Modeling Effects of Environmental Change on Wolf Population Dynamics, Trait Evolution, and Life History

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Supporting Online Material

www.sciencemag.org/cgi/content/full/334/6060/1272/DC1
Materials and Methods
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Modeling Effects of Environmental Change on Wolf Population Dynamics, Trait Evolution, and Life History

Tim Coulson,[1]* Daniel R. MacNulty,[2]† Daniel R. Stahler,[3] Bridgett vonHoldt,[4] Robert K. Wayne,[5] Douglas W. Smith[3]

Environmental change has been observed to generate simultaneous responses in population dynamics, life history, gene frequencies, and morphology in a number of species. But how common are such eco-evolutionary responses to environmental change likely to be? Are they inevitable, or do they require a specific type of change? Can we accurately predict eco-evolutionary responses? We address these questions using theory and data from the study of Yellowstone wolves. We show that environmental change is expected to generate eco-evolutionary change, that changes in the average environment will affect wolves to a greater extent than changes in how variable it is, and that accurate prediction of the consequences of environmental change will probably prove elusive.

Populations of the same species living in different environments often differ genetically or phenotypically. For example, the frequency of the genotype that determines whether a gray wolf (*Canis lupus*) has a black or gray coat varies with forest cover throughout North America (*1*). Similarly, wolves that predominantly feed on large prey are typically larger than those that specialize on smaller species (*2*). Numerous studies of a range of species also have reported that population dynamics and life history can vary across populations living in different environments (*3, 4*). In addition to these cross-population differences, environmental change within a population can generate rapid change in life history parameters such as generation length, in phenotypic trait and genotype distributions, and in population dynamics (*5, 6*). The eco-evolutionary consequences of environmental change are sometimes repeatable (*7*) but are frequently not (*8*). The wide range of population responses means that predicting likely dynamics has become one of the greatest challenges currently facing biology (*5*). This is particularly true for species, such as the gray wolf, that play important roles in structuring ecosystems, because their response to environmental change can have cascading effects across trophic levels (*9*). Given that environmental change can lead to potentially complex genetic, phenotypic, life history, and demographic responses, how can its likely consequences be explored? We show how integral projection models (IPMs) (*10*) provide a powerful framework to simultaneously investigate the ecological and evolutionary consequences of environmental change. We developed, applied, and analyzed one to explore how Yellowstone wolves may respond to environmental change.

Yellowstone National Park has experienced substantial environmental change in recent decades, with elk numbers declining, bison numbers increasing, and woody vegetation regenerating in some areas. These changes have been attributed variously to climate change, fluctuations in culling rates, and the reintroduction of wolves (*11*–*14*). Change is ongoing, with elk and bison numbers still trending in the same directions and further climate change being predicted (*15*). The

