.04
1971	0.30	1.12	1.14	0.02
1972	0.25	1.12	1.12	0.00
1973	0.35	1.12	1.11	-0.01
1974	0.40	1.12	1.09	-0.03
1975	0.25	1.12	1.08	-0.04
1976	0.6	1.12	1.07	-0.05
1978	0.75	1.12	1.03	-0.09

[a] Density values were estimated by eye from Figure 3 in Rolley and Keith (1980).

[b] Rates of increase for 1965, 1967, and 1968 were based on the fecundity and survival values for a stable age distribution, not on the reported calculated rate of increase (see Figure 3 in Rolley and Keith 1980).

[c] Observed rate of increase values estimated by eye from Figure 3 in Rolley and Keith (1980).

[d] Immigration rate = observed rate of increase - calculated rate of increase.

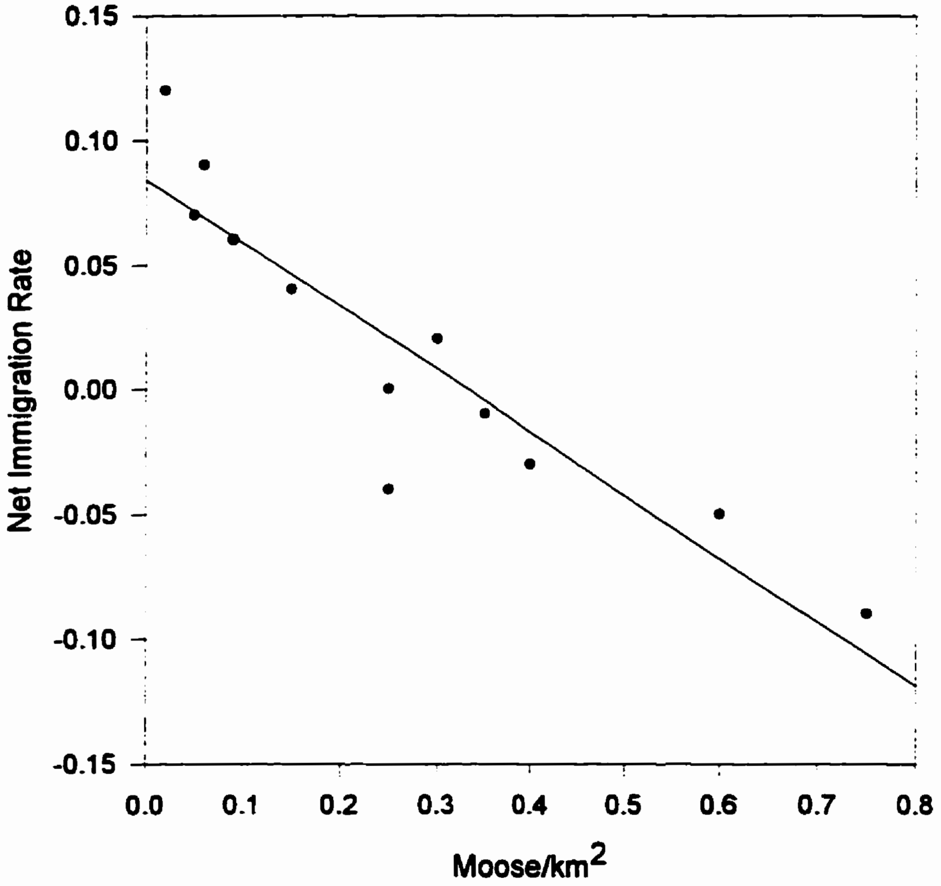

Figure 3-1. Net moose migration rates into Rochester, Alberta as a function of population density ($y = 0.084 - 0.253x$; $r^2 = 0.82$, df = 10, $P < 0.01$).

described above to a simulation with only the HDS portion modelled. The two-population scenario provides two possibilities: 1) persistence occurs because high-density subpopulations have high enough densities and growth rates that they always stay out of the anti-regulatory region of the LDE predation model; the fact that density estimates of these systems are low is an artifact of there being large areas of low-density moose, and for this reason an average will always be low; the low-density parts do not directly affect the dynamics of the high-density parts, or the overall dynamics of the system; 2) persistence occurs only because there is a low-density area that acts as a refuge that protects moose from predation, and it provides moose that sustain the high-density parts and the overall dynamics of the system. If the first hypothesis is true, then removing the LDS part of the model should not affect the dynamics of the HDS. If the second is true, then removing the LDS will result in a model that always ends in extinction.

RESULTS

The system that included the LDS produced a time-series that remained relatively constant. Mean long-term densities (SD) for the HDS, LDS, and the weighted average were 0.49 (0.04), 0.21 (0.03), and 0.23 (0.03) moose/km^2 respectively. Although these densities are generally realistic, I believed that the weighted average was too high to represent a true northern low-density system (< 0.2 moose/km^2). I fine-tuned the model by adjusting the intercept. This changed the migration rates between subpopulations by increasing the rate of movement out of the LDS. This resulted in a LDS net migration rate equation of $E = 0.253x$. This change would decrease the LDS density while increasing the HDS density (Figure 3-2). After this adjustment, the resulting mean

