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Congenital bone deformities and the inbred wolves (*Canis lupus*) of Isle Royale

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ABSTRACT

The wolf (*Canis lupus*) population on Isle Royale, a remote island in Lake Superior, North America, is extremely inbred. Nevertheless, the consequences of genetic deterioration have not been detected for this intensively studied population, until now. We found that 58% ($n = 36$) of Isle Royale wolves exhibited some kind of congenital malformation in the lumbosacral region of the vertebral column and 33% exhibited a specific malformity, lumbosacral transitional vertebrae. By contrast, only 1% (1 of 99) of wolves sampled from two outbred, wolf populations exhibited this malformity. Moreover, in domestic dogs (*Canis lupus familiaris*) lumbosacral transitional vertebrae are associated with cauda equina syndrome, which can cause paresis, paralysis, locomotor difficulties in the rear legs and tail, and back pain. Whereas many studies illustrate how genetic deterioration affects population-level phenomena, such as survival and reproduction, these results are distinctive for demonstrating how genetic deterioration has compromised the morphology of individuals in a free-ranging population. The results are also significant because many policy makers and stakeholders and some conservation professionals use examples like Isle Royale wolves to downplay the consequences of genetic deterioration.

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1. Introduction

The detrimental effects of genetic deterioration are well documented for both captive and free-ranging populations and for a diverse set of taxa (e.g., Hedrick and Kalinowski, 2000; Keller and Waller, 2002). Nevertheless, many conservation professionals and policy makers downplay the threats posed by genetic deterioration for many populations of conservation concern (see Spielman et al., 2004; Jamieson, 2007). For example, genetic concerns are addressed by only a small portion of recovery plans for species protected by the US Endangered Species Act (Fallon, 2007). Some believe this lack of concern is justified, in part, by instances where populations appear viable and unaffected by genetic deterioration, despite being small and isolated.

One such instance is the wolf (*Canis lupus*) population on Isle Royale, an island in Lake Superior, North America. Since being founded by one female and one or a few males in the late 1940s, the population's average census size has been 24, and its long-term effective population size, N_e, has been ~3.8 (Peterson et al., 1998). Despite being highly inbred and despite intense, long-term observation, the effects of genetic deterioration have not, to date, been detected for this population. Here, we present the first evidence that this population has long been adversely affected by genetic deterioration.

The finding has relevance because this population is among several prominent examples that some conservation workers and policy makers use to support arguments that small populations can avoid genetic deterioration and be viable (e.g., Table 6.9 of Fuller et al., 2003; Fritts and Carbyn, 1995; Boitani, 2003; see also Wehausen, 1999 and references therein). These arguments have affected conservation policy. For example, the US Fish and Wildlife Service has used (and continues to use, as of 2008) such reasoning to defend, in part, their position that populations in the Northern Rockies and Great Lakes regions are large enough to be considered viable (USFWS, 2000). Similar reasoning has also affected conservation policies for the Scandinavian wolf population (Ekström, 1999; Regeringens Proposition, 2000).

1.1. The study population

Wolves first colonized Isle Royale (544 km^2) in the late 1940s by crossing an ice bridge connecting Isle Royale to the mainland, which are separated by ~24 km. Moose (*Alces alces*) represent about 90% of these wolves' diet. Humans do not harvest wolves or moose on Isle Royale. The wolf and moose populations of Isle Royale have been monitored intensively since 1958 (see Peterson et al., 1998; Vucetich and Peterson, 2004a; Vucetich et al., in press).

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The Isle Royale wolf population possesses a single mtDNA type, which is extremely rare in nearby mainland populations (Wayne et al., 1991). This suggests the population may have been founded by a single female. More recent evidence suggests the population was founded by one female and one, or perhaps two, males (J. Adams, unpublished data). Otherwise Isle Royale's wolf population is thought to be isolated from other populations.

Between 1958 and 2008, the population has been typically comprised of approximately 24 wolves (interquartile range = [17, 27], range = [12, 50]), living in three packs (interquartile range = [3, 3], range = [1, 5]), or breeding groups. In a typical year only three pairs of wolves breed. These and other factors (Nunney, 1996) cause the long-term, effective population size (N_e) of the wolf population to be approximately 3.8 (Peterson et al., 1998).

