protein are present before the onset of diabetes in the BB rat is therefore of particular interest. Earlier we observed that antibodies to the 64-kD human islet cell component were present 2 years before clinical onset of IDDM in a twin brother of an IDDM patient. At that time islet cell cytoplasmic antibodies were still negative, and an increase in glycosylated hemoglobin A_{1c} was yet to be detected (*17*). We therefore speculate that the 64-kD component is a major target antigen in an immunopathological process that culminates in a loss of pancreatic B cells to such an extent that fasting blood sugar can no longer be controlled. Recent evidence from studies of the BB rat (*18*) suggests that immune intervention with cyclosporin A allows prevention of IDDM, provided that therapy was initiated before the onset of IDDM. This discovery has already been used to direct a similar attempt to prevent IDDM in humans (*19*).

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Wolves, Moose, and the Allometry of Population Cycles

Abstract. *After a decade of dramatic population fluctuations, protected populations of wolves and moose in Isle Royale National Park in Lake Superior returned in 1983 to the levels observed in the 1950's. Inherent lags in this predator-prey system and the strong recovery of the moose population following a wolf population crash suggest that these populations may continue to cycle with a period length of about 38 (95 percent confidence interval, ±13) years. Such a long-term cycle is consistent with the proposal that period length of herbivore population cycles will characteristically scale according to the fourth root of body mass, a basic allometric relation linking physiological cycles to population processes.*

From long-term studies (*1–3*) of protected wolves (*Canis lupus*) and moose (*Alces alces*) on Isle Royale in Lake Superior (an area of 544 km^2), we report evidence supporting long-term oscillatory behavior in a large mammal predator-prey system. Our data are consistent with the proposal (*4*) that herbivore populations should fluctuate at periods proportional to the fourth root of body mass ($M^{1/4}$).

The characteristic time periods of animal population cycles have not been explained. Widely recognized are 4-year cycles of microtine rodents (*Microtus* and *Lemmus*) and 10-year cycles of snowshoe hare (*Lepus americanus*) and ruffed grouse (*Bonasa umbellus*) (*5*). Cycle period has been considered proportional in some way to the generation time of a species (*6, 7*). Finerty (*5*) has queried why muskrats and lemmings, although closely related, exhibit vastly different cycle periods. Explanations for such variation have focused on different lag times for recovery of woody and herbaceous forage (*8, 9*).

It may seem obvious that large, long-lived mammals would fluctuate at longer intervals than smaller species, but the relation between body size and life cycles is so simple and pervasive that it can be overlooked (*10*). The dependence of cycle period on body size follows logically from the premise that animal cycles reflect the interaction of natality, mortality, and dispersal (*11*), coupled with the allometric scaling of physiological functions and life history characteristics to $M^{1/4}$ (*12–14*).

After wolves colonized Isle Royale in the late 1940's, their numbers stabilized, then fluctuated dramatically, culminating in a population crash in 1980 to 1982 from malnutrition and intraspecific killing. Their population returned in 1983 to the level observed 27 years earlier when first surveyed (*1*). The available data set suggests a period length of about 30 years for this fluctuation, which tracked

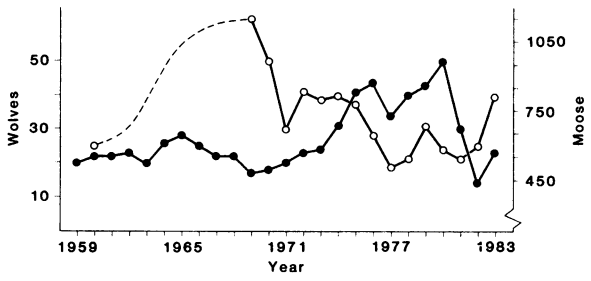

Fig. 1. Wolf (●) and moose population (○) fluctuations on Isle Royale, 1959 through 1983 (*2, 28*).

changes in the moose population. A strong moose recovery after the wolf die-off provides the potential for similar, recurring oscillations, reminiscent of cyclic fluctuations observed in some small animals (Fig. 1).

These findings led to a search for empirical generalizations applicable to cycling vertebrates of all sizes. The hypothesis that cycle period scales according to $M^{1/4}$ was tested by regressing observed cycle period against body mass for 40 species of birds and mammals. A previous analysis was limited to only nine species of mammals, and data on mammals larger than 2 kg was derived from irruptions of ungulates on islands (*4*). Poikilotherms (for example, insects) were not considered because their physiological cycles are influenced fundamentally by environmental temperature and variable periods of dormancy.