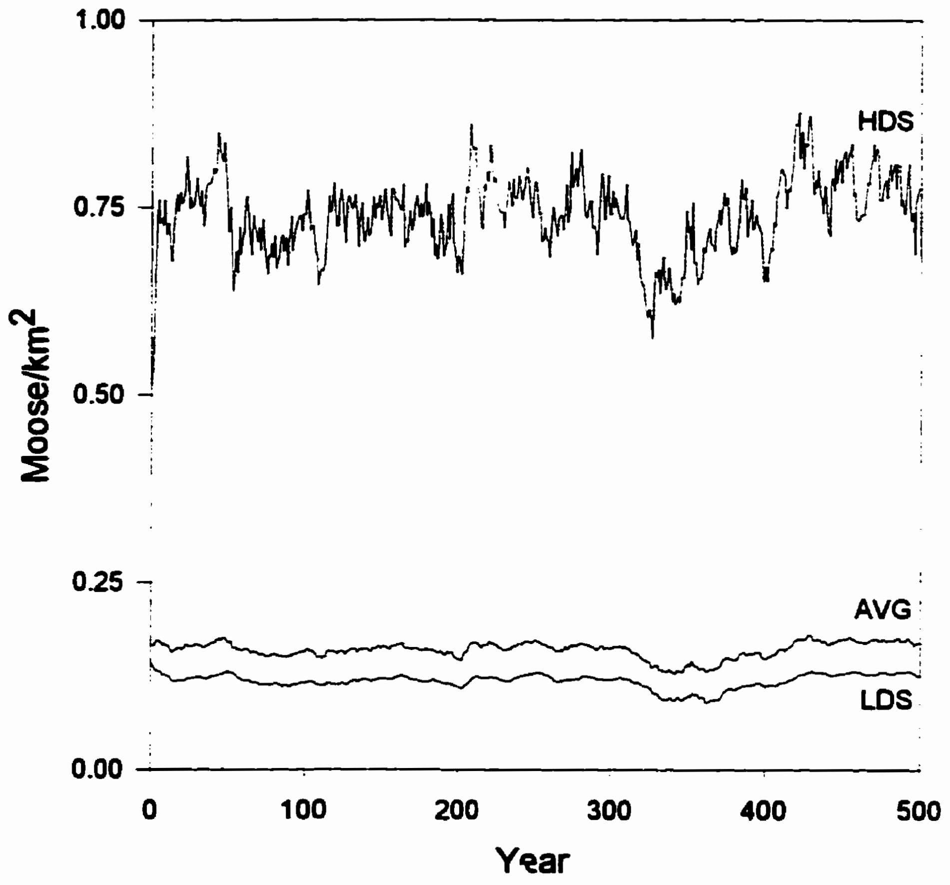

Figure 3-2. Simulated time series of moose density over 500 years. HDS is the high-density subpopulation in good habitat, LDS is the low-density subpopulation in poor habitat, and AVG is the weighted average based on area comprising the HDS and LDS habitats.

densities (SD) for the HDS, LDS and weighted average was 0.76 (0.05), 0.12 (0.01), and 0.16 (0.01) moose/km^2, respectively, and the final densities were 0.76, 0.17, and 0.13 moose/km^2 for the HDS, LDS and weighted average, respectively (Figure 3-2). I based all further analysis on this adjusted model.

This system remained relatively constant over time. The HDS quickly increased from 0.50 moose/km^2 until it reached 0.75 moose/km^2, when it began to fluctuate around that density. I repeated this simulation 50 times and none of the runs ended in an extinction over the 500-year interval. The density for the HDS and LDS remained with in the bounds for high and low-density strata (0.51-0.97 moose/km^2 for high and 0.05-0.37 moose/km^2 for low). I believe that the simulated densities of 0.76 and 0.12 moose/km^2 compare well with the measured densities.

When I removed the LDS portion of the simulation, the parts that remained were the HDS moose subpopulations, growth of those subpopulations, and removal in the form of wolf predation following the LDE predation model. The HDS density increased from 0.5 to 0.75 moose/km^2, as in the two-subpopulation model, and was capable of persisting over time without the LDS part of the system.

DISCUSSION

The two-subpopulation simulation provides the possibility that moose can persist over time at low densities while experiencing wolf predation that is not density-dependent. The simulation suggests that persistence is possible if 1) the moose population is spatially divided into subpopulations; 2) some of the subpopulations exist in high-quality habitat, have higher productivity, and have higher potential growth rates; and 3) wolves concentrate their predation on only the high-density, high-quality part of the subpopulation. Removing the LDS part of the simulation indicated that persistence depends for the most part on the HDS, and the LDS does not necessarily act as a refuge.

There is little information available on the migration of moose. Rolley and Keith (1980) present the only real relationship of density-dependent migration. However, this was not directly measured, but inferred from population growth rate, fecundity, and survival information. One problem with using this migration relationship in the simulation as I have is that it provides net rates of movement, the rate of leaving a subpopulation subtracted from the rate of entering that subpopulation. In the simulation I have used this as a gross rate for each subpopulation, the rate of leaving only. This means that I probably have an underestimate of the true rate of leaving a subpopulation, and it suggests that true movement of moose between subpopulations might occur more rapidly than it does in the simulation. With this in mind, I feel confident about adjusting the LDS emigration rate to increase rate of movement from the LDS to the HDS. This is probably a closer representation of the gross rather than the net migration rate, and in a biological sense could represent selecting areas of better habitat.

The manner in which I modelled migration might suggest that moose choose between habitats of differing quality. High-quality habitats provide forage and cover that benefit moose. Because moose concentrate in these areas, there is higher intraspecific competition for food and space, and higher risk of wolf predation because wolves concentrate their hunting efforts where there is a higher availability of prey. The trade-off is poor habitat with poor forage and cover, making moose more susceptible to disease, starvation, and stress from severe weather, but less susceptible to predation by wolves. This trade-off could take two forms. Either a moose chooses one habitat entirely over the other, or it chooses how much time to spend in a good versus a poor habitat. Ideally, there would be a proportion of time spent in each habitat that optimizes the benefits of good habitat but minimizes predation risk. Those moose that are forced through competition to spend more than the optimal amount of time in poor habitats could become more susceptible to predation for the time that they do spend in the high-quality habitat.

I believe that describing moose populations as tending to aggregate in small areas is reasonable. Moose select favourable habitats where they find adequate forage and cover. Since habitats themselves are heterogeneous, moose responding to that heterogeneity should then end up in clumps. Evidence for this response can be seen in standard stratified moose survey data. For areas in the Yukon that have low moose densities, such as the Mayo survey area (Ward and Larsen 1994), moose density estimates for the entire area is 0.12 moose/km^2. However, the estimate for the high-density stratum alone is 0.51 moose/km^2. If densities and dynamics change considerably over the landscape, then modelling dynamics according to large-scale average density might not be appropriate.

Supporting evidence for wolves responding to locally high densities of moose comes from various sources. For example, Huggard (1993) found that wolves move quickly between areas where prey are predictably available, thereby increasing their efficiency in encountering prey. Kills that occur in other than these predictable areas Huggard (1993) considers random kills of prey encountered as wolves moved between predictable prey areas. While Huggard's (1993) study was of wolves in Banff National

Park hunting primarily elk (*Cervus elaphus*), these findings suggest similar behaviour could occur when moose are the prey. Wolves could concentrate on hunting where they have learned that moose are at higher densities and more readily available, rather than search their entire territory.

Hayes (1995) studied wolf predation on moose in the Yukon, and measured kill rate values that were much higher than those predicted by the hyperbolic functional response model presented earlier in this paper (theory predicts 1.3 to 1.7 moose/wolf/100 days for densities of 0.25 to 0.43 moose/km^2, while Yukon kill rates were 2.2 to 3.4 for the same densities). He concludes that wolves might concentrate their predation effort and even cause local extinctions that are not observable given current moose survey methods.

The findings of Huggard (1993) and Hayes (1995) suggest that wolves are always experiencing prey densities that are higher than the average density for a large area, because they concentrate their effort where prey is most available. This means that rather than relating kill rates and predation rates to density over a wide area, as those found in moose surveys, it might be more appropriate to relate these rates to the local densities of prey that wolves directly experience. At the patch scale, wolf predation might be more clearly found to be density-dependent or even regulatory. Heads and Lawton (1983) found that they only could detect density-dependent mortality on holly leaf miners (*Phytomyza ilicis*) caused by a larval parasitoid (*Chrysocharis gemma*) when they used a sample unit size that was smaller than 1m^2, with the smaller sized sample units showing stronger density dependence. From this they define a patch as an area at a scale where the grouping of prey shows density-dependent predator-caused mortality.