Given a N_e of 3.8, and that the generation time for Isle Royale wolves is ~4.3 years, this population is expected to have lost approximately 80% of its neutral genetic diversity during its time on Isle Royale, between 1950 and 2008 (see Peterson et al., 1998). Preliminary, unpublished analyses comparing microsatelite DNA from the Isle Royale population and from wolves on the nearby mainland suggest that Isle Royale wolves have lost approximately 60% percent of their genetic diversity during their first twelve generations on Isle Royale (J. Pollinger, unpublished data). Although the discrepancy between predicted and observed levels of genetic diversity has not yet been explained, a similar pattern has been observed and assessed for Scandinavian wolves (Liberg et al., 2005).

Despite high rates of inbreeding, key demographic properties of the Isle Royale population are comparable to outbred populations of wolves (see Fuller et al., 2003). The average annual mortality rate is 0.25 (0.17 SD) and the average annual recruitment rate (i.e., pups surviving to their first January) is 0.27 (0.14 SD). Pack size and per capita kill rates are also comparable to other populations that feed primarily on moose.

To date, the only evidence for inbreeding depression has been circumstantial. Between 1980 and 1982 the wolf population crashed from 50 to 14 wolves and remained in the low teens until the mid-1990s. The population crash was caused by the coincident occurrence of canine parvovirus (CPV) and food shortage (Peterson et al., 1998). However, the long period of low wolf abundance that followed is not easily explained by CPV or shortage of food. Moose had exceeded their long-term average abundance by 1984, and CPV had disappeared by 1989 (Peterson et al., 1998).

Moreover, wolf population dynamics differ dramatically before and after the outbreak of CPV. Prior to 1981, the average number of moose required to support a wolf was 36.3 ± 3.3 SE; afterward, that ratio increased to 53.3 ± 7.5 SE (Vucetich and Peterson, 2004b). Also, wolves represented an important influence on moose population dynamics prior to 1981; afterward, the influence of wolves was almost entirely replaced by the influence of winter climate (Wilmers et al., 2006). It is plausible, but far from certain, that these patterns are associated with genetic deterioration in the wolf population.

2. Materials and methods

Skeletal material was collected from 36 wolves that died between 1964 and 2007. These specimens included the entire vertebral column from atlas to sacrum. In some cases one or a few thoracic/lumbar vertebrae were missing due to fractures or scavengers that had taken parts of specimens before they were recovered. The coccygeal region was far from complete in most specimens (Table 1). Following methods described in Räikkönen et al. (2006), we examined the vertebrae of these wolves for congenital malformations. We focussed on vertebral malformations because such malformations are well studied in other populations of *Canis spp.* (see below). For context, the backbone of a normal wolf consists of seven cervical, C (1–7); 13 thoracic, T (1–13); seven lumbar, L (1–7); three fused sacral S (1–3); and around 20 coccygeal vertebral segments, Co (1–22) (Gilbert, 1997).

We also compared the rates of lumbosacral transitional malformations among samples collected from two small, inbred populations, contemporary Scandinavia (Liberg et al., 2005) and Isle Royale; and two large, outbred populations, contemporary Finland (Aspi et al., 2006) and historic Scandinavia. The contemporary population from Scandinavia was founded in the 1980s from three founders, one female and two males (Vilá et al., 2003). The materials from Finland and Scandinavia are further described in Räikkönen et al. (2006).

3. Results

3.1. Isle Royale wolves

Fifty-eight percent (21 of 36) of the individuals from Isle Royale had congenital malformations in the lumbosacral region of the vertebral column (Figs. 1, 2 and 4; see also Table 1). Among samples of known sex, males and females did not differ with respect to incidence of vertebral malformity ($p = 0.85$, $G = 0.033$, $df = 1$).

The incidence of vertebral malformity also increased substantially during the past five decades among specimens for whom the date of birth could be estimated ($p = 0.02$, $n = 33$, Fig. 5). For the six specimens whose age at death is known only to be within some range, the year of birth was estimated from the mid-point of that range. In these cases, the error in date of birth would be no more than 1 year. For 10 specimens, death is only known to have occurred during adulthood. We assumed these wolves died at the mean age of death for Isle Royale wolves (4 years). Because few wolves live beyond the age of 6, the estimated year of birth is unlikely to be in error by more than 2 years. Inspecting the scale of the *x*-axis in Fig. 5 suggests that these uncertainties are minor and unimportant. More generally, our inference (i.e., Fig. 5) is likely robust to these uncertainties, because slopes tend to be steeper than estimated when predictor variables are measured with error (Fuller, 1987).