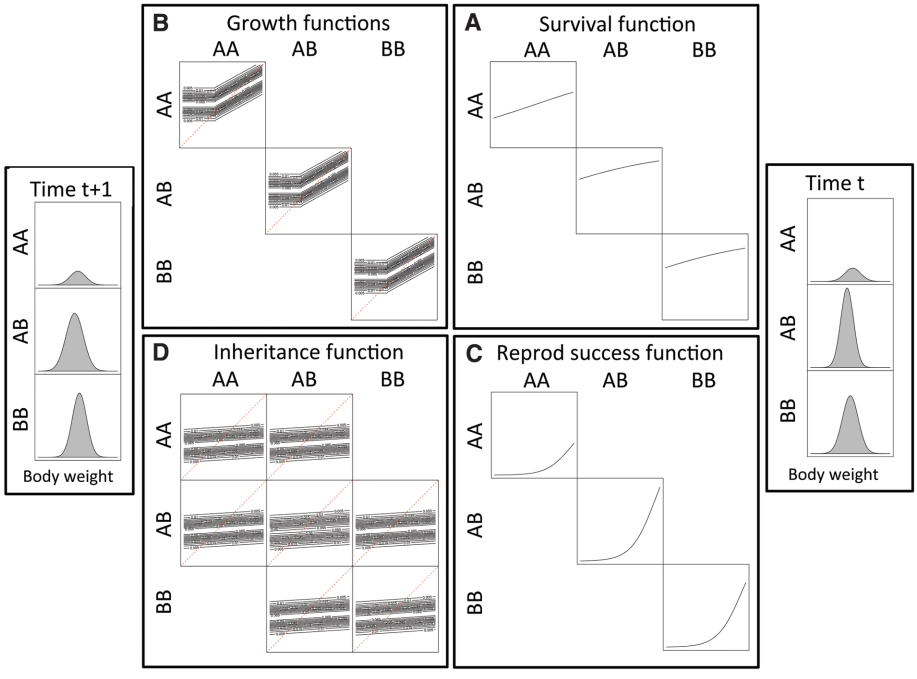

Fig. 1. (A to D) Graphical representation of the IPM that maps the bivariate distribution of genotype and body weight at time *t* to a new distribution at time *t* + *1*. Functions (B) and (D) are probability density functions showing the range of *y* values for each *x* value; both of these functions are identical across genotypes. Associations between body weight and both survival and reproductive success varied with genotype, whereas growth rates and inheritance did not. Equations for these functions and parameter values can be found in tables S1 and S2. The body weight and genotype distributions at times *t* and *t* + *1* are, respectively, on the right and left of the functions to provide a graphical representation of the mathematical structure of the IPM (SOM).

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Yellowstone wolf population has been extensively monitored since its introduction in 1995–1996 (*16*). We used survival and reproductive success data, body weights, and genotype at the K locus (*CBD103*, a β-defensin gene that has two alleles and determines coat color) collected from 280 radio-collared wolves living in the park between 1998 and 2009. Body weight and genotype at the K locus vary across U.S. wolf populations, and both traits influence fitness (*1, 2, 17*).

We constructed an IPM (Fig. 1) describing the temporal dynamics of the bivariate distribution of body weight and genotype [supporting online material (SOM)]. The model consists of functions (Fig. 1) describing how density dependence and environmental variation influence associations between body size and genotype at the K locus and (Fig. 1A) annual survival, (Fig. 1B) the probability of a surviving individual growing from weight z at time t to weight z' at time $t+1$ (the growth function), (Fig. 1C) annual reproductive success, and (Fig. 1D) the probability that a parent of body weight z at time t produces an offspring with body weight z' at time $t+1$ when the offspring recruits to the population (the inheritance function). Plasticity is captured by the growth and inheritance functions (Fig. 1), which capture how individuals of identical genotypes and body weight at time t can develop to different sizes at $t+1$ and produce recruiting offspring of different sizes. The functions constituting the IPM describe how "mass" of the genotype–body weight distribution is added, removed, and transformed by the fundamental biological processes of reproduction, inheritance, survival, and development (*10*). IPMs are a very general class of model, because all populations can be characterized as fluctuating distributions of phenotypic traits and genotypes, and because adding, removing, and transforming mass are the only ways to change the shape of a distribution or its size (the area under the distribution) (*18*). Because of their generality, it is possible to calculate many population biology parameters of both ecological and evolutionary interest from IPMs (*18–22*). We calculated population size, the mean and variance of body weight, the strength of viability and fertility selection on body weight, and genotype frequencies at each time step in a 500-year simulation and the means and variances in lifetime reproductive success and generation time for each cohort (*18*). Although we report results for populations at equilibrium, IPMs can be used to investigate transient dynamics.

IPMs can be parameterized for any system where repeated phenotypic measurements are taken from marked individuals, survival and reproductive rates are recorded, and the phenotype is measured across parents and offspring (*10*). Complete population coverage is not necessary, and biases in data can be statistically corrected (*20*). Stochastic IPMs require data collected from multiple censuses and are straightforward to parameterize (SOM). We used generalized linear mixed models (*23*) to statistically identify the survival, annual reproductive success, growth, and inheritance functions. The function describing how body weight and genotype influenced annual reproductive success (Fig. 1) is the product of two functions: one describing how body weight and genotype influenced the probability of reproducing (fertility function), and one describing the number of offspring produced conditional on successful reproduction (offspring number function). The growth function consists of two probability density functions, one each for wolves <41.7 kg and ≥41.7 kg. Survival and annual reproductive success functions differed with genotype; growth and body weight inheritance functions did not (Fig. 1). Population density was retained as a fixed effect in all functions (table S1 and fig. S1), and year was always retained as a random effect (SOM). Each function includes an intercept (for the average year) and an associated standard error describing how the intercept varies with time as the environment fluctuates. In Yellowstone wolves, such fluctuations are caused in part by temporal variation in snow depth, prey availability, and disease (*24–26*). We explored the consequences of environmental change by altering the means and standard deviations of the intercept distributions. Increasing the value of the mean intercept for the survival function, for example, mimics the effect of environmental change that improves average annual survival rates, whereas increasing the standard error of the distribution mimics environmental change that increases temporal variation in survival rates. We initially assumed no correlation in intercepts across functions. However, by imposing covariation between intercepts across functions, we explored how both positive and negative correlation in the values of function intercepts affects conclusions (SOM).

