POPULATION LIMITATION AND THE WOLVES OF ISLE ROYALE

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Population regulation for gray wolves in Isle Royale National Park, Michigan, was examined in 1987–1995 when wolves were in chronic decline following a crash of the population in 1981–1982. Canine parvovirus (CPV-2) was probably influential during the crash, but it disappeared by the late 1980s. High mortality abruptly ceased after 1988, but low recruitment in the absence of disease and obvious shortage of food prevented recovery of the wolf population. In 1983–1995, with a comparable number of moose ≥10 years old as potential prey, wolves were only half as numerous as in 1959–1980. A simulation of annual fluctuations in effective population size (N_e) for wolves on Isle Royale suggests that their genetic heterozygosity has declined ca. 13% with each generation and ca. 80% in the 50-year history of this population. Inbreeding depression and stochastic demographic variation both remain possible explanations for recent low recruitment.

Key words: *Canis lupus,* wolf, *Alces alces,* moose, canine parvovirus, inbreeding, predator-prey, Isle Royale National Park

Based on their analysis of fluctuations of gray wolves (*Canis lupus*) in Isle Royale National Park, Peterson and Page (1988:97) concluded that "the only natural limits to wolf density are those ultimately imposed by food supply." Specifically, they attributed a major crash of wolves on Isle Royale in 1981–1982 to shortage of food. They predicted that wolves would remain at a relatively low level through the 1980s and would increase as the number of old moose in the population rose in the 1990s. Instead, wolves declined further; hypotheses as to the cause included lack of food, disease, or genetic deterioration (Peterson and Krumenaker, 1989), or demographic stochasticity. In this study, recent demography for this population is analyzed, and those four hypotheses are evaluated.

Wolves on Isle Royale are isolated geographically from mainland wolves, and there is no evidence of successful immigration of breeding individuals after the population was founded in the late 1940s (Wayne et al., 1991). Wolves on Isle Royale have been protected from human-caused mortality and have existed in dynamic equilibrium with moose (*Alces alces*), its most important prey (Peterson et al., 1984). Size of the wolf population has been monitored annually since 1959 (Jordan et al., 1967; Mech, 1966; Peterson, 1977; Peterson and Page, 1988; Wolfe and Allen, 1973).

MATERIALS AND METHODS

Isle Royale is a roadless island (544 km^2) in Lake Superior ca. 25 km from the Ontario mainland. It is almost entirely forested by a mix of boreal and mixed-hardwood tree species (Peterson, 1977). Wolves on Isle Royale subsist primarily on moose, with beaver (*Castor canadensis*) as a secondary prey that may be seasonally important (Mech, 1966; Peterson, 1977; Peterson and Page, 1988; Thurber and Peterson, 1993). Almost the entire island is managed as a designated Wilderness Area by the United States National Park Service, and human activity is

limited largely to shoreline camp grounds and 280 km of foot trails. The park is staffed and open to recreational visitors only mid-April through October. Mammalian pets are prohibited on the island.

During 1987–1995 wolves were studied annually during 2 periods, mid-January through February and mid-May through August. During winter, wolves were snow-tracked, counted, and observed from a Piper 18-150 Supercub with skis. A complete count of all wolves was made several times each winter. Wolves, especially territorial packs, were back-tracked to determine their extent of travels. Those packs usually occupied exclusive, defended space, and scent-marked regularly during their travels (Peters and Mech, 1975). Continuity of packs from 1 year to the next was indicated by the presence of at least one identifiable wolf in the pack in both years. In summer, wolves were radio-tracked from aircraft and on foot, and sites with repeated use were monitored for evidence of reproduction. Pups were counted opportunistically during group howls. Pups were enumerated more accurately in winter by aerial observations of behavior and appearance (Peterson and Page, 1988), using a 15× gyro-stabilized monocular (Fraser-Volpe, Inc., Warrington, PA). Annual mortality was estimated from total size of the population and the number of recruits each winter. Emigration and immigration were assumed to be zero because none was detected in February 1991 or January–February 1994, the only periods when an ice bridge to the mainland was present.

During 1988–1994, wolves were captured using custom-built Newhouse-type 14 leghold traps (Kuehn et al., 1986) or a similar commercially available trap (Rancher's Supply, Alpine, TX). Traps were checked daily, and captured wolves were anesthetized for handling as approved by the Institutional Animal Use and Care Committee at Michigan Technological University (Thurber and Peterson, 1993). Wolves were weighed, and blood samples were collected for disease screening and genetic studies (Lehman et al., 1991; Wayne et al. 1991). Wolves were released wearing motion-sensitive radiocollars (Telonics, Inc., Mesa, AZ).

During each winter, alpha (socially dominant and breeding) wolves were individually recognizable and could often be distinguished in successive years on the basis of physical appearance. Female wolves in breeding condition were identified by vaginal bleeding that was evident in urine on snow, and those in breeding condition were considered potential reproducers if they were courted actively by a male.

Size and age structure of the population of moose on Isle Royale were reconstructed through cohort analysis of recoveries of dead moose during 1958–1993 (R. O. Peterson, in litt.). Wolf predation was selective for moose ≥10 years old (43% of wolf-killed moose—Peterson, 1977), so their abundance was used to index the supply of food for wolves.

The Mann-Whitney U test was used to compare litter sizes in different periods (Sokal and Rohlf, 1969). Simple linear regression (Sokal and Rohlf, 1969) was used to determine if size of the wolf population was related to abundance of old moose and to examine relationships between changes in density of wolves and underlying mortality and reproduction.