Several wolves where characterized by more than the normal number of vertebrae. Specifically, 25% (9 of 36) of wolves had eight, rather than the normal seven lumbar vertebrae. One wolf (#3122) was very unusual for having 29 presacral vertebrae. The normal number is 27, and the extra vertebrae of this wolf were found in the thoracic and lumbar spine.

Thirty-three percent (12 of 36) of the wolves exhibited lumbosacral transitional vertebrae, LSTV. Transitional vertebrae exhibit features typical of two adjacent divisions of the vertebral column, can be found at any division (Morgan, 1968), and can also occur within a spinal segment (Breit and Künzel, 1998). Transitional vertebrae have also been reported in a variety of mammals (e.g. see Winckler, 1949; Searle, 1954; Wegner, 1959; Simoens et al., 1983; Junge et al., 2001; Newitt et al., 2008).

Lumbosacral transitional features exist in a variety of forms, including unilateral or bilateral patterns (Morgan, 1999; Flückiger et al., 2006). Most of the LSTV in Isle Royale wolves were bilateral asymmetries. Some unilateral asymmetrical patterns were also found. For example, wolf #378 had a sacral vertebral segment with a sacral process on the right side and a lumbar process on the left side (Fig. 1).

Some individuals exhibited a failure of the normal union of sacral segments. When the first sacral segment is transitional two fused sacral segments may remain. Several wolves also exhibited sacralization of the first coccygeal segment, where the first coccygeal segment is shifted to result in three sacral segments

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Table 1
Wolves collected at Isle Royale between 1964 and 2007. Age at time of death is based on counting annual cementum rings in the canines or information from having radio-collared some of the wolves. When date of death is known it is based on collecting the carcass within a year of the animal's death. In some cases, not all vertebrae were inspected because not all vertebrae were collected from the field.

Specimen	Sex	Age in years	Date of death	Vertebrae examined[a]	Nature of observed congenital vertebral malformity
377	Male	6	1964	Atlas-Co1	None
378	Female	5	1966	Atlas-sacrum	Lumbosacral transitional
532	?	5	1969	Atlas-T7, T9-Co2	None
436	Male	4	1969	Atlas-sacrum	None
503	?	1	1970	Atlas-Co1	None
1301	?	Adult	?	Atlas-Co3	None
1429	?	4	1978	Atlas-Co13	None
1521	Male	10	1980	Atlas-Co1	Sacrococcygeal transitional
1565	Male	10	1980	Atlas-Co18	Lumbosacral transitional
1667	?	6–7	1981	Atlas-T3, T5, T10-Co9	Sacrococcygeal transitional
1757	Male	4	1982	Atlas-Co11	None
1828	Female	6	1986	Atlas-L3, L5-Co4	Severe asymmetry at Co1
1872	Male	10	1987	Atlas-Co14	Lumbosacral transitional
2147	Female	5	1990	Atlas-Co8	None
2224	Male	11	1991	Atlas-Co12	Eight lumbar vertebrae
2509	Male	11	1993	Atlas-Co16	Thoracolumbar, lumbosacral transitional
2518	Male	10	1994	Atlas-Co9	Lumbosacral transitional
2542	Female	9	1994	Atlas-Co17	Lumbosacral transitional
2613	Female	1–2	1995	Atlas-sacrum	None
3114	?	4	1997	Atlas-T5,T7-Co3	None
3086	Female?	Adult	1997	Atlas-S2 (fracture at S3)	Thoracolumbar, 8 lumbar vertebrae
3020	?	Adult	1997	L3-sacrum	Lumbosacral transitional
3529	?	Adult	Late 1990s	Atlas-T11, L1-Co1	Cervical intrasegmental transitional, asymmetry at C6, L1 and lumbosacral transitional
3122	?	Adult	1998	Atlas-S2 (fracture at S3)	Cervical intrasegmental transitional, extra thoracic vertebra and eight lumbar vertebrae
3722	Male	Adult	2003	Atlas-Co19	Atlas foramina anomaly (developmental)
2475	Male	Adult	?	Atlas-T4, L1-Co12	Lumbosacral transitional
520	?	Adult	?	Atlas-S2 (fracture at S3)	Lumbosacral transitional
3387	?	Adult	2004	Atlas-Co18	Thoracolumbar, lumbosacral transitional
9876	?	Adult	2004	Atlas-Co13	Thoracolumbar, 8 lumbar vertebrae
3397	?	2–3	2005	Atlas-sacrum	Eight lumbar vertebrae
3398	?	2–3	2005	Atlas-T12, L1-Co3	None
4045	Female	8–10	2005	Atlas-sacrum	Eight lumbar vertebrae
4052	Male	8–10	2006	Atlas-sacrum	Lumbosacral transitional
4112	Male	Adult	2006	Atlas-sacrum	Eight lumbar vertebrae
4118	Male	Adult	2006	Atlas-sacrum	Minor asymmetry at C7, thoracolumbar transitional, 8 lumbar vertebrae
4282	Female	Adult	2007	Atlas-sacrum	Eight lumbar vertebrae, caudal asymmetry at sacrum