Wolves that intentionally travel to areas with high densities of prey, kill rates that are much higher than those predicted by theory, and problems in other species with patch-size and scale-dependent predation all point to the possibility that the dynamics of wolf predation actually operate at a much smaller scale than at the aerial survey-sized areas presented in most wolf predation studies. At these smaller scales, density-dependent predation may be clearly evident. Because research often occurs at the "study area" scale, such relationships are not so clearly seen.

CONCLUSION

The simulation presented here suggests that in systems with moose and wolves, moose need not experience density-dependent predation to persist at low densities over many years. If moose populations are spatially divided into groups, some of those groups are high and low density, and wolves concentrate their predation effort on the high-density parts of the population, then an anti-regulatory total predation model can still result in long-term persistence of moose. These ideas suggest that further wolf-moose predation research should concentrate at smaller scales to look at how moose and wolves respond to their immediate surroundings.

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CHAPTER 4. POWER ANALYSIS OF WOLF-MOOSE FUNCTIONAL RESPONSES

This chapter discusses the ability of researchers to distinguish between different shapes of functional response in the predator-prey dynamics of wolf-ungulate systems. Here, I make the argument that while functional responses are important for fully understanding how wolves respond to ungulates, and consequently what their predatory effects are on ungulate populations, the ability of researchers to empirically distinguish among potential functional response models is severely limited due to poor statistical power. Because of this problem, alternative methods need to be considered when trying to determine the effects of predation on ungulate populations.

Functional responses have been argued to be a very important part of the interaction between wolves and their prey (Messier and Crête 1985, Theberge 1990, Dale et al. 1994, Messier 1994). A functional response that can cause stability in a wolf-ungulate system will allow for persistence of ungulate populations over time (Oaten and Murdoch 1975). According to conventional predator-prey theory, persistence occurs if predation is density-dependent, meaning that proportion of the ungulate population killed increases as ungulate density increases. If this condition exists, predation will have the potential to regulate an ungulate population to an equilibrium density.

Wolf predation can be divided into functional and numerical responses (Solomon 1949, Holling 1959, Dale et al. 1994, Messier 1994). The functional response deals with how an individual wolf changes the rate that it kills prey as prey availability changes. The other describes how wolves change their numbers in response to prey availability. Functional responses for wolves are usually described as type II or type III responses (Dale et al. 1994, Messier 1994). The type II curve is a continually decelerating hyperbolic curve (Figure 4-1). As prey becomes more available at low densities, a predator quickly increases its kill rate of that prey. As prey continues to increase, the predator starts to become limited by handling time and the functional response plateaus. The type III response (Figure 4-1) operates in a similar way; however, at low densities a predator kills prey at an increasing rate before the kill rate begins to plateau because of predator satiation. This results in a curve with a sigmoid shape.

Researchers have been trying to distinguish between type II and type III functional responses in wolf-ungulate systems because the shape of the functional response that wolves show has important implications to the dynamics of the ungulates on which they prey. A type III functional response implies stability in wolf-ungulate systems, where stronger sigmoid shapes promote greater stability. If the numerical response is density-independent or lagging (conditions that promote instability in wolf-ungulate systems), a type III response will still produce a density-dependent, regulatory predator response, because of a decreasing predation rate at very low densities (Dale et al. 1994).

In the past, most studies looking at functional responses of predators relied on visual inspection to evaluate the functional response type (Murdoch and Oaten 1975, Hassel et al. 1977). Others tried to establish the inadequacy of a particular model shape by fitting that model statistically and seeing a non-significant fit (Hassel et al. 1977). One problem with this approach is that in highly variable systems, such as wolf-moose systems,

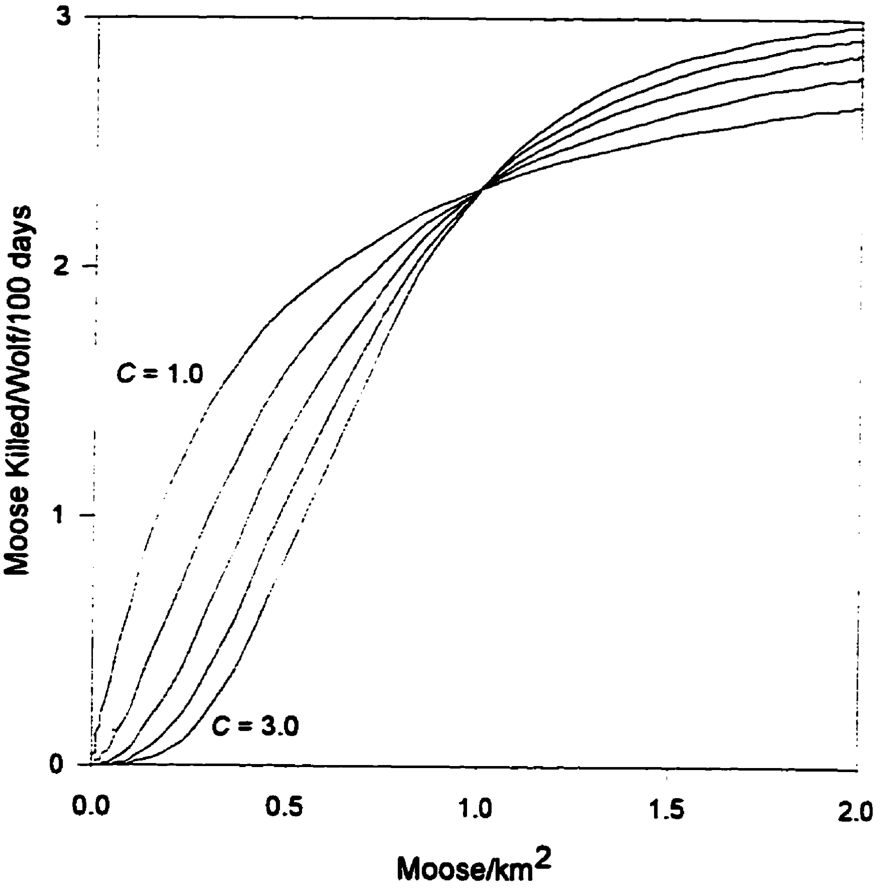

Figure 4-1. Functional response of wolves to moose. Equation is $y = 3.36x^C / (0.46 + x^C)$, with C taking the values 1.0, 1.5, 2.0, 2.5, and 3.0. $C = 1.0$ produces a type II functional response. $C > 1.0$ produces a type III functional response.

researchers can easily find a non-significant fit, even if the underlying distribution can be described by the model (Livdahl and Stiven 1983).