Parasites and diseases were evaluated by direct external examination and subsequent laboratory studies of blood and feces. Antibodies to canine parvovirus (CPV-2) were detected with the hemagglutination-inhibition (HAI) test (Carmichael et al., 1980). Serial dilutions of serum were tested, and the HAI titer was determined to be the reciprocal of the highest dilution at which HAI activity was evident. The serum-neutralization test was used to detect antibodies to canine distemper (Bailey et al., 1995). Serologic tests for antibodies to infectious canine hepatitis and rabies were performed at the National Veterinary Services Laboratory (United States Department of Agriculture, Ames, IA)(Zarnke and Ballard, 1987). Tests for serum antibodies to *Borrelia burgdorferi* were conducted at the University of Wisconsin (Madison, WI) (Kazmierczak et al., 1988). Blastomycosis serology was performed at the Centers for Disease Control (United States Public Health Service, Department of Health and Human Services, Atlanta, GA) (Thiel et al., 1987). The rapid-slide-agglutination test detected antibodies to *Brucella canis,* and the microscopic-agglutination-lysis test was used for *Leptospira interrogans* serovars *pomona, icterohaemorrhagiae, hardjo, grippotyphosa,* and *canicola* at the Wisconsin Animal Health Laboratory (Wisconsin Department of Agriculture, Madison, WI). Serology for canine heartworm (dirofilariasis) was performed at Colorado State University (Fort Collins, CO)

(Grieve et al., 1981). Knott's test detected microfilaria in blood, and fecal flotation or sedimentation and direct-smear examination were used to survey for internal parasites (Ash and Orihel, 1987). Feces were screened for CPV-2 using a test kit for canine-parvovirus antigen (IDEXX Laboratories, Inc., Westbrook, ME). Necropsies, including gross and microscopic examination, were performed on carcasses of dead wolves that were recovered intact, and supplementary laboratory tests in bacteriology, virology, and parasitology were used to determine causes of death and document incidental abnormalities.

Genetic deterioration (loss of neutral genetic diversity) in the isolated population of wolves on Isle Royale was modeled using estimates of effective population size (N_e) and generation time. Neutral genetic heterozygosity (H) is lost at the rate of $(2N_e)^{-1}$ per generation, and N_e is reduced by increased variance in fecundity among individuals (Wright, 1938). Variance in fecundity arises from at least three factors: variance in adult life span, variation in individual fitness, and random annual variation (Nunney, 1996). These parameters were estimated for wolves on Isle Royale, incorporating what is known about the population from this and previous studies (Appendix I). The harmonic mean for annual estimates of N_e (Wright, 1938) was used to evaluate rate of genetic deterioration.

Where pups were present but the number of pups was uncertain, we estimated the number present based on annual change in pack size and prevalent annual mortality rates of 15–30% (Peterson and Page, 1988; this study). Likewise, longevity of identifiable breeding wolves was used to estimate turnover probabilities for wolves in breeding positions in packs when their longevity was uncertain. To assess the sensitivity of our calculations to uncertainties in estimating numbers of pups and identity of their parents, we also estimated N_e by assuming values in the case of uncertainty that would underestimate variance in fecundity the most (resulting in the greatest overestimate of N_e).

Results

Prior to this study the wolves of Isle Royale fluctuated between 14–50 individuals, and average size of the population was 27 during 1959–1986 (Jordan et al., 1967; Mech, 1966; Peterson, 1977; Peterson and Page, 1988; Wolfe and Allen, 1973). The population was stable in 1959–1973; then it increased steadily and peaked in 1980 (Fig. 1a). A crash of the population in 1981–1982 left only 14 wolves alive, but the population recovered to ≥20 wolves during the mid-1980s (Peterson and Page, 1988). The crash of 1981–1982 provided a

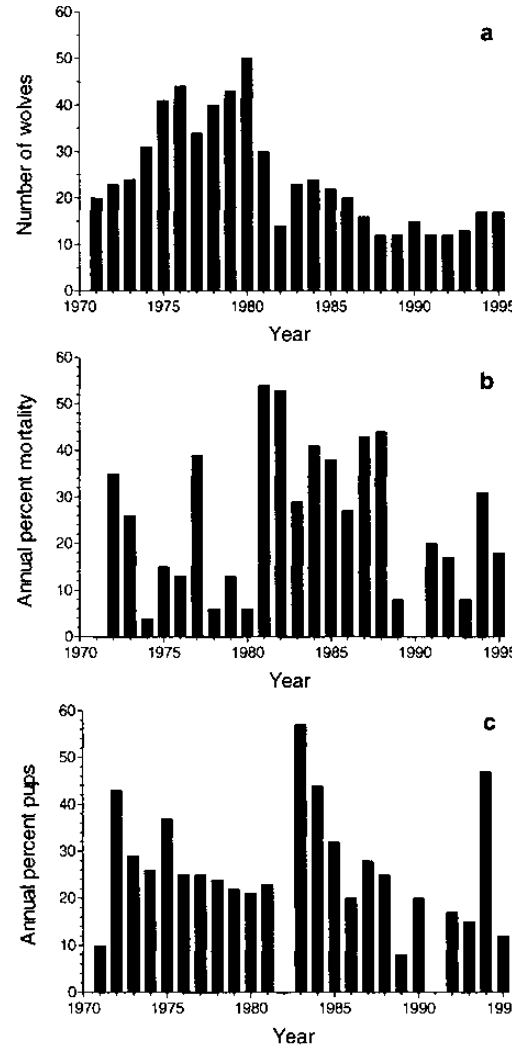

FIG. 1.—For the wolf population on Isle Royale, 1971–1995, a)number of wolves, b)annual mortality, and c)proportion of pups in mid-winter. Sources were Mech (1966), Jordan et al. (1967), Wolfe and Allen (1973), Peterson (1977), Peterson and Page (1988), and this study.