[a] Vertebral abbreviations: C: cervical, T: thoracic, L: lumbar, S: sacral, Co: coccygeal.

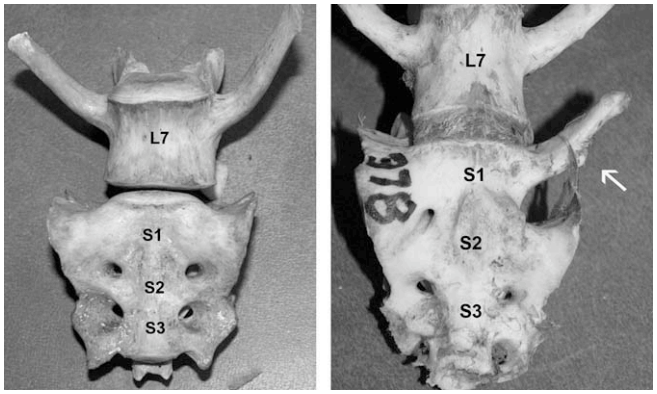

Fig. 1. The right photo shows the ventral view of wolf # 378 which exhibited a unilateral transitional segment at its sacrum, S1, see arrow (photo by J. Räikkonen). The left photo shows a normal wolf sacrum (photo by J. Räikkonen).

(see also Morgan et al., 2000). For example, the first sacral segment of wolf #3387 was separated with an abnormal disc space and exhibited lumbar features like a transverse process causing a very unequal illial attachment. Only two fused sacral segments remained. The associated asymmetries in the rest of sacrum were severe. The first coccygeal segment was sacralized and only connected dorsally to sacrum (Fig. 2). There were wolves that exhibited changes coupled to congenital malformations like secondary bony osteophytes and narrowed vertebral canals (see also Räikkönen et al., 2006).

The Isle Royale sample also contained a variety of transitional vertebrae at other regions in the vertebral column and other congenital malformations. Specifically, some wolves exhibited thoracolumbar transitional vertebrae at L1, two wolves (#3529 and #3122) had intrasegmental transitional vertebrae at the same vertebral location of the cervical spine (C7) (Fig. 3). Specimen #3529 also exhibited asymmetry at C6 and specimen #4118 exhibited minor asymmetry at C7. Specimen #1828 exhibited severe asymmetry at Co1.