Dale et al. (1994) and Messier (1994) analysed wolf functional responses for evidence of prey-switching resulting in a type III response. These studies rejected this possibility because: 1) they judged their data to follow a type II curve, based on the distribution of data points, or 2) the type II and III models fit equally well, but the type II model was chosen because of parsimony (Trexler et al. 1988). In both of these cases, neither researcher considered how likely they would detect a type III curve if it really existed with the sample size and variance that they observed. In the case of Dale et al. (1994), they looked for a type III curve by plotting proportion killed per wolf by caribou density, where a region of positive slope would indicate a type III curve. Although this analysis was more convincing than using number killed per wolf, it still relied on visual inspection of data points, and not on a statistical method.

METHODS

Researchers often attempts to estimate some of the parameters of a population under study. The estimates obtained have some measure of certainty in their closeness to the true values, but these are only probabilities. The closeness of an estimate to a true value cannot be evaluated absolutely without the complete enumeration of that population, and this is usually impossible. One way that can allow a researcher to evaluate how close an estimate is to a true population value, and consequently how well a particular statistical method estimates those parameters, is to use simulated data. I used a method of data simulation similar to that of Trexler et al. (1988) to evaluate the power of distinguishing between shapes of functional response curves for moose-wolf systems.

The functional response model I used for this was the modified Michaelis-Menton equation $y = Ax^C / (B + x^C)$, where A is the asymptote of the functional response, B is the values of x when $y = A / 2$ (how quickly the line rises to the asymptote, or the efficiency of the response), C determines the shape of the curve at low density (for hyperbolic, $C = 1$; for sigmoid, $C > 1$), x is the density of moose (individuals/km^2), and y is the kill rate (moose/wolf/100 days). I used SAS statistical software (SAS Inst. Inc. 1989) to simulate data and then analyse them. The computer-generated data set came from a known model with a predetermined variance (the source model), and I used non-linear regression (Proc NLIN, SAS Inst. Inc. 1989) to fit a curve to that data set using the same general model, but with the parameters to be estimated. This SAS procedure has several methods for fitting a line to nonlinear data. I chose the simplest of these, DUD, a derivative-free method. Because Proc NLIN is an iterative least-squares method, it requires initial values for the parameters to be estimated. For the initial values I set $A = 3.0$, $B = 0.5$, and when fitting the sigmoid model, $C = 1.5$. For the hyperbolic model, C was not included. These initial values should be as close as possible to the final estimates, because initial values that are too far from the true values converge on other estimates, which are often orders of magnitude from the true values. For empirical studies, these initial values would be estimated visually from plots of functional response data.

The source model for the data was a modified Michaelis-Menton equation with the parameters $A = 3.36$, and $B = 0.46$ (Messier 1994). I began by setting $C = 1.5$. I set the mean square error value to 0.30. I calculated the functional response in the same way as

Messier (1994) did for the same set of points, and found the data around the model had a MSE of 0.49. I chose to be conservative by setting this value lower. I randomly chose several moose densities from a uniform distribution ranging from 0 to 2 moose/km^2 using the SAS function RANUNI (SAS Inst. Inc. 1989), and then calculated the appropriate kill rate according to the source model. Next, I added an error to the calculated kill rate using the RANNOR function (SAS Inst. Inc. 1989) based on the MSE value of 0.30, which scattered the points around the source model.

With this source model established, I generated 1000 data sets and fit a type II and a type III model to each data set. I used Fisher's F-test for full and reduced models to determine if a type III model fitted each simulated data set better than a type II model. I then summarized the proportion of times that I could statistically distinguish between type II and type III curves at $\alpha = 0.05$. For each group of 1000 simulations I varied the number of points generated from 10 to a sample size necessary to distinguish between a hyperbolic and sigmoid curve 80% of the time. I called this the necessary sample size. I repeated the analysis with C = 2.0, 2.5, and 3.0 to see how necessary sample size changed with the degree of curvature of the sigmoid model.

RESULTS

My ability to distinguish between type II and type III functional responses was low. With a curvature of $C = 1.5$, the necessary sample size was over 300 (Figure 4-2). At 300 points, the probability of making the correct conclusion was 0.75. As the amount of curvature increased, necessary sample size decreased. For $C = 2.0$, necessary sample size was $n = 109$; for $C = 2.5$, $n = 55$; and for $C = 3.0$, $n = 38$. For all cases except $C = 1.5$, the

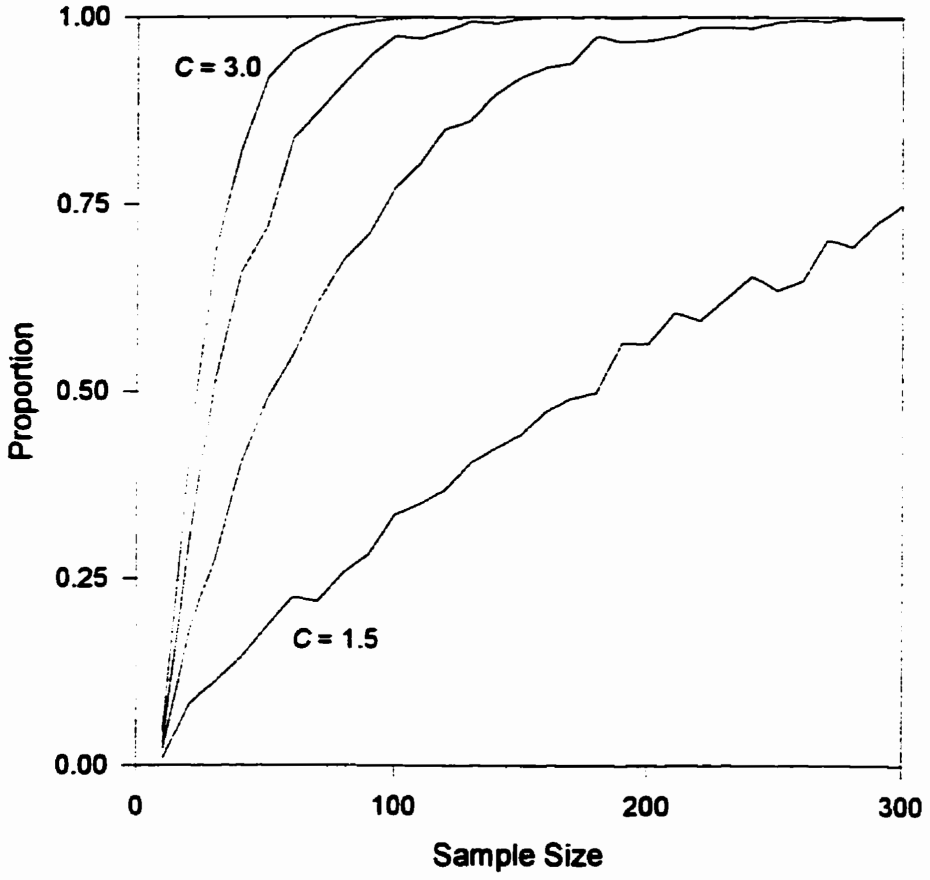

Figure 4-2. Effect of sample sizes on proportion of times that I correctly distinguished between a type II and a type III functional response.

proportion of correct conclusions increased rapidly with sample size and then plateaued. At $n = 300$, all cases except $C = 1.5$ had a proportion of at least 0.99.