TABLE 1.—*Organization and demography of wolves on Isle Royale, 1987–1995.*

Year	Territorial packs[a]					Number of other groups		Total wolves	Total pups	Number of litters	Percentage annual mortality[b]
	EII	WII	MI	EIII	MII	Two wolves	One wolf				
1987	4	8	3[c]				1	6	4–5	2	40–45 (8–9/20)
1988	2	6				1	2	12	3	1	44 (7/16)
1989	2[c]	3		3		1	2	12	1	1	8 (1/12)
1990		2		5	2	1	4	15	3	1	0 (0/12)
1991		2		4	2	1	2	12	0	0	20 (3/15)
1992		2		5	2		3	12	2	1	17 (2/12)
1993		2		7	2		1	13	2	1	8 (1/12)
1994		2		9	6			17	8	2	31 (4/13)
1995		2		6	3	1	4	17	2	1	18 (3/17)

[a] Acronyms for packs as follows: EII = east II, WII = West II, MI = Middle I ("Harvey Lake" in Peterson and Page [1988]), EIII = East III, MII = Middle II.

[b] In parentheses is the proportion of the population that died in the previous year. Population size in 1986 was 20 (Peterson and Page, 1988).

[c] Pack occupied same territory as previous year, but identification was uncertain.

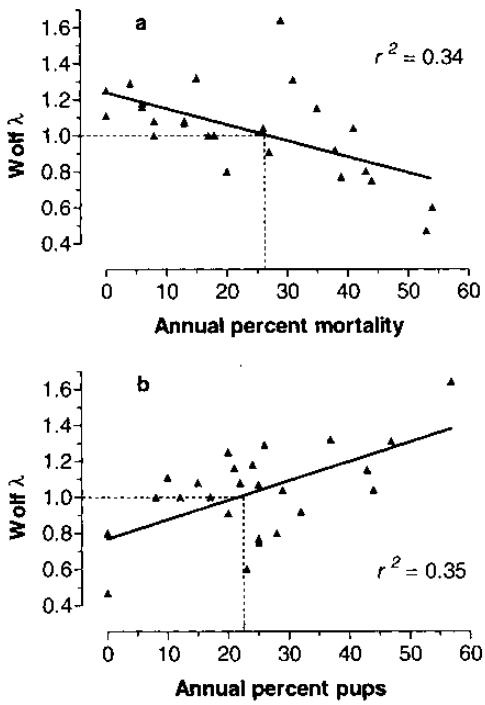

FIG. 2.—Influence of a) percentage of mortality and b) percentage of pups (b) on annual finite rate-of-increase (λ; Caughley, 1977) for wolves on Isle Royale, 1971–1995. Wolf λ = −0.009 (Percentage of annual mortality) + 1.24 (F = 11.5, P = 0.002) and wolf λ = 0.011 (percent pups) + 0.77 (F = 13.0, P = 0.002).

natural breakpoint for analysis of demographic parameters over time.

Wolf numbers and organization.—The number of wolves on Isle Royale declined from 20 in 1986 (Peterson and Page, 1988) to 16 in 1987 and 12 in 1988—the lowest level attained since annual counts were initiated in 1959. Thereafter, the population stabilized and then increased to 17 wolves in 1994 (Fig. 1a and Table 1).

Both reproduction and mortality influenced the rate-of-increase for the wolf population (Fig. 2). Stability was achieved when both the percentage of pups and annual mortality were ca. 25%. Divergence (± 15% or more) from that equilibrium level for either demographic parameter led to substantial population change (ca. one-half of the years for mortality and one-third of the years for percentage of pups). Mortality influenced dynamics of the population more than reproductive success because it was more variable (Figs. 1b and 1c). Mean annual mortality in 1971–1995 was 23.5% ± 3.3 (*SE*) compared to 24.4% ± 2.8 for reproductive success.

In each winter, the population of wolves on Isle Royale comprised two or three territorial packs that exhibited scent-marking by alpha wolves (Fig. 3). Those packs typ-