Specimen #3722 exhibited incomplete ossification of the cranial border of atlas (C1) left lateral vertebral foramen, a minor developmental variant (see also Richards and Watson, 1991). This wolf also exhibited syndactyly, fusion of the soft tissue between the middle toes on both front feet. Syndactyly is a congenital malformity, a recessive trait, and more common among individuals from inbred lineages for a variety of mammals (e.g., Fossey, 1983; Gul and Oktenli, 2002; Drögemüller and Distl, 2006; Naruse et al., 2007). Italian wolves also exhibit partial fusion of the soft tissue between the middle toes on both front feet (F. Marucco, *pers. comm.*)

3.2. Interpopulation comparison

The incidence of lumbosacral transitional vertebrae (LSTV) is greater among Isle Royale wolves compared to those of Scandina-

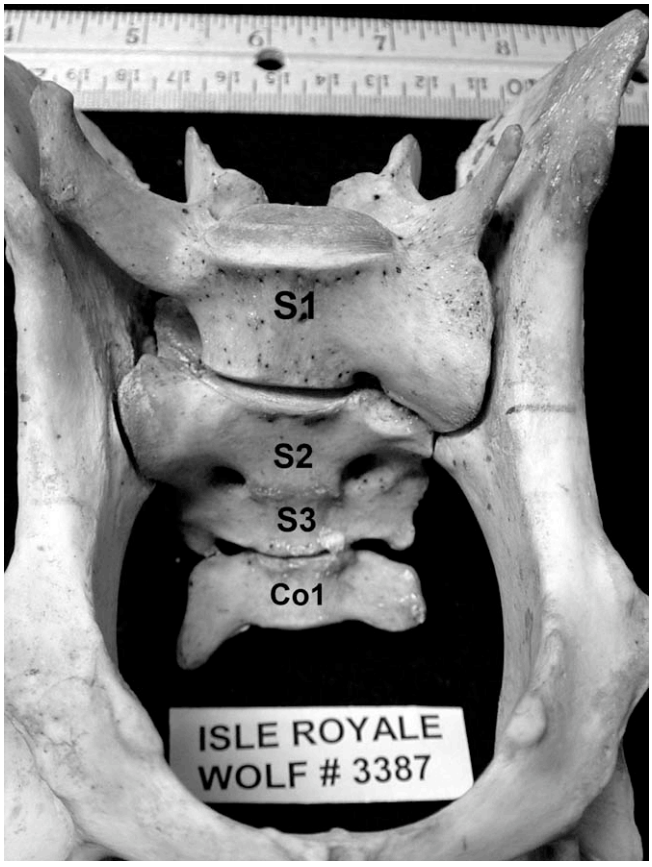

Fig. 2. Ventral view of wolf # 3387 which exhibited lumbosacral transitional vertebrae with severe changes at sacrum with an anomalous disc space (photo by J. Bump).

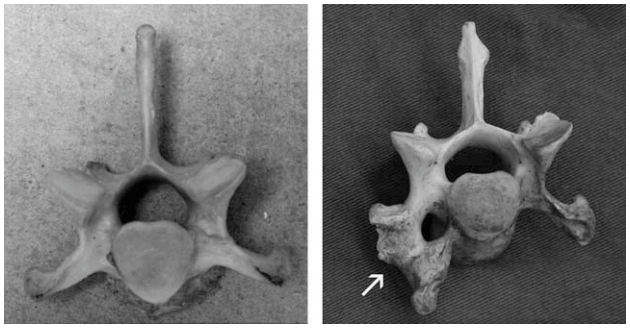

Fig. 3. The right photo shows the cranial view of wolf #3529 that exhibited a unilateral intrasegmental transitional vertebra at C7 (photo by J.A.Vucetich). One side of the vertebra resembles C6 with a transverse foramen (arrow). The left photo shows a normal C7 without transverse foramen (photo by R.O. Peterson).

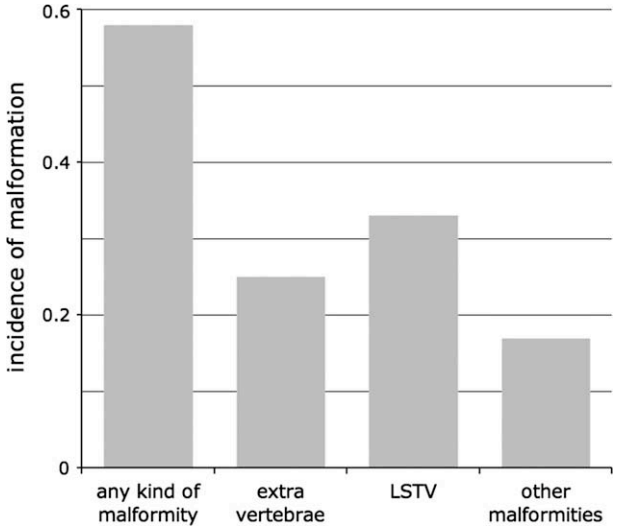

Fig. 4. The incidence of vertebral malformations in a sample of 36 Isle Royale wolves. The last three categories sum to more than the first category because some wolves had more than one kind of malformity. LSTV refers to lumbosacral transitional vertebrae. "Other malformities" include thoracolumbar transitional vertebrae, intrasegmental transitional vertebrae, asymmetrical vertebrae, and incomplete ossification of the cranial border of the first cervical vertebrae.