The model performed well in predicting key features of the wolf population (Table 1A) and provided insight into the dynamics of the coat color genotype. The IPM predicts that black heterozygotes have higher annual survival rates and annual reproductive rates, longer generation times, and greater lifetime reproductive success than either of the homozygotes (Table 1B). The substantial difference in fitness between black heterozygote and black homozygote wolves suggests that coat color per se might not be the cause of the heterozygote advantage—camouflage cannot explain the maintenance of the polymorphism. Presumably some other function of the gene, perhaps via its role in cellular immunity, determines the fitness differences (*27*).

Altering the mean environment affected all of the population biology parameters we calculated, with different parameters being most sensitive to changes in the mean value of intercepts of different functions (Fig. 2). For example, population size was most sensitive to perturbation of the intercept for the fertility function; coat color frequency was most sensitive to perturbations of the survival function intercept; the strength of viability and fertility selection was most sensitive to perturbation of the intercept of the body weight growth function for wolves ≥41.7 kg; and generation length was most sensitive to perturbation of the inheritance function. The way a population responds to environmental change, and which ecological or evolutionary parameters are most affected, depends on which functions are altered.

The direction of change in pairs of parameters can differ depending on the function intercepts that are perturbed (Fig. 2), demonstrating that different types of environmental change can

Table 1. Model performance. (**A**) Comparison between parameters estimated directly from data and those predicted from the baseline model. (**B**) Genotype-specific predictions of demographic rates and selected life history parameters.

A

Population biology parameter	Observed	Predicted
Mean population size	104.83	111.23
Minimum population size	59.00	93.72
Maximum population size	174.00	149.25
Frequency of gray coat	0.56	0.62
Generation length	5.05	4.70
Dispersion of reproduction	10.13	9.87
Mean body weight	44.25	45.15
Strength of viability selection	0.27	0.24
Strength of fertility selection	4.89	4.06

B

Genotype	AA	AB	BB
Phenotype	Black	Black	Gray
Annual survival rate	0.47	0.77	0.75
Annual recruitment rate	0.08	0.28	0.24
Generation length	2.4	4.91	4.5
Mean lifetime reproductive success	0.031	2.35	1.83
Mean frequency	0.02	0.36	0.62

generate a wide range of eco-evolutionary responses. For example, perturbing the mean intercept of the fertility function reduces the strength of viability selection and increases mean body weight, whereas perturbing the growth rate function for wolves ≥41.7 kg increases both the strength of viability selection and mean body weight. These results help explain why such a wide range of eco-evolutionary responses to environmental change is observed in nature (*5, 6*): the consequences of environmental change depend on whether survival, reproduction, development, or inheritance is most affected.

What are the consequences of altering how variable the environment is? Perturbing the standard deviation of the intercept distributions for each function, and the correlation in intercepts across functions, had little effect on all population biology parameters (Fig. 2). In a population model, it is straightforward to independently perturb the mean environment or how variable the environment is (*28*). In reality, environmental change alters both means and variances of year effects. However, our results suggest that changes in the average environment are likely to affect Yellowstone wolves to a much greater extent than changes in environmental variability.

Why do we see these results? Environmental variation causes the shape and size of the distribution to change from one time step to the next, but density dependence means that no part of the distribution consistently grows or shrinks with time—the genotype–body weight distribution attains a stationary stochastic distribution. When a function is changed, a new stationary stochastic distribution is attained, and the number of individuals at each genotype–body weight combination changes. As the shape and size of the stationary stochastic distribution change, so do the summary statistics that population biologists use to characterize aspects of the distribution, whether these parameters are calculated for each time step or for each cohort (Fig. 2). Perturbing different functions changes the stationary stochastic distribution that the population converges to.