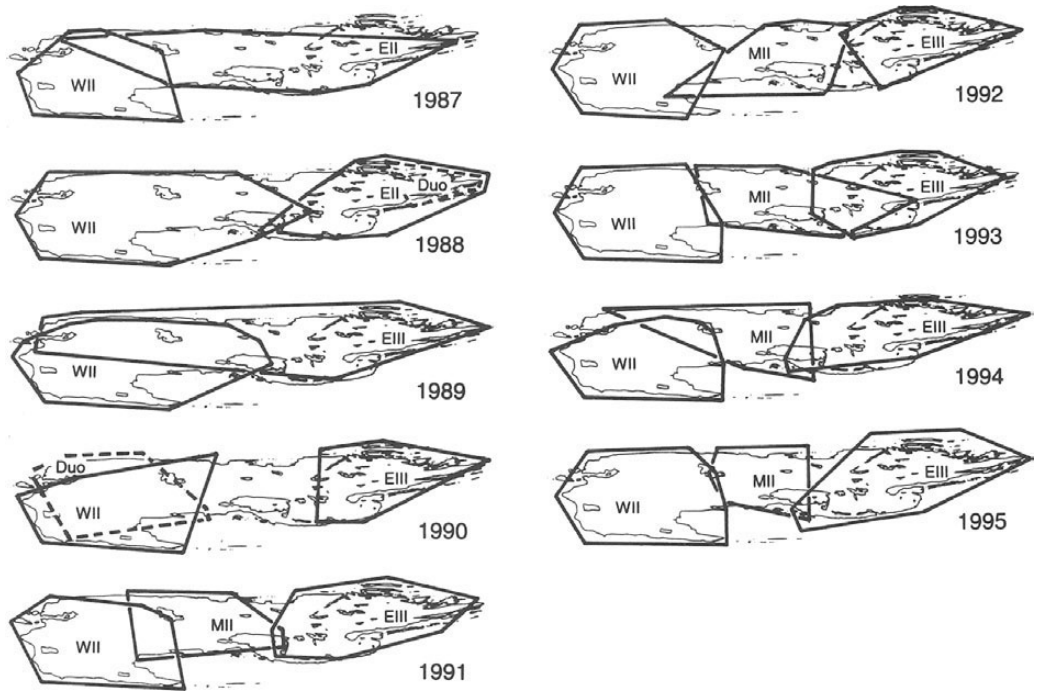

FIG. 3.—Spatial organization of territorial packs of wolves on Isle Royale, 1987–1995. WII = West II pack, MII = Middle II pack, EII = East II pack, and EIII = East III pack (acronyms follow Peterson and Page, 1988).

ically occupied territories for several years, and areas used by new packs (East III [EIII] pack in 1989, Middle II [MII] pack in 1990) were usually similar to territories of previous packs that disappeared. In such cases, it was possible that a single non-collared wolf remained as a founder of a subsequent pack.

The West II (WII) pack formed no later than 1982 and was the only pack to persist throughout 1987–1995. The EII pack on the east end of Isle Royale declined to just an alpha pair by 1988, which was its last year as a recognized territorial pack. By 1990 a new pack (EIII) had fully replaced it and assumed the same territory (Fig. 3). The Middle I (MI) pack ("Harvey Lake pack" in Peterson and Page, 1988), persecuted for many years by both neighboring packs, continued as a remnant group of three wolves in 1987 after the EII pack killed one wolf in the MI pack in 1986. In 1987, the EII pack again attacked the MI pack and killed an adult male. An adult female and a smaller wolf, probably a pup, survived, but they did not scent mark in subsequent observations; their former territory was utilized by the EII and WII packs in 1988 and 1989. In 1991, a male and female of unknown origins, designated as Middle II (MII) pack, claimed a territory in the middle of the island. In 1994, both of those wolves died, and their territory was claimed by a yearling male offspring that paired with a yearling female that probably originated in the adjacent EIII pack.

Packs of wolves often chased and occasionally killed lone wolves in their territory. Outcomes of encounters between packs and single wolves were variable and partially dependent on individual circumstances and previous associations. The WII pack chased a lone wolf and killed it 2 days later after a second chase. Another lone wolf was chased at least twice by this pack but not attacked. After reproducing successfully,

TABLE 2.—*Chronology of identifiable breeding wolves and reproductive success on Isle Royale, 1968–1995*[a].

	West pack			Middle pack			East pack			Southwest pack		
Winter	Male	Female	Pups	Male	Female	Pups	Male	Female	Pups	Male	Female	Pups
1968	M1	F1	?									
1969	M1	F1	?									
1970	M1	F1	?									
1971	M2	F1	2									
1972	M2	F1	4				M3	F2	6			
1973	M4	F3	4	M5	F4	0	M3	F2	3			
1974	M4	F3	4	M5	F4	0	M6	F2	4			
1975	M8	F3	5	M5	F4	5	M7	F2	4–5			
1976	M8	F3	4–5	?	?		M7	F2	4–5			
1977	?	?	1–2	?	?		M7	F2	≥4	?	?	0
1978	?	?	?	?	?	?	M7	F2	≥3	?	?	?
1979				?	?	?	M7	F7	≥3	?	?	?
1980				M10	F5		M9	F2	3–4	?	?	≥3
1981				M10	F5		M9	F2	0	?	?	?
1982	M11	F8	0–2	M10	F6	2	M9	?	0	?	?	0
1983	M11	F8	4	?	F6	1	M9	?	5			
1984	M11	F8	5–6	?	F6	0	M9	?	3			
1985	M11	F8	4	?	?	4	?	?	3			
1986	M11	F8	3	?	?	2	?	?	1			
1987	M11	F9	4	?	?	0	?	?	0			
1988	M12	F9	3				?	?	0			
1989	M12	F9	1			0–1						
1990	M12	F9	0	M13	F10		M14	F11	3			
1991	M12	F9	0	M13	F10		M14	F12	0			
1992	M12	F9	0	M13	F10	0	M14	F12	2			
1993	M12	F9	0	M13	F10	0	M14	F12	2			
1994	M15	F9	0	M13	F10	4	M14	F12	4			
1995	M15	F9	0	M16	F14	0	M14	F13	2			

[a] From Peterson (1977), Peterson and Page (1988), and this study. "?" reflects uncertainty in identity of breeding wolves or number of pups.

one alpha female became intolerant of another female and threatened her during a chase and a non-fatal attack; the two wolves previously had been close associates.