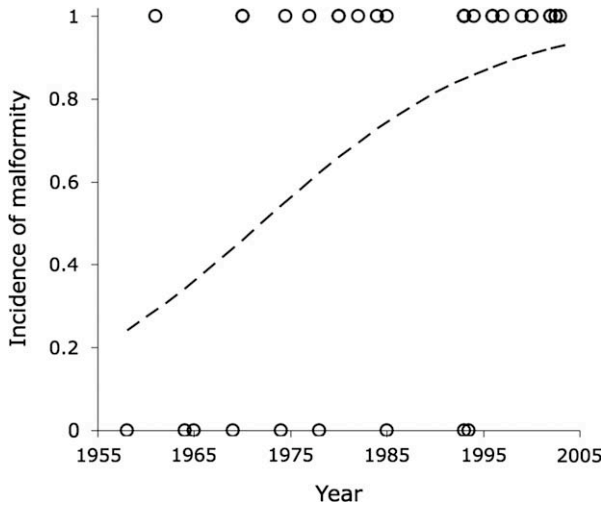

Fig. 5. The incidence of vertebral malformity in relation to the estimated year of birth for Isle Royale wolves. Symbols are observations and the curve is the best fit logistic regression ($p = 0.02$).

via and Finland (Fig. 6). An exact test comparing these proportions is not possible because the incidence for the historic Scandinavian population is zero. However, a conservative test can be conducted by presuming that one of the 25 samples from the historic Scandinavian population was malformed. The result of this test is that the incidence of LSTV is not equal among the populations ($p < 10^{-4}$, G-statistic = 24.9, $df = 3$).

More precisely, the incidence of LSTV is greater for Isle Royale wolves compared the other populations, when those samples are pooled ($p < 10^{-5}$, G-statistic = 20.8, $df = 1$), and compared to contemporary Scandinavia, the other inbred population ($p = 0.01$, G-statistic = 6.72, $df = 1$). Finally, the incidence of deformities is also greater for the inbred populations than for the outbred populations ($p < 10^{-4}$, G-statistic = 17.3, $df = 1$).

4. Discussion

4.1. Consequences of congenital vertebral malformations

The clinical significance of the cervical asymmetries we observed is unknown. However, the kinds of vertebral malformations common among Isle Royale wolves are likely detrimental (Morgan et al., 1993; Morgan, 1999; Steffen et al., 2004; Flückiger et al., 2006). For example, domestic dogs (*Canis lupus familiaris*) with lumbosacral transitional vertebrae tend to suffer from cauda equina syndrome, CES (Morgan et al., 1993). CES entails injury to the cauda equina, the most caudal region of the spinal cord and associated nerve roots (Berzon and Dueland, 1979). The consequences

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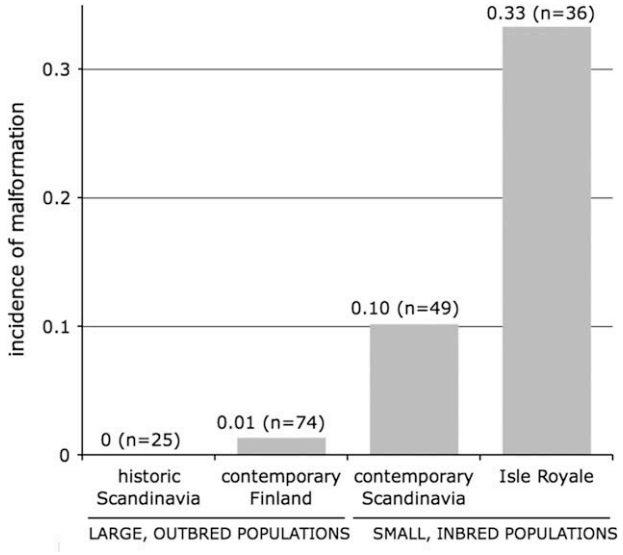