DISCUSSION

In general, the power analysis shows that, given sample sizes and variance of wolf-moose predation studies, the power of distinguishing a type II functional response from a type III functional response using nonlinear least-squares regression is very low. Unless I had very large samples or extreme curvature, rarely was I able to distinguish between the two functional response types. Even for $C = 3.0$, a very strongly sigmoid model, I needed almost 40 data points, which is a very large sample for most wolf predation studies.

Statistical power is defined as $1-\beta$, where β is the probability of rejecting the null hypothesis when it is true. Then power is the probability of rejecting the null hypothesis (in this case, that both models fit a data set equally well) when the alternate is true, or the probability of concluding that the sigmoid model fits better when the data came from a sigmoid model. This power depends on several things including the amount of variance in the system, the sample size and the amount of difference in the alternative hypothesis. For small samples, high variance and small differences, power is very low. Low power seems to be a problem in wolf moose studies that cannot be easily remedied. Because sample sizes are often based on agency budgets, and not measurements of variability, increasing sample sizes may not be an option in many situations, particularly if sample size must be in the hundreds. Unfortunately, the other two aspects of power, variability and the differences between values in the tested hypothesis, cannot be changed for they are part of the nature of the system under study.

Sample Size Difficulties

Insect and large mammal predation researchers have very different standards by which they use replication in their studies. Trexler et al. (1988) presents a summary of several laboratory insect predation studies where they run many independent trials at the same availability of prey. At any particular availability of prey there were as many as 20 different predators allowed to forage among the same densities of prey to produce 20 independent measurements of kill rate at a single prey density. However, these studies occur in an environment where researchers have strict control over the predator and the availability of prey. For wolf-moose studies, replication involves measuring kill rates of several packs at a single moose density in a single study area and treating each pack as an independent observation of kill rate; replication is of wolves or packs, but not of densities of prey. Obviously, this reflects the difficulty and cost of replicating studies of wolf predation in different areas with the same moose density. However, using replicate packs might not be statistically correct because of the influence a pack may have on its neighbours. Each pack is not an independent sample. For the data in Table 2 of Messier (1994), each point is an average kill rate based on several packs, and included two kill rate measurements at one moose density, but only one kill rate for the other reported moose densities. Dale et al. (1994) had a single kill rate for a particular density, and used 3-4 wolf packs over three years in one study area. Messier (1994) and Dale et al. (1994) have the only two papers that have several measurements of wolf kill rate over a range of prey densities, but they illustrate the differences between insect and mammal predation studies, and the inherent difficulties with statistical power that large-mammal researchers face.

Variance Difficulties

Juliano and Williams (1987) found that for insect predation studies, using the average of the kill rate for any density of prey and the individual observations produce similar point estimates for the parameters in functional response equations. However, using average kill rates produces underestimates of the standard errors around the parameter estimates, and so gives an investigator the false perception of precision that is higher than it probably really is. As a result, hypothesis tests concerning the value of parameters are more likely to conclude a difference when there is no difference (Juliano and Williams 1987). This could be a potential problem with Messier's (1994) analysis of functional responses, since the *B* (response efficiency) parameter is not significantly different than zero. This makes the validity of the significant parameters suspect, and so the model might not appropriately fit the data (chapter 2). This would also indicate that my use of 0.30 as MSE in my simulations was a very conservative value. Considering the measured MSE from Messier (1994) was 0.49 and that this was based on average kill rates, the true variability in the system is actually much higher. This means that my estimates of power are very hopeful and probably much higher than the true power.

Because of the possible problems with variation outlined above, I evaluated the variation in kill rates from the original pack data in wolf-moose systems to explore which alternatives might be most feasible when trying to measure functional response in wolves. I went to the original studies from which Messier (1994) gathered predation data, and also added kill rates from studies completed since 1994. The studies that presented detailed kill information for each pack usually presented the size of the pack under study, the number of days that the pack was studied during the winter, and the number of moose and other prey that the pack killed. From this information, I computed a kill rate in units of moose/wolf/100 days. Even with the problems of independence between packs, I chose to stay with the pack-year as the sample unit, because it is common to wolf predation studies. Packs that were studied in the same area were considered independent observations, and the same pack that was studied more than one year was considered more than one independent observations. This was to provide a "best-case scenario" with respect to possible sample sizes.

Using these kill rates, I fitted a type II functional response model to evaluate the variance (MSE) around the line, but not to estimate parameters for that line. Because of the high variance in kill rates, the pattern in no way resembled the hyperbolic shape of the type II response. The MSE was 6.97 (with pairs included as packs) or 1.51 (without pairs). This compares to 0.49 for when using the averages for each study that Messier presents. I also compared ranges of kill rates to the averages used by Messier (1994). The lowest and highest kill rates were 0.18 and 12.50 moose/wolf/100 days (with pairs) or 0.68 and 6.73 moose/wolf/100 days (without pairs). By comparison, ranges of kill rates for the averaged data was 0.37 to 3.75 moose/wolf/100 days. If my summary indicates a "realistic" variance, as compared to the "hopeful" variance of my simulation or the averages used by Messier (1994), then it seems clear that detecting slight changes between model shapes, even with the help of additional sample size, is extremely unlikely. Using averages for each study appears to be the only way to reduce the variance enough to get any idea of shape of the functional response of wolves on moose. This idea of shape can be at best a general idea, for the ability to detect detailed shape changes with density will be lost in the variance.