In August 1993, the 12 wolves that remained on Isle Royale included three females, seven males, and two wolves of unknown sex, which was the lowest point in the known number of wolves of either sex on the island. The only females known to exist were the breeding females in each pack.

Social organization of wolves on Isle Royale in 1988–1993 was typified by long tenure of individual breeding wolves in territorial packs (Table 2). During 1993–1995, however, there was turnover in four of the six alpha positions; three of those vacancies were filled by wolves born in 1993.

Reproduction.—Pups were counted accurately during winter when they were 8 months old. The annual number of recruits steadily declined in the mid-1980s and then remained low during 1988–1995, except in 1994 when eight pups survived from the 1993 cohort (Table 1).

Average size of litters declined only slightly after the 1981–1982 crash of the population, and was highly variable (median 3.5 in 1971–1980 versus 2.3 in 1984–1995—$U = 99.5$, $P = 0.05$). Before the crash, alpha females consistently raised young each year, but after 1985, actual reproduction increasingly fell short of poten-

tial reproduction because established packs often failed to produce any pups that survived at least 8 months.

Lifetime reproduction for identifiable alpha wolves was highly variable, and a few individuals bore a disproportionate number of young. During 1971–1995, 128–137 pups were identified in winter counts. Over 72% of those pups were produced by only five alpha wolves (Table 2). Two females produced ≥ 62% of the 68–73 pups identified in 1971–1980 before the crash of the population, and one female bore ≥ 26% of the 58–61 pups counted in 1982–1994 after the crash.

Mortality.—Mean annual mortality was high in 1981–1988 (41.3% ± 3.6 *SE*), whereas in all other years during 1971–1995 mean annual mortality was 15.2% ± 0.13 (Fig. 1b). There was little direct information on causes of mortality for most of the wolves that died in 1971–1988. About 17 wolves disappeared between successive studies in winter 1976 and 1977, but an exceptional ice connection to the mainland in 1977 could have allowed emigration (Peterson, 1979). Between counts in 1980 and 1982 when no ice bridge to the mainland existed, 43 wolves died that had been counted in previous winters (Peterson and Page, 1988). An additional nine pups alive in summer 1981 also perished. Starvation of old wolves and direct killing by other wolves caused death of five dead wolves recovered in 1980–1982, but >90% of the mortality remained unexplained (Peterson and Page 1988).

Circumstantial evidence indicated that CPV-2 contributed to mortality of wolves in 1980–1981. An epizootic among domestic dogs (*C. l. familiaris*) occurred in 1981 in Houghton, Michigan, a common point of departure for visitors to Isle Royale (J. Pepper, pers. comm.).

Aside from opportunistic recovery of carcasses of three wolves killed by other wolves, the only other documented mortality of wolves during 1981–1988 was a pup that died in 1986 in the WII pack. Its skeleton was recovered a year after death in dense vegetation near a summer rendezvous site. Based on skeletal size, its body weight was estimated at 20 kg, which suggested death between August and October (Van Ballenberghe and Mech, 1975).

Causes of mortality were determined after 1988 using radiotelemetry. Of the 30 wolves that were alive during any part of 1988–1995, 15 died by March 1995. Mortality for 10 wolves that were radiocollared was attributed to killing by wolves (two), malnutrition at an advanced age (two), accidents (one fell through ice in Lake Superior and one died of probable trauma), and unknown causes after radiofailure (five, including 1 female 11–15 years old). Additionally, one uncollared wolf died of malnutrition at an advanced age and another uncollared wolf was killed by other wolves.

Parasites and disease.—Seven wolves handled in 1988–1989 were screened for endo- and ecto-parasites and diseases of viral, bacterial, and fungal origin. Little evidence of exposure to infectious diseases was found. There was no serologic evidence of exposure to rabies (0/4) or canine distemper (0/7 and 0/4 additional wolves handled in 1990–1995), *Brucella canis* (0/3), *Leptospira* (0/3), or *Blastomyces dermatitidis* (0/4). Serological results for canine heartworm (*Dirofilaria immitis*) were equivocal in two of four wolves, but no microfilaria were detected (0/7) so they were considered negative. All four wolves tested for infectious canine hepatitis had antibodies, similar to high prevalences reported for wild wolves in North America (Zarnke and Ballard, 1987). Three of four wolves had low positive antibody titers to *Borrelia burgdorferi* (the human Lyme disease agent), but there were no clinical indications of Lyme disease.

Serology indicated that four live-captured wolves had been exposed to CPV-2, but such evidence declined after 1988 (Fig. 4). Two of the first four wolves handled in 1988 had positive antibody titers to CPV-2. In 1989, two of four wolves were margin-

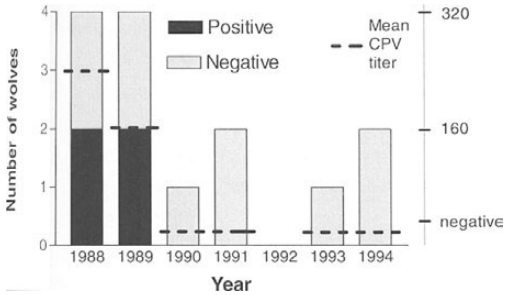

FIG. 4.—Blood levels of antibody titers to canine parvovirus in wolves live-captured on Isle Royale, 1988–1994; prevalence of positive titers and mean titer levels both decreased through time.