Fig. 6. The incidence of lumbosacral transitional malformations for different wolf samples. The Finnish and Scandinavian samples are described in Räikkönen et al. (2006).

of CES, which are independent of its etiology (Morgan et al., 2000), are variable and include paresis or paralysis; deficits in placing reactions when walking; deficits in voluntary movement of the tail; loss of muscle tone causing weakness of the hind limbs and flaccidity of the tail, low back pain and incontinence (Morgan et al., 1993, 2000).

Several Isle Royale individuals exhibited a sacral process with strong illial attachment on one side and a process of lumbar nature on the other side. This condition weakens the sacroiliac joint, may accelerate degeneration of the disc and result in disc protrusions (Morgan et al., 2000). Such disc degeneration with dorsal protrusion is also a common cause of CES. Dogs exhibiting disc protrusion also tend to suffer low back pain and lameness (Steffen et al., 2004). Asymmetrical sacroiliac attachment can also be associated with pelvic obliquity that can result in gait irregularities (Larsen, 1977) and detrimental development of the hip joints (Morgan et al., 2000).

4.2. Evidence for inbreeding depression

There is now good reason to think that Isle Royale wolves have been suffering from genetic deterioration. A substantial portion of Isle Royale wolves exhibit vertebral malformities that tend to be detrimental (Figs. 1–4). Lumbosacral transitional vertebrae are associated with inbreeding in dogs (Damur Djuric et al., 2006; Morgan et al., 1993, 1999; Morgan, 1999). Also, the incidence of these malformities tends to be associated with the extent of inbreeding among the examined populations (Fig. 6). For context, the Isle Royale wolf population has had a smaller N_e for a longer period of time than the wolf population of contemporary Scandinavia. Moreover, these Scandinavian wolves exhibit significant inbreeding depression in juvenile survival (Liberg et al., 2005). Comparable analyses have yet to be conducted for Isle Royale wolves, but are forthcoming.

Although Isle Royale wolves appear to exhibit compromised phenotypes in response to genetic deterioration, survival or reproduction remain comparable to those of other healthy wolf populations. Nevertheless, the observed vertebral malformities could affect wolf population dynamics. After a population bottleneck caused by introduced disease in 1980–1982, the number of moose required to support a wolf increased substantially after 1980, even while reproduction and survival did not exhibit detectable changes. The most plausible mechanism to explain these patterns is – using the parlance of predator–prey theory – a reduction in attack rates and (or) efficiency of conversion (Vucetich and Peterson, 2004b). It is possible, though far from certain, that these changes in predation ecology are associated with the skeletal malformities described here. This hypothesis is consistent with the observation that many Isle Royale wolves with vertebral malformities lived long lives (Table 1). That is, the vertebral malformities may have impacted the predatory performance of individual wolves, without our detecting an effect of the malformities on other vital rates.

For these inferences to be reliable, one would expect that either the incidence or severity of malformities to have increased after 1980. Such an increase has been observed (Fig. 5).

The observed malformations may have been caused by either inbreeding depression (i.e., decreased fitness for the offspring of related parents) or genetic load (i.e., a reduction in population fitness from the optimum possible) (Kirkpatrick and Jarne, 2000). Genetic load is increasingly found to be the cause of reduced fitness for small, isolated populations (e.g., Roelke et al., 1993; Fredrickson et al., 2007). In such cases, the introduction of non-related individuals is expected to result in "genetic rescue" (Tallmon et al., 2004), as was observed for Florida panthers and Mexican wolves. Forthcoming analysis may be able to distinguish between these potential causes for the Isle Royale population.

4.3. Mitigation

The effects of genetic deterioration might be mitigated by gene flow from non-related wolves raising the question of whether mitigation should be attempted. Because wolves often kill non-territorial wolves, such mitigation could be technically challenging. However, the appropriateness of such mitigation also depends on important, unresolved ethical issues. Peterson (1995) discusses some of these issues to better understand the appropriateness of reintroduction should Isle Royale wolves go extinct. That discussion focused on (i) the aesthetic and scientific values of perpetuating a predator–prey system largely unaffected by humans, and (ii) how to balance mandates associated with Isle Royale's designation as US Federal Wilderness, which values minimizing human intervention but also values actively mitigating past anthropogenic effects.