If dispersal can be ignored, simultaneously predicting the dynamics of individual genotypes and phenotypes, life history parameters, and population dynamics only requires the identification of survival, reproductive success, development, and inheritance functions. There are many systems where such models could be constructed (SOM). Despite this, accurately predicting eco-evolutionary responses to environmental change for density-dependent populations living in variable environments is challenging. Environmental drivers that influence functions need to be identified. Biologists have made progress in characterizing how the environment can influence parameters in some of the functions that constitute

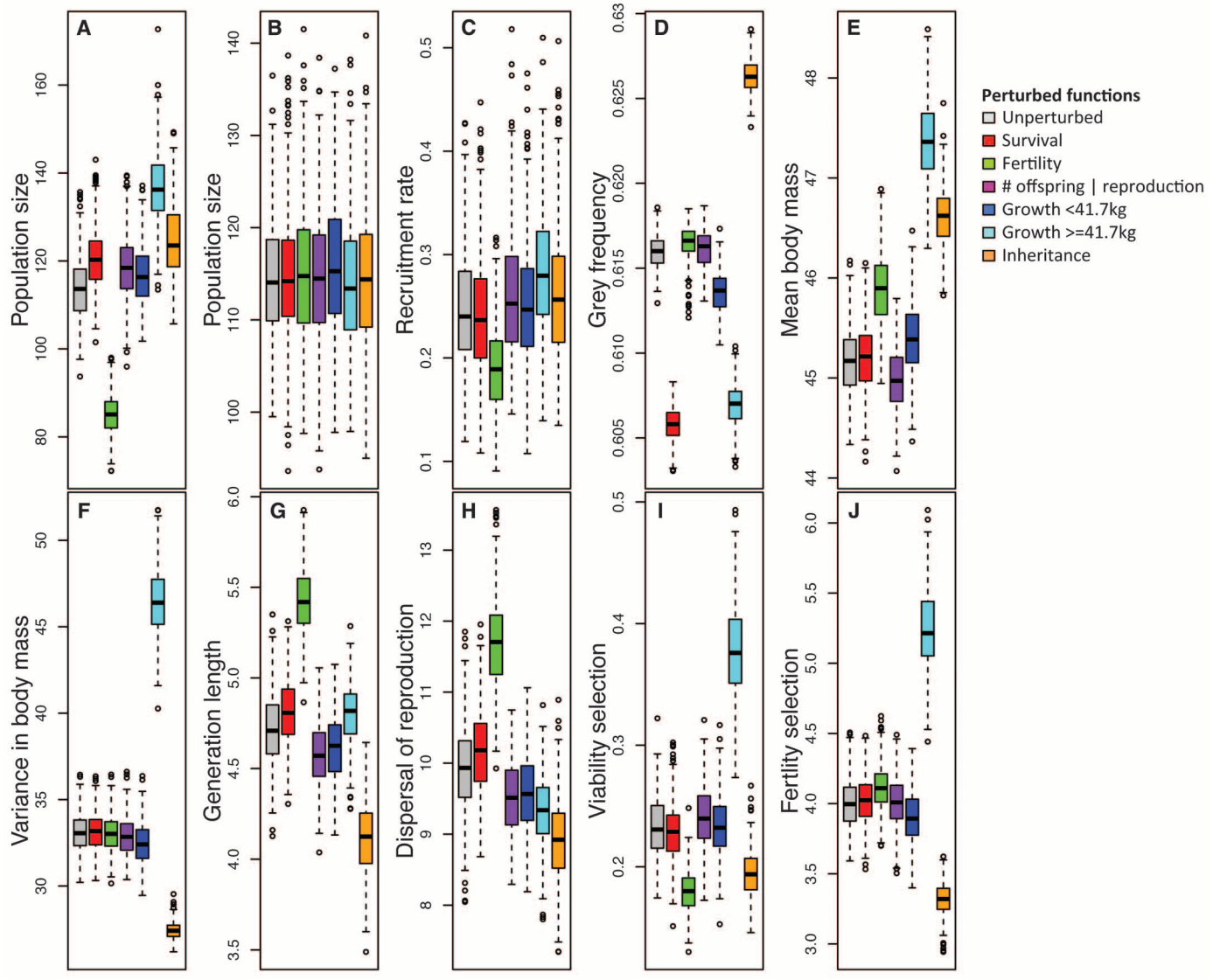

Fig. 2. Consequences of perturbing the mean value of function intercepts (**A**) and (**C** to **J**) and the standard deviation of the intercept distribution (**B**) on the distribution of various population biology parameters. The gray distributions represent values from a simulation with no function perturbed, and the colored distributions are from simulations in which one intercept distribution was perturbed. The dispersion of reproduction is the variance in generation length (SOM).