The regression equation for cycle period (t_p) on body mass (M) in kilograms, $t_p = 8.15M^{0.26}$ ($r^2 = 0.86$), provides strong empirical support for the dependency on body size of cycle period and population dynamics in general (Fig. 2). Moreover, this analysis provides evidence of the dominant role played by the rate of population increase in the dynamics of cycling species. Intrinsic rate of natural increase (r_m) scales according to $M^{-0.26}$ (*12*), suggesting a constant inverse relation between rate of increase and period length of population cycles. Rate of increase depends heavily on generation length (defined as the average age of females giving birth to all offspring) (*15*), which conforms to the basic allometric relationship between $M^{1/4}$ and physiological time (*16*). Thus, cycle period can be considered a function of generation length, with allometric equations predicting that cycle period will be approximately four to five times the generation length, which in turn is almost twice the average age at maturity (*16*).

Time lags in strong regulatory feedback mechanisms tend to produce an overshooting of equilibria and overcompensation, sometimes resulting in continual population oscillations (*8*). Notable lags are inherent in wolf-moose systems and have produced cycles in previous models of wolf-moose dynamics (*17*). An increasing moose population is characterized by large, vigorous animals that are less vulnerable to wolves until they are at least 7 or 8 years old, when age-related skeletal pathology begins to increase (*18*). This is the lag before food supply for wolves can increase. A longer lag, which will be rather site-specific, is the time required for an increasing moose population to reach a density limited by food, when a major increase in vulnerability to wolves occurs. Increased kill rates and wolf numbers can prompt a major reduction in prey. Wolves increase to a high plateau but face a dwindling food supply as culling of the prey population becomes complete. On Isle Royale, lack of vulnerable prey finally caused a crash in wolf numbers about 10 years after the moose decline began. When populations are modeled with a delay-differential equation (*8*), lags (t) of 7 to 10 years tend to produce cycle periods of about 28 to 40 years ($4t$), approximately the predicted cycle period of 38 ± 13 years (*19*) for moose-sized prey.

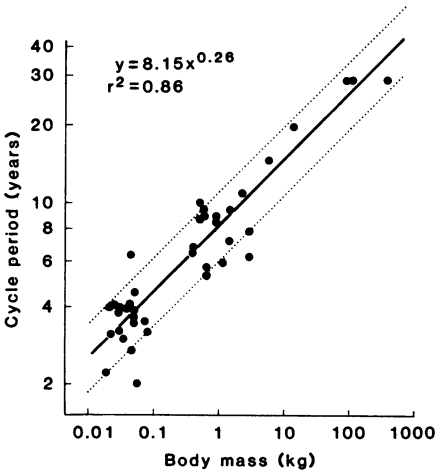

Fig. 2. Population cycle period as a function of body mass for 41 species of birds and mammals. The term "cycle" is used to indicate recurring population fluctuations at approximately regular intervals. The number of data points (populations) per species ranged from 1 to 19 (mean of 3.9 and standard deviation of 4.1). The 95 percent confidence interval for the power function exponent is 0.23 to 0.29 and the corresponding interval for predicted cycle period is shown (*28*).

Recognition of empirical generalizations among cycling species across the range of body sizes is likely to bring us closer to a common conceptual model and, at the same time, highlight the qualitative differences in population dynamics of large and small vertebrates. Many comparisons between the snowshoe hare and microtine cycles (*11, 20*) may be extended to wolf-moose dynamics.

As is often the case with small mammal predators, wolves were not able to prevent an increase in moose from low densities. As herbivore density increased, density-dependent herbivore-vegetation interactions led to a decline in population growth rate through low reproduction, longer maturation time, and higher mortality among adults (*2*). The specific nature of the moose-forage interaction that underlies moose vulnerability to wolves needs to be clarified.

It is thought that small mammal predators do not limit peak prey numbers (*11*). We believe, however, that moose at Isle Royale would have increased to a higher peak in the absence of wolves. In the early 1970's, when the moose decline began, wolves were killing moose in younger age classes than those that were dying of malnutrition, the only other major cause of mortality (*2*). Wolf predation, at all levels of moose density, accounts for a higher proportion of total prey mortality than is generally the case for smaller predators and prey (*20, 21*). Although typically 80 to 90 percent of moose mortality on Isle Royale can be attributed to wolf predation (*2*), we estimate that wolf predation could have accounted for only about 42 percent of the total loss of moose from 1969 to 1972, when adult losses directly to malnutrition were greatest.