Peterson (1995) took for granted the appropriateness of not intervening while wolves persisted in order to maximize the prospect of improved scientific understanding of population viability in small populations. Assessing the appropriateness of this position now seems complicated by several new considerations. First, we now know genetic deterioration has at least compromised the anatomy of these wolves. Given current knowledge about population viability and the non-experimental circumstances characterizing Isle Royale, as much scientific insight might be gained by assessing the potential effects of genetic rescue as from continuing to observe the effects of population isolation.

Second, the potential benefits of gene flow to wolves may be unexpectedly detrimental to the viability of wolf–moose interactions on Isle Royale. That is, because ticks and other factors associated with climate warming have recently increasingly impacted moose (Vucetich and Peterson, 2008), a more vigorous wolf population could be importantly detrimental to moose. Third, genetic deterioration now seems to have been causing individual wolves to suffer – suffering that might be mitigated by intervention. Any decision about intervening on Isle Royale seems to involve balancing the value of basic scientific knowledge, health of ecological collectives (i.e., population viability and ecosystem health), the welfare of individual animals, and what is taken to be a virtue for wilderness areas, non-intervention (see Vucetich and Nelson,

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2007). In this way, the Isle Royale case is an example of a general and profound challenge for environmental ethics.

4.4. Conservation implications

Attitudes that downplay the threats posed by genetic deterioration seem associated with a set of beliefs. The first of these beliefs seem to be that the consequences of genetic deterioration are serious, but only for the very smallest populations. More specifically, concern for genetic viability is relatively unimportant for populations large enough to be considered viable when demographic concerns are taken into account. This overly simple conception has roots extending back to ideas presented in Lande (1988). Second, that effects of genetic deterioration would be detrimental should they arise, but that risk is relatively small. Being of small risk, its expected cost (invoking the parlance of risk analysis) is small compared to the social and financial costs (which would be large and certain) for recovering or maintaining populations to sizes that would ensure genetic viability.

The justifications offered to support these beliefs include: (i) arguments that demographic factors are a greater threat to viability than genetic factors (e.g., Lande, 1988), (ii) purging mitigates inbreeding depression (Templeton and Read, 1984; Shields, 1993), (iii) the perceived lack of examples where genetic deterioration has affected viability or caused extinction (e.g., Caro and Laurenson, 1994; Caughley, 1994; Maehr and Caddick, 1995), and (iv) taking the existence of small, isolated populations as evidence that such populations are viable, demographically and genetically (e.g., Fritts and Carbyn, 1995; Wehausen, 1999; Fuller et al., 2003).

Unfortunately, these justifications refer to outdated literature or fallacious reasoning. For example, purging is now understood to be unreliable for mitigating inbreeding depression (Boakes et al., 2007; see also Lynch and Walsh, 1998). Also, the rarity of populations identified with inbreeding depression is not evidence for inbreeding depression's rarity, but evidence for the difficulty of detecting inbreeding depression. The detection of inbreeding depression is favored by experimental control, replication, and precise measurement of fitness-related traits over a sufficiently long period of time. These conditions characterize the investigation of few free-ranging populations. Finally, counterexamples (i.e., the existence of small, isolated populations) are not evidence for the viability of small populations. Empirical experiments and mathematical analyses clearly indicate that, even when inbreeding depression is generally important, the magnitude and timing of inbreeding depression is highly variable among species and even among populations within the same species (e.g., Lynch, 1988; Lacy et al., 1996; Vucetich and Waite, 1999). Counterexamples also exist because, for any specified extinction risk (i.e., probability of extinction over some time frame), the realized time to extinction is highly variable (Vucetich and Waite, 1998). These sources of variation and the difficulty of detecting the effects of genetic deterioration are the explanation for counterexamples.

Despite these explanations, apparent counterexamples are still used to buoy arguments that downplay the effects of genetic deterioration. The findings of this paper remove one more example that some use to make poor arguments that have the effect of working against conservation.

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