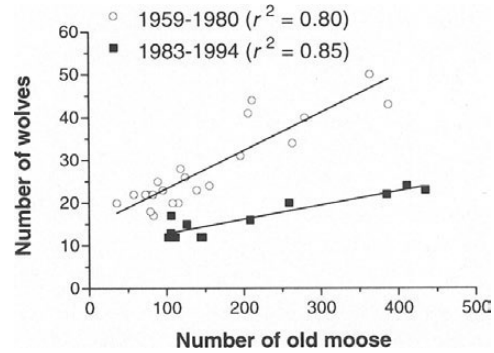

FIG. 5.—Relationship between abundance of moose ≥ 10 years old and size of the wolf population on Isle Royale, 1959–1980 (Y = 0.089X + 14.5) and 1983–1994 (Y = 0.033X + 9.49).

ally positive for exposure to CPV-2, and one recaptured wolf was negative in both 1988 and 1989. In 1990–1994, all six wolves handled were negative for CPV-2. Serological testing of 31 red foxes (*Vulpes vulpes*) on Isle Royale in 1988 and 1989 revealed negative titers for CPV-2 in this species. Canine parvovirus was not detected in the feces of nine wolves handled in 1988–1991.

Parasites in wolves on Isle Royale were similar to those reported in other North American wolves (Rausch and Williamson, 1959; N. J. Thomas, in litt.). Fresh fecal samples from seven captured wolves and 18 other samples collected throughout Isle Royale in 1988–1994 were examined for parasites. Helminths were found in 13 of the 25 fecal samples. One fecal sample contained three different species of helminths, three other samples contained two, and nine samples contained one. Hookworm was most prevalent in feces (11 of the 25 samples). Taeniid eggs were found in four samples, and one sample contained *Filaroides, Trichuris, Alaria,* and *Eucoleus aerophilus.* Upon necropsy, complete examination of the gastrointestinal tracts of three emaciated wolves with heavily-worn teeth revealed *Echinococcus granulosus* and *Taenia krabbei* or *Taenia* sp. in all three animals, and *Uncinaria* sp. and *Alaria* sp. in one wolf. One *Ixodes* sp. tick was found on one carcass, and no ectoparasites were found on trapped wolves.

Long-term trends in food supply.—Abundance of old moose (≥10 years old) was correlated positively with numbers of wolves before (r^2 = 0.80) and after (r^2 = 0.85) the crash of the population in 1980–1982 (Fig. 5). Density of wolves after 1982 was lower and the response of wolf numbers to increased food supply was less pronounced than before 1980 (F = 79.03, $d.f.$ = 1, 32, $P < 0.01$). Using data from before the crash, the correlation between numbers of old moose and wolves was higher assuming no lag in wolf responses (r^2 = 0.80) than with lags of 1 year (r^2 = 0.69), 2 years (r^2 = 0.51), or 3 years (r^2 = 0.55).

Genetic deterioration.—A model estimating effective population size (N_e) and resulting rate of genetic deterioration (loss of neutral genetic diversity) for wolves on Isle Royale in 1971–1995 revealed a high expected genetic loss (Appendix I). With best estimates of demographic parameters used in the model, the harmonic mean of annual estimates of N_e was equal to 3.8 individuals (annual values ranged from 1.5 to 6.8). That corresponded to a 13% loss in neutral genetic diversity with each generation, or every 4.2 years.

Harmonic mean for annual estimates of N_e was calculated to be 5.0 when parameter

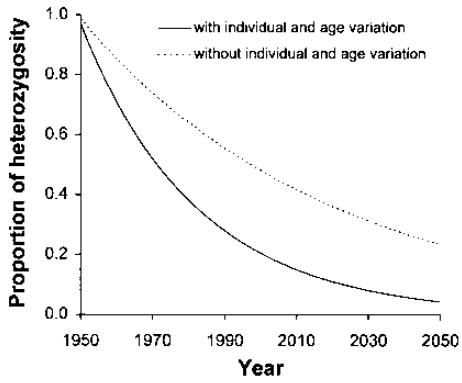

FIG. 6.—The predicted deterioration of neutral genetic heterozygosity (H) over time for the wolf population on Isle Royale with (solid curve; effective size was $N_e = 3.8$) and without (dotted curve; $N_e = 8.3$) the influence of variation in individual fitness (i.e., average annual fecundity) and variation in the length of tenure in the alpha position (Appendix I). Both curves were obtained by letting H deteriorate at the rate $(2N_e)^{-1}$ per generation, with a generation time of 4.2 years.

values were used which maximized N_e within the limits of uncertainty in estimating parameters (Table 2). The corresponding rate of genetic deterioration is 10% per generation. When age of initial breeding was 6 years instead of 3 years, the harmonic mean of annual N_e was 4.4. When variance in individual and annual reproduction was ignored, calculated harmonic mean of annual N_e rose to 8.3 (Fig. 6). All of these calculations may overestimate N_e because they fail to account for deviations from an even sex ratio (Caballero, 1994).