However, one striking pattern that the pack kill rate data show is higher variances at low densities of moose. This pattern appears to be opposite to what is found in insect functional response studies, where the variance increases with availability of prey rather than decreases as in wolf-moose studies. Insect functional response studies usually evaluate their variance using the coefficient of variation (CV), calculated at each availability of prey at which the researchers measured kill rate. Trexler et al. (1988) reviewed several laboratory insect predation studies and found that CVs around a mean kill rate at a particular prey availability ranged from 18% to 165% with the lower CVs usually accompanying the estimates at lower prey availabilities. Comparison to Messier's (1994) functional response data was difficult, because there were only single values of kill rate at most densities of moose (there were two at 0.23 moose/km^2), rather than several values at a single prey density in the insect studies. This meant that I could not determine a CV at a single density of prey. To attempt a comparison, however, I divided Messier's (1994) data set into kill rates above and below 0.5 moose/km^2, and determined a mean kill rate and standard deviation for each. The resulting CVs where 59% below 0.5 moose/km^2, and 31% above. Of note here is that the higher CV occurred at low moose density, a pattern, as stated earlier, that is opposite of that found in insect predation studies. This is important because, even though the CV for the wolf-predation data is not that large, as compared to the range of CVs for insect studies, at low densities there is a considerable difference (56% versus 18%), and low-density is where the critical region is for distinguishing type II from type III functional responses. Trexler et al. (1988) could not objectively pick between type II and type III curves as providing the best fit when comparing fits of various models (both hyperbolic and sigmoid) to various insect functional response data sets found in the literature. Trexler et al. (1988) also simulated several data sets with 90-110 data points collected at 9-11 different densities of prey, and with a constant CV of 20%. With these simulated data Trexler et al. (1988) could only distinguish between type II and type III functional responses if the kill rates were intermediate to high (with the plateau for intermediated kill rates being about 10 prey killed per trial). These results are not easily comparable with wolf-moose systems; however, these insect studies occur in controlled laboratory conditions, have relatively low variance, and the researchers still have difficulty objectively determining if functional responses are type II or type III. Given this, it seems unlikely that a statistical discrimination between type II and type III functional responses could occur in highly variable natural-system studies involving wolves and moose. Because of the limitations of determining functional responses in large predators, alternatives need to be considered.

Alternatives to Measuring Functional Response

Proportion versus Number of Prey.--Rather than looking at a change in the number of prey killed per predator and using a non-linear, least-squares technique, Trexler et al. (1988) suggest an alternative approach for analysing functional response data. This involves looking at the change in the proportion of prey killed out of the total population available. The shapes of these curves for type II and type III functional responses are much easier to distinguish (Figures 4-3c and 4-3d) than when using numbers of prey (Figures 4-

3a and 4-3b). This technique uses logistic regression rather than least-squares regression and will identify an increase in proportion killed per predator (Trexler et al. 1988). This method works by using prey availability as the dependent variable and the response is either a capture or a non-capture of a prey individual. The function then predicts probability of capture of prey individuals given an availability of prey. This approach was originally conceived for laboratory predation experiments, where all individuals in a prey population can be watched. Because this is not possible for moose populations, a solution is to use radio-collar mortality data to determine instances of capture or non-capture.

Radio-collar mortality in moose could also be used to assess predation changes with changing moose densities. Boutin (1995) suggests this approach for studying small mammal dynamics where prey population densities can change a great deal over only a few years. However, Boutin's (1995) approach is to study total predation rate changes with density of prey, not functional response changes alone. If wolves could be kept constant while studying predation rate (as during a removal experiment), then any changes in predation rate on collared moose that occur with moose density could be attributed to the functional response. If density of wolves cannot be kept constant, then looking at predation rate using radio-collared moose would still show how predation rate changes with moose density.

Radio-collar studies to look for density-dependent predation could be used in manipulative experiments involving wolves and moose (see Figure 1 in Boutin 1992). I

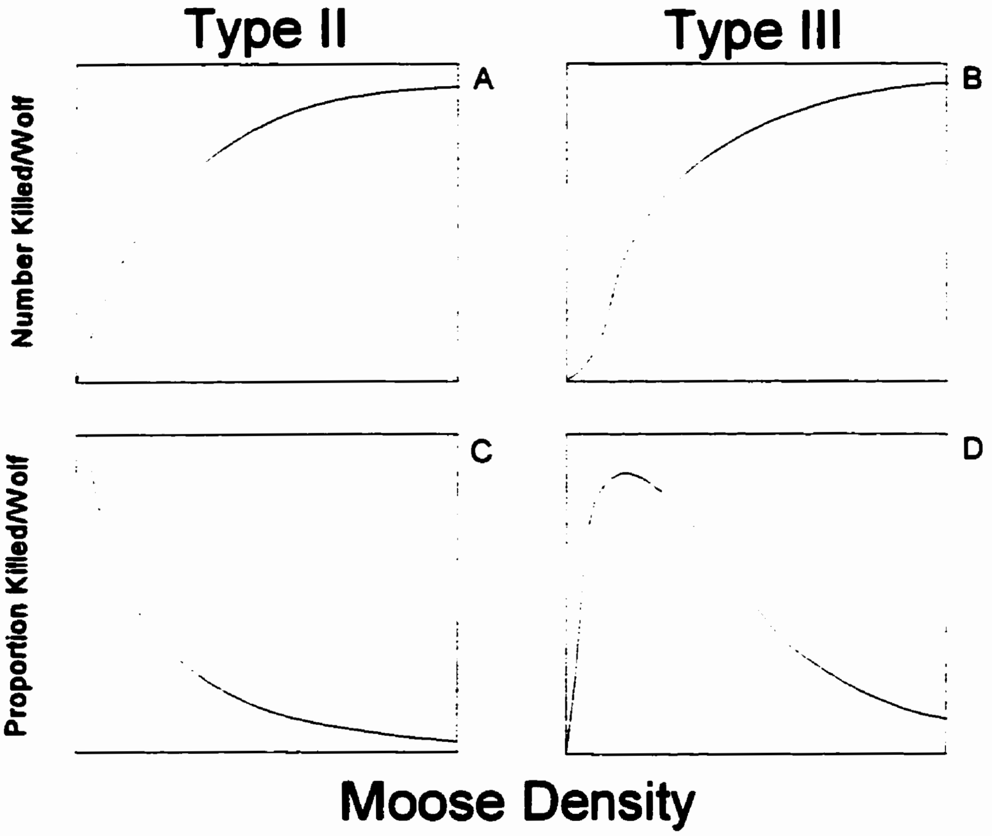

Figure 4-3. Identifying functional responses using proportion of prey killed rather than number killed.

propose two manipulations. The first is to remove wolves while studying radio-collared moose and compare predation rates on moose directly before wolf removal to predation rates after removal has stopped and wolves have returned to their naturally-regulated densities. If moose densities have increased during wolf control, then density-dependent predation will be indicated by an increase in mortality rate due to wolf predation from periods before to after wolf control.

The second manipulation is the reduction of moose in areas where they are at intermediate densities (Boutin 1992). If predation is density-dependent, two things should happen: 1) mortality due to wolf predation should decrease as moose density decreases; 2) when moose are no longer reduced, they should increase to their original pre-reduction density. Reductions of this sort could occur with harvest management, having areas with higher or lower moose harvest (Messier and Crête 1985) while carefully measuring moose densities and mortality. Although these manipulations would not address functional response directly, they would still provide a picture of the nature of wolf predation on moose populations. If wildlife biologists began to look at predation using mortality rates, then analysis of wolf functional responses would no longer be necessary. Density dependence could be measured directly by mortality on the prey rather than indirectly through behaviour of the predators.