an IPM (*29*), but we are unaware of any field studies where drivers have been identified for all functions. Even if environmental drivers are identified, predicting how they may change in the future is currently unfeasible, because the environment that populations experience is complex, consisting of abiotic and biotic drivers that can interact, sometimes nonlinearly (*30*). Currently, the best that can probably be done is to explore the consequences of environmental change scenarios. For example, if we assume changes that reduce the mean of each intercept by 10%, we predict decreases in mean population size and the strength of both viability and fertility selection; no change in coat color frequencies; and increases in the variance in population size, mean body size, and generation length. In reality, we have little idea of the extent to which environmental change will affect each function, because key environmental drivers have yet to be identified for all functions, and the dynamics of those that have been identified are not well understood (*24*–*26*).

Although accurate prediction is currently not possible, our results do reveal that, for Yellowstone wolves, (i) environmental change will inevitably generate eco-evolutionary responses; (ii) change in the mean environment will have more profound population consequences than changes in the environmental variance; and (iii) environmental change affecting different functions can generate contrasting eco-evolutionary dynamics. Because IPMs are sufficiently general and because density dependence and environmental variation affect most populations, these conclusions are likely to extend to other systems. The construction and analysis of IPMs across a range of systems may provide support for this proposition. In addition to providing a tool to explore eco-evolutionary dynamics, IPMs have also been extended to include spatial variation and to identify evolutionarily stable strategies (*21, 22*), giving them potential to unify several subdisciplines of population biology, including population ecology, quantitative genetics, population genetics, and life history theory. They have not yet been extended to incorporate processes that generate novel genetic variation; the results we report arise via the shuffling of existing phenotypic and genetic variation via selection and plasticity. Our findings suggest that existing phenotypic and genetic variation within Yellowstone wolves is sufficient for environmental change to generate substantial evolutionary change that will occur in tandem with shifts in wolf life history and population dynamics. Although accurate prediction of the eco-evolutionary consequences of environmental change is currently unfeasible for most natural populations, our results help explain why it so widespread, and perhaps inevitable.

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Supporting Online Material

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Inhibition of Pyruvate Kinase M2 by Reactive Oxygen Species Contributes to Cellular Antioxidant Responses

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Control of intracellular reactive oxygen species (ROS) concentrations is critical for cancer cell survival. We show that, in human lung cancer cells, acute increases in intracellular concentrations of ROS caused inhibition of the glycolytic enzyme pyruvate kinase M2 (PKM2) through oxidation of Cys^{358}. This inhibition of PKM2 is required to divert glucose flux into the pentose phosphate pathway and thereby generate sufficient reducing potential for detoxification of ROS. Lung cancer cells in which endogenous PKM2 was replaced with the Cys^{358} to Ser^{358} oxidation-resistant mutant exhibited increased sensitivity to oxidative stress and impaired tumor formation in a xenograft model. Besides promoting metabolic changes required for proliferation, the regulatory properties of PKM2 may confer an additional advantage to cancer cells by allowing them to withstand oxidative stress.

Control of the intracellular concentrations of reactive oxygen species (ROS) is critical for cell proliferation and survival. In cells treated with growth factors, transient increases in ROS concentrations are implicated in enhanced cell proliferation through inhibition of phosphotyrosine phosphatases and PTEN, which allows amplification of tyrosine kinase and phosphatidylinositol-3 kinase (PI-3K) signaling pathways (*1*). However, high concentrations of ROS can also damage cellular components and compromise cell viability (*2*). Tumor suppressor and oncogenic pathways frequently mutated in cancer commonly result in increased accumulation of ROS (*3*–*7*). Furthermore, conditions associated with tumorigenesis such as hypoxia, matrix detachment, mitochondrial dysfunction, and inflammation can all lead to excess production of ROS (*8*–*12*). Therefore, cancer cells are particularly challenged in dealing with oxidative stress (*2*, *13*).

Supporting Online Material for

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Supporting Online Information

MATERIALS AND METHODS

The results reported in the paper are based on an integral projection model (IPM) of Yellowstone wolves. In the SOM we provide information on the IPM structure, functional forms used to construct the IPM, and parameter values for all functions.