DISCUSSION

Territorial packs are the typical reproducing unit of wolves, and the enduring nature of packs of wolves on the east and west ends of Isle Royale probably arises from these areas being reliable sources of prey in winter (Peterson, 1977). During the 1980s, a pack struggled unsuccessfully to colonize the middle of the island (Peterson and Page, 1988), and a pack (MII) was finally established there in the 1990s. Meanwhile, the EIII pack on the east end maintained a moderate size while the WII pack declined substantially in numbers and slightly in territorial size. Decline of the WII pack and growth of the population of moose on the island enhanced prospects for a reproducing pack in the middle of Isle Royale.

Our study underscores the important role of food, in this case moose ≥ 10 years old, in limiting abundance of wolves on Isle Royale (Fig. 5), and it suggests that an epizootic of disease likely caused the precipitous population decline in 1980–1982, contrary to conclusions of Peterson and Page (1988). Before and after the crash of the population, there was an obvious link between supply of food and density of wolves, but after the crash, numbers of wolves were depressed as mortality exceeded recruitment. In 1982–1988, this resulted from unusually high mortality, but in 1985–1995, low reproductive success limited rate of increase of wolves on Isle Royale.

It is unlikely that any single factor was responsible for the overlapping but asynchronous changes in both mortality and reproduction after 1982. Four hypotheses are proposed that involve demographic stochasticity, shortage of food, disease, and genetic losses in the decline of wolves on Isle Royale.

Demographic stochasticity.—Random fluctuation in birth and death processes that operate independently among individuals (i.e., demographic stochasticity) may lead to decline and extinction of small populations (Caughley, 1994; Lande, 1993). The decline in population size for Isle Royale wolves in the 1980s and early 1990s resulted from 7 years of high mortality (1981–1988) and an overlapping decline in reproductive success in 1985–1992 (Fig. 1). These patterns are consistent with expectations of a stochastic demography hypothesis, which comprises individual variation in lifetime reproductive success.

While the importance of demographic stochasticity is difficult to evaluate without controlled experiments, this hypothesis

would seem less plausible if young wolves that attain breeding status in the late 1990s exhibit reproductive failure similar to those of the 1980s. Alternatively, if all other hypotheses for population decline could be rejected, demographic stochasticity would remain a reasonable cause for the decline.

Food shortage.—After wolves were reduced to low levels in 1982, shortage of food may have led to the chronic decline and low numbers of wolves that persisted into the mid-1990s. Messier (1987) found that packs of wolves preying on moose at low density (0.2/km^2) had low reproductive success (61%). When density of moose was higher (0.4/km^2), 93% of the packs of wolves reproduced. On Isle Royale in 1985–1995 when density of moose was >2.0/km^2 and two or three territorial breeding pairs were present, successful reproduction occurred in only 45% (n = 29) of the possible occasions. That represented unprecedented lack of reproduction for wild wolves, with no evident explanation in shortage of prey.

Nevertheless, the low numbers of wolves around 1990 corresponded to a relative low in supply of food. Before the crash, the average number of available old moose per wolf was 10.1 ± 0.7 *SE* (range, 4.8–19.1), and reproduction of wolves was maintained consistently in territorial packs. During 1985–1995 with decreasing reproductive success, the average number of old moose per wolf was 16.2 ± 0.9 and was never <13.3. Thus, available data on abundance of prey on Isle Royale do not suggest food shortage as the primary cause for the decline of wolves but do support the importance of food in dynamics of wolves. After 1982, numbers of wolves continued to track supply of food, but density of wolves was consistently lower than before 1980. Accompanying the lower size of the population was a less-pronounced response of wolves to increase in food. The food-shortage hypothesis alone does not explain adequately the overall reduction in number of wolves in the 1980s and early 1990s and the reduced response to supply of food in this period. The addition of one or more new limiting factors could explain these findings.

Disease.—The initiation of high mortality in wolves on Isle Royale in 1981 coincided with the probable initial arrival of CPV-2. After its first appearance in domestic dogs in 1978, CPV-2 spread worldwide by 1980 (Parrish et al., 1991). This virus was clearly present in other populations of wolves and coyotes in North America by 1979 and 1980 (Bailey et al., 1995; Brand et al., 1995). Following an outbreak of parvovirus in coyotes (*Canis latrans*) in Texas in 1980, the ratio of pups to adults dropped from an 11-year mean of 0.42 to 0.09, and in 1981 numbers of coyotes reached the minimum measured in 11 years (Thomas et al., 1984; Windberg, 1995). CPV-2 also proved to be virulent in wolves upon first exposure and was responsible for death of 11 of 12 pups and yearlings in a captive pack (Mech and Fritts, 1987). The prevalence of CPV-2 was correlated with population trends of wolves in Minnesota (Mech and Goyal, 1993, 1995) and mortality of pups in Montana (Johnson et al., 1994).

If wolf pups succumbed to CPV-2, mortality is expected in their first 4 months if they are unprotected by maternal antibodies (Johnson et al., 1994; Pollock and Charmichael, 1990). In 1981 when we speculate that CPV-2 was responsible for high mortality, at least three pups were detected in each of three packs in August, but none was subsequently present during aerial surveys 5 months later. The skeleton of a pup that died on Isle Royale in 1986 was of a size that suggested mortality at ca. 4 months of age.