Like small mammal predators, wolves may amplify and extend the period of low prey numbers (*22*). The Isle Royale moose population remained depressed until the wolf crash, allowing a general forage recovery.

The moose population began to increase immediately after the wolf decline. Calf abundance then increased more than twofold, reaching the highest level ever recorded on Isle Royale (*23*). We estimated that 44 percent of the moose alive in 1983 were born in 1981 and 1982.

It had been proposed that wolf predation on Isle Royale moose acted primarily to sensitively adjust moose density to limits set by forage (*2*). The decline of moose in the early 1970's followed a major shift of moose out of areas burned in 1936 and 1948 (temporarily used habitats) into mature forest with a stable but more limited forage supply. The forage limitation hypothesis predicted that moose would decline to a lower equilibrium, where they would remain until a major change occurred in the forage resource (for example, by fire). That prediction is clearly at odds with more recent empirical findings. A partial test of our findings on predator-prey cycles would be short-term predictions for wolf and moose populations: moose should continue to increase for several years, and wolves should remain relatively stable at 20 to 30 animals.

Elephants are thought to fluctuate at intervals of decades (*24*), but our analysis would predict a period of 71 ± 21 years (*19*) for oscillating elephant populations. Species with two or more different characteristic cycle periods may be considered as having a primary, internally driven cycle that can be predicted

allometrically plus secondary cycles that reflect external factors (*25*). For humans, life-span is unusually long relative to body size and is correlated with large brain size (*14*). Calculations based on the "encephalization quotient" for *Homo sapiens* (*26*) indicate that a human-sized brain is appropriate for an animal weighing 804 kg, from which we predict a period for population oscillations of 46 ± 15 years (*19*); this is approximately the period length of stable limit cycles predicted by models of human demography in 20th-century America (*27*).

Slobodkin (*7*) speculated 20 years ago that "population stability has some relatively simple relation to the physiological properties of the particular species." Allometric analysis of life history parameters and population processes should prove to be a unifying influence.

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Competition Controls the Growth of an Identified Axonal Arborization

Abstract. *The shape of the axonal arborization was studied in an identified insect sensory neuron. The distribution of presynaptic varicosities within an axonal arbor was shown to be modulated by the density of neighboring terminals. Removal of neighbors near one portion of the axon terminal increased the growth rate in the denervated region and caused a compensatory retraction in other regions. The results support the hypothesis that the size of an axonal arbor is determined intrinsically, whereas the distribution of varicosities within the terminal is determined extrinsically by neighboring terminals. These findings provide a direct demonstration of the effects of competition on an identified nerve cell, as well as one of the first examples of competitive interactions in an invertebrate central nervous system.*

Flexibility is a common feature of developing neuronal connections. In many parts of the nervous system, much of this flexibility is determined by the presynaptic terminal arborizations of the afferents (*1*). One commonly reported observation (*2*) is that neighboring terminal arborizations interact with one another in a manner that restrains their growth. Removal of neighboring terminals can release a cell from this restraint, allowing the arbor to expand or invade the neighbors' vacated territory. This restraint is usually called competition (*2, 3*). Most of the evidence for this phenomenon is indirect in the sense that the behavior of single terminals is inferred from the behavior of large populations (*1, 4*). We now demonstrate the effects of such competition directly by showing its role in determining the shape of the axonal arborization of an identified insect sensory neuron. The results show how competition differentially controls the growth rates along various regions of a single arbor, and they provide direct support for theories of the control of terminal growth (*2, 4*).

In insect sensory systems, the axonal arborizations of some identified sensory neurons can be stained by a simple method (*5*). Many insect sensory neurons are associated with external hairlike struc-

Table 1. Number of varicosities (*9*) (mean ± standard deviation) in region R (ipsilateral to the soma) and in region L (contralateral to the soma). Juveniles were in the third instar.

Group	Number	Number of varicosities		
		In region R	In region L	Total
Control	10	222 ± 57	150 ± 53	373 ± 73
Experimental	10	52 ± 20*	269 ± 36*	321 ± 51†
Juvenile	11	131 ± 35‡	91 ± 23‡	225 ± 35‡

*Difference from control values, $P \leq 0.01$, determined by *t*-test. †Not significant (*10*). ‡Difference from experimental values $P \leq 0.01$ determined by *t*-test.