Scat Analysis.--Some researchers have used predator scats to evaluate functional responses [see Boutin (1995) for examples in small mammal studies]. Such an approach could also be taken with wolf scats with certain limitations. Scat analysis could be used to get a general idea of the shape of the functional response (type II or III), but not what the actual kill rates are at changing densities (Boutin 1995). To explore this possibility, I gathered scat data from studies that presented moose densities (x) and contents of scat as percent occurrence of moose in the scat (y). I began by treating packs and years as independent samples. The resulting hyperbolic model [$y = 61.2x / (0.11 + x)$, Figure 4-4a] explained 53% of the variation, and both the A and B parameters were significantly different ($\alpha = 0.05$) than zero. A sigmoid model explained 54% of the variation, but the B and C parameters where not significantly different than zero.

Because of concern about independence between points from the same study, I repeated the analysis using only average percent occurrence of moose for a study over packs and years for the same moose density. In this analysis, the hyperbolic model [$y = 66.7x / (0.20 + x)$, Figure 4-4b] explained 65% of the variation, and both the A and B parameters were significantly different ($\alpha = 0.05$) than zero. For the type III model, none of the parameters where different than zero.

Given the limitations of using scat analysis, it still seems to provide some picture of the functional response of wolves to moose. A sigmoid shape was not detected even with values from studies where moose were not the primary prey (Huggard 1993, Larter et al. 1994). Given the results of my power analysis, I would not expect a sigmoid shape to be detectable without a much larger sample size. But gathering enough data to analyse the possibility of a type III response using scat analysis is considerably less expensive than following radio-collared wolves and recording kills. For scat analysis to show a sigmoid shape, studies must include densities of moose that are extremely low to see the sigmoid portion of the response. If this sigmoid shape is due to switching, scats must be collected

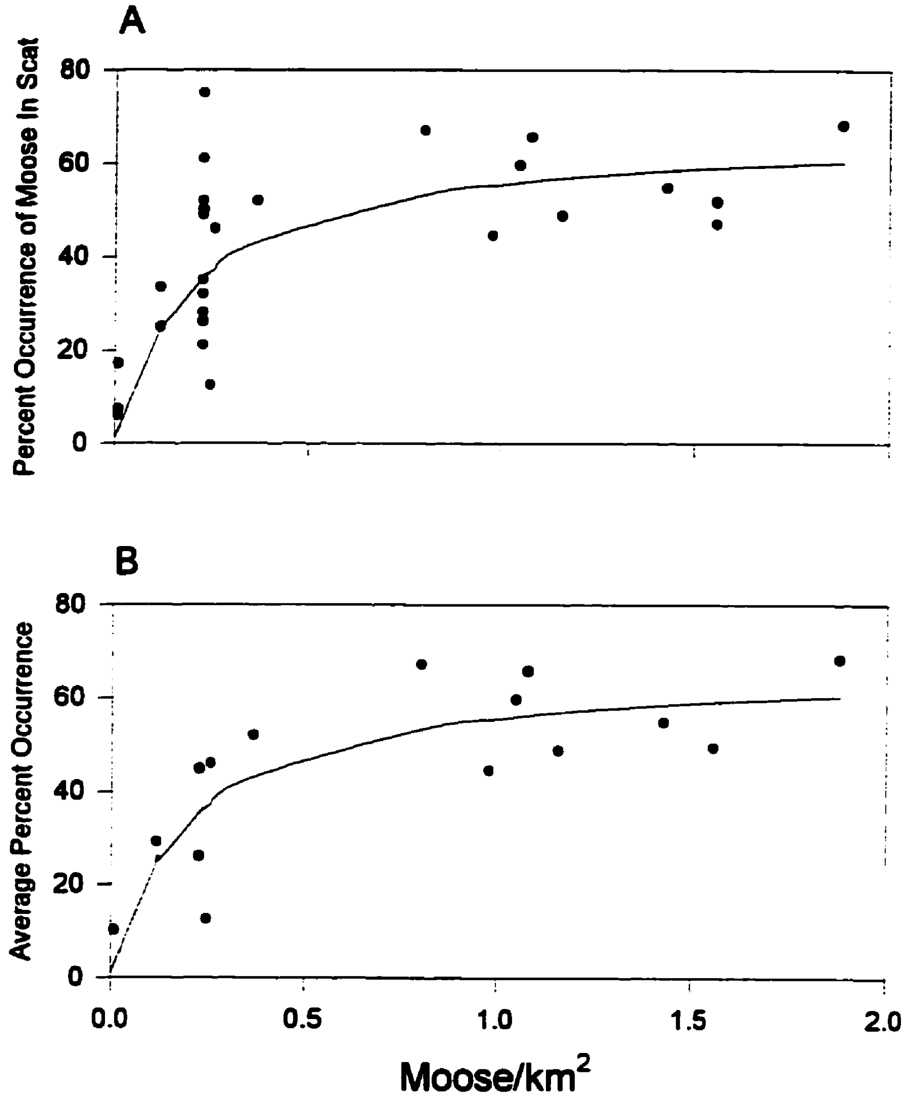

Figure 4-4. Hyperbolic functional response model fitted to percent occurrence of moose in wolf scat. A) Model using all percent occurrence values as reported in the literature. Equation is y = 61.2*x* / (0.11 + *x*). B) Model using averages within a study across packs and years for the same density of moose. Equation is y = 66.7*x* / (0.20 + *x*).

where moose are at low densities, and where they are not primary prey.

So far I have considered the importance of functional responses only at low to intermediate densities, where the sigmoid portion of a type III response occurs. There is also the question of measuring functional responses at high densities, and the power associated with detecting a response that asymptotes. Researchers might assume that a plateau in kill rates is apparent at the higher ranges of densities for a prey species, but as with selecting a hyperbolic or sigmoid model at low densities, they do not test this assumption, or consider what sample size is needed to be able to establish with any certainty that an asymptote occurs. Given a certain sample size and variance, what is the probability of distinguishing a functional response that asymptotes from a response that continues to increase? This will depend on the rate at which kill rates continue to increase, or the difference between an asymptoting curve and a continually increasing curve. However, given the results of this paper, I believe that at high densities, just as at low densities, high variance in the system will make it necessary to collect far more data to be certain that a functional response asymptotes than can be reasonably collected in a large mammal predation study. According to theory, kill rates should plateau as predators become satiated, but Boutin (1995) gives examples of how the functional response may continue to increase even at the upper limits of prey abundance because of surplus killing and partial consumption. If kill rates continue to increase, then it may have implications about how predation operates at high densities, as is suggested by the two-state or multiple equilibrium model (Sinclair 1989). Continually increasing kill rates might preclude the possibility of a high-density stable equilibrium.