During 1983–1988 following the crash of the population of wolves on Isle Royale, it is less clear that CPV-2 caused the prevalent high mortality among wolves > 8 months old, an age group not typically susceptible to disease from CPV-2 (Meunier et al., 1981; Parrish et al., 1982). Yet the only fully documented case of mortality to CPV-2

in a wild wolf involved an 8-month-old pup in Minnesota (Mech and Goyal, 1995). After 1988, annual mortality among wolves on Isle Royale declined to typical levels (<20%), and there was evidence that exposure to CPV-2 decreased because the frequency and level of titers of antibodies declined (Fig. 4). Because the probable period of CPV-2 exposure coincided with high mortality in 1981–1988, we suspect that this disease played a significant role both in the crash of the population in 1981–1982 and the chronic decline through 1988. The effects of CPV-2 on wolves on Isle Royale might have been exacerbated by shortage of food in the early 1980s (Peterson and Page, 1988), loss of genetic heterozygosity (Wayne et al., 1991), and parasitism by helminths. In any case, there was no apparent involvement of disease in the reproductive decline that persisted into the 1990s.

Genetic deterioration.—If wolves on Isle Royale experienced high rates of genetic deterioration, and genetic deterioration reduces fitness, viability of the population may become increasingly limited. Molecular estimates (Wayne et al., 1991) and calculations based on demographic data for wolves from Isle Royale (Appendix I) indicate that this population has experienced a high rate of inbreeding and genetic deterioration since the population's founding. In addition to small population size and social suppression of reproduction among non-alpha wolves (Mech, 1970), variance in annual and individual reproduction leads to a further loss of genetic deterioration, approximately halving N_e (Fig. 6).

For wolves on Isle Royale, N_e was typically <4.0. For the sake of comparison, a N_e of 4.0 is equivalent to mating between first cousins. Molecular estimates of genetic deterioration in wolves on Isle Royale suggest a comparable value for N_e for this population (Wayne et al., 1991).

It is impossible to predict precisely the impact of genetic deterioration on fitness in Isle Royale wolves because direct experimental evidence is unavailable. The only published study of inbreeding in a (captive) wolf population revealed that inbreeding significantly reduced fitness components (Laikre and Ryman, 1991), so it is plausible that fitness in wolves on Isle Royale has also been reduced by genetic deterioration. This inference is strengthened by considering that in a stressful environment (characteristic of wild populations) inbreeding depression is more severe (Keller et al., 1994; Miller, 1994; Schmitt and Ehrhardt, 1990), and wolves on Isle Royale may have experienced a higher rate of genetic deterioration than the population studied by Laikre and Ryman (1991). Unfortunately, the inference is limited by the observation that inbreeding depression varies greatly (Ralls et al., 1988), even within taxa (Frankham, 1995; Lacy et al., 1996). Thus, genetic deterioration is plausibly a critical factor limiting dynamics of this population, but the evidence required to reject this hypothesis will be very difficult to obtain (Lynch, 1988).

Factor interaction.—While factors limiting growth of the population of wolves on Isle Royale have varied over time, it is also likely that more than one factor operated simultaneously. The significance of food was pervasive in 1959–1995, but the arrival of new disease coincided with a period of high mortality that dominated the 1980s. Less certain was the degree of genetic decay arising from isolation; inbreeding depression should appear gradually and thus be difficult to detect against a background of other factors. Demographic stochasticity in a population with so few breeding individuals could be substantial. Full recovery of wolf numbers to a food-limited level (matching levels in 1959–1980 determined by moose >10 years old) would underscore disease as singularly important in the 1980s, allow rejection of the genetics hypothesis explaining the chronic decline, and suggest that reproductive failure in 1985–1995 can be attributed to demographic variation in combination with persistent constraints of availability of food. If numbers

of wolves or reproductive performance fail to fully recover in the next generation, genetic losses in the absence of outbreeding likely will be implicated as an important limiting factor to the population.

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APPENDIX I

Genetic-deterioration model.—The influence of variance in fecundity on annual N_e was quantified by (Nunney, 1996):

$$N_e = [2NT]\,[2 + 2I_A + (2I_S/A) + I_{Zm} + I_{Zf} + I_A I_{Zm} + I_A I_{Zf}]^{-1} A^{-1},$$

where N is the population size, T is the generation time, A is the average adult life span, and I is the standardized variance in fecundity (=[variance in fecundity among individuals]/[average fecundity per individual]^2) due to variation in adult life span (I_A), individual fitness (I_{Zm} for males or I_{Zf} for females), and random annual variation (I_S). This equation is a simplification of Equation 3 from Nunney (1996) that assumes age structure and maturation time are identical for both sexes.

Relying on this formula and population data from the Isle Royale wolf population (Peterson, 1977; Peterson and Page, 1988; this study), we estimated the seasonal N_e for Isle Royale wolves for each season from 1971 thru 1995. N was estimated from census data. Based on survivorship data, A was estimated to be 4 years (Vuc-

etich et al., 1997). T is given by $M - 1 + [A(k + 1)/(2k)]$ (see Nunney and Elam, 1994), where M is the age of sexual maturity (assumed to be 3 years), and k is the age of senescence (estimated to be 9 years from survivorship data). I_A is given by $(k - A)/Ak$ (Nunney and Elam, 1994).

The standardized variance due to random variation, I_{Si} was estimated from the reproductive success for each wolf in each year (assuming an even sex ratio). I_{Zi} was estimated for each season from the lifetime reproductive success of each wolf that was alive during that season. Estimates of I_{Zi} were initially based on the assumptions that wolves were 3 years of age when they first reproduced, and each breeding wolf died within a year of its last breeding attempt.