CONCLUSION

Type III functional responses allow for density-dependent predation by a predator, even when the numerical response is density-independent. Because of the high variances and low sample sizes of wolf predation studies, a sigmoid functional response is unlikely to be found empirically. This means that biologists studying wolf predation, should consider alternatives to measuring functional responses when evaluating predation effects on a prey population. The most promising of these alternatives involves measuring mortality to wolf predation using radio-collared moose over various densities.

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CHAPTER 5. CONCLUSION

Large mammal systems in general are highly variable and very costly systems from which to gain information. Only recently have enough data been gathered about wolf predation on moose to provide any idea of the dynamics occurring in that system. However, much of this knowledge has come from other predator-prey systems of which researchers have a far better understanding: insect predation, host-parasitoid, and small mammal systems. Making generalizations across systems could cause problems if the primary factors that influence dynamics are different from system to system. That means that researchers studying large mammal systems must be particularly rigorous when trying to determine the dynamics, and they must be careful about the assumptions they make in the absence of information about the system. This is particularly important in systems where an ungulate species is to be managed for sustainable harvest.

A re-analysis of numerical and functional response data from Messier (1994) showed that linear models and data transformation allowed for a better statistical fit of wolf predation data than did theory-based hyperbolic models. When I combined these models into a total predation model, I found that predation rate was anti-regulatory, being inversely density-dependent at low densities of moose, and then showing weak direct density-dependence at intermediate to high densities of moose. This finding was opposite to the findings of Messier (1994), where predation was density-dependent at low densities and then inversely density-dependent at intermediate to high densities. As a result, the model I produced showed no low-density equilibrium. Rather, it suggested that moose populations that decrease to low enough densities could be driven to extinction.

This possibility of extinction could only occur if moose populations are considered as homogenous units that are evenly spread over a study area. Evidence from moose survey reports suggests that moose tend to aggregate in favourable habitats, and so have higher local densities of moose than surrounding, less favourable areas. Further evidence from wolf predation studies suggests that wolves detect these locally abundant pockets of moose and spend a majority of their time foraging in them (Huggard 1993). A simulation model based on theses two ideas and the linear functional and numerical responses indicates that persistence of moose at low densities is possible without density-dependent predation if: moose populations are divided into subpopulations, some have high growth rates and densities, and wolves concentrate their predation on only the high density subpopulations. Persistence then occurs because the high-density regions are at densities or have growth rates higher than that are affected by anti-regulatory wolf predation. In addition, the low densities reported in moose surveys are averages of large, low-density areas, and small, high-density areas.

Although my re-analysis does not support a functional response that intersects zero, this still must be the case, for the simple fact that wolves cannot kill prey when they are absent. If, however, data can be collected to show more of a conventional hyperbolic functional response, researchers must next be able to look at another level of resolution: is the functional response hyperbolic (type II) or sigmoid (type III)? Using simulated data sets, I found that making this distinction in large-mammal predation studies will be very difficult due to low statistical power, through small sample sizes or high variability in the system. This means that alternatives that have a greater distinguishing ability (using proportion killed rather than number killed per wolf), or that avoid the functional response altogether (using radio-collared moose) should be pursued in further studies of wolf-moose dynamics.

The sum of these re-analyses indicates that there are many assumptions that researchers studying wolves and moose take for granted. Often these assumptions must be made because of the lack of available information, and the cost involved in gathering that information. However, making assumptions can bias conclusions about a system's dynamics. As I have shown here, the models of population dynamics, and the management decisions based on those dynamics, can change considerably depending on which assumptions a researcher chooses to maintain.

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Appendix A. Information used for variance in kill rates based on pack size.

Study	Moose/km^2	Pack Size	Moose killed/wolf/100 days
Ballard et al. 1987	0.33	9	3.23
		8	2.08
		7	0.68
		7	3.13
		2	5.47
Bjorge and Gunson 1989	1.3	7	1.96
Fuller and Keith 1980	0.23	9.2	2.4
		10.5	1.9
		9.8	2.1
		8.5	0.9
		6.5	2.7
Hayes 1995	0.25	17	2.52
		2	9.68
		2	4.84
		5	3.33
		6	3.51
		2	2.63
		2	5
		11	1.26
		6	5.21
		4	4.49
		2	10
		2	10.71
	0.3	2	5.56
		2	12.5
		2	8.3
		2	3.13
		7	1.79

Appendix A (continued). Information used for variance in kill rates based on pack size.

Study	Moose/km^2	Pack Size	Moose killed/wolf/100 days
		2	11.11
		7	4.4
	0.36	2	8.93
		3	4.17
		9	1.59
		11	2.37
		2	7.89
		6	3.51
		2	4.35
		10	3
		10	2.08
		10	1.85
		2	5.26
		10	2.17
		2	7.14
		11	1.5
		11	1.93
	0.43	20	2.04
		11	1.4
		11	2.1
		2	4.17
		6	2.38
		5	2.86
		4	6.73
		13	2.66
Hayes and Baer 1986	0.42	5.5	1.74
Hayes et al. 1991	0.26	7	2.3

Appendix A (continued). Information used for variance in kill rates based on pack size.

Study	Moose/km^2	Pack Size	Moose killed/wolf/100 days
		5	3.53
		4	1.14
		7	1.39
		6	2.78
		2	5
		2	5.26
Peterson 1977	1.62	10	5.43
	1.5	8	3.27
		10	4.05
	1.56	8	3.52
		13	1.82
	1.61	12	2.5
		16	1.94
Peterson et al. 1984	0.8	2	3.35
		7	2.32
		8.2	1.43
		16.3	1.92
		11.2	1.26

Appendix B. Information used for functional response analysis using wolf scats.

Study	Moose/km^2	Percent occurrence of moose in scat
Fuller and Keith 1980	0.23	49, 35, 32, 21, 28, 61, 52, 50, 75
Average		44.8
Hayes et al. 1991	0.26	46
Huggard 1993, Paquet 1993	0.011[1]	17.0, 7.3, 6.0
Average		10.1
Larter et al. 1994	0.25	12.5
	0.12	33.3, 24.8
Average		29.1
Messier and Crête 1985	0.23	26
	0.37	52
Peterson 1977	1.56	47.0, 51.8
Average		49.4
Peterson et al. 1984	0.8	67
Thurber and Peterson 1993	1.16	48.7
	0.98	44.5
	1.05	59.5
	1.08	65.6
	1.43	54.7
	1.88	68.1

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[1] Density determined by best estimate of moose in Spray wolf pack's territory (12, Huggard 1993) divided by territory size (1058 km^2, Paquet 1993).