The integral projection model and its assumptions

Integral projection models (IPMs) describe the dynamics of individual characters that may be continuous, like body size, or discrete, like genotype or age class (*10, 18, 20, 31*). They consist of functions that describe how demographic processes remove mass from the distribution (mortality and emigration), add mass to it (reproduction and immigration) and transform mass within it (development). In our IPM we do not consider immigration or emigration. We consequently have functions describing how

body weight and genotype influence survival, reproduction and inheritance and growth.

In the main text we refer to the functions as the survival function, the growth function, the annual reproductive success function and the inheritance function. The annual reproduction success function is the product of the fertility function and the number of offspring produced function; the growth function is a threshold function, with different intercepts and slopes above and below a threshold of 41.7kg. Here we define the functions, and give more detailed definitions:

The survival function: expected probability that an individuals with genotype, κ, and body weight, z, at time *t* survives to time *t+1*, $S(t, z, \kappa)$

The growth function: probability of an individual with genotype κ, and body weight, z, at time *t* that survives to *t+1*, grows to body weight, z' at *t+1*, $G(t, z'|z, \kappa)$

The annual reproductive success function: the expected number of offspring produced by an individual with genotype κ, and body weight, z, between times *t* and *t* and *t+1* that survive to recruit to the population at *t+1*, $R(t, z_1, \kappa_1)$

The inheritance function: the probability that a reproducing individual with genotype κ, and body weight, z, produces an offspring with genotype κ', and body weight, z', at time *t+1*, $I(t, z', \kappa'|z, \kappa)$.

These functions operate on the bivariate distribution of the genotypes and body weights at time *t*, $n(t, z, \kappa)$ to produce a bivariate distribution of the genotype and body weight at time *t+1*, $n(t + 1, z', \kappa')$. We can now write the IPM as:

$$n(t + 1, z', \kappa') = \sum_{\kappa} \int [G(t, z'|z, \kappa)S(t, z, \kappa) + I(t, z', \kappa'|z, \kappa)R(t, z, \kappa)]n(t, z, \kappa)\, dz.$$

The summations are taken over the discrete genotypes (AA, AB, BB) and across all values of body weight. The inheritance function contains several processes. The

distribution of offspring genotypes will depend on the breeding system and the inheritance mechanism. We assume Mendelian inheritance for the genotype and random mating with respect to the genotype and body weight. The assumption of Mendelian inheritance is justified for nuclear genes like the K locus in disploid species. The assumption of random mating would be violated if mates select one another as a function of their genotype. We are unaware of any evidence supporting assortative mating with respect to genotype at the K locus in gray wolves. To keep the model simple, we do not track the number of males and females within the population. We consequently assume that males and females have identical demographic functions. Gray wolves are sexually size dimorphic (*17*), so males do grow faster at some ages, but survival rates are similar. Given the mating system of wolves, and that typically only the dominant pair mate per pack, it is reasonable to assume that the distribution of reproductive success is similar between males and females. We consequently suspect that our assumption about identical male and female demography is unlikely to be substantially violated.

The final assumption we discuss concerns the inheritance mechanism for body weight. To avoid introducing further complexity into the model we assume that only one parent determines offspring weight at time t+1. If body weight is genetically determined then this assumption will not be supported in wolves. In general, the heritability of body weight in wild mammals tends to be low, in the order of 0.1, which means that additive genetic effects determine 10% of body weight. Maternal effects are typically more important in determining weight in mammals, so we argue that our assumption that only one parent influences offspring body weight is not too outrageous. All of the assumptions described above could be relaxed within the IPM framework, but doing so would complicate the model.

To explicitly incorporate random mating, Mendelian inheritance and one parent (the mother) determining offspring body weight into the model IPM we expand the $I(t, z', \kappa' | z, \kappa)$ function to,

$$I(t, z', \kappa' | z, \kappa) = P(\kappa' | \kappa_1, \kappa_2) D(t, z' | z_1, \kappa_1) M(z_1, \kappa_1 : z_2, \kappa_2)$$

where $M(z_1, \kappa_1 : z_2, \kappa_2)$ describes the probability of mating between a female with genotype $\kappa_1$and body weight z_1, and a male with genotype κ_2 and body weight z_2, $D(t, z' | z_1, \kappa_1)$ is a probability kernel describing the probability that a reproducing female with genotype $\kappa_1$and body weight z_1 produces an offspring that recruits to the population at *t+1* with body weight z', and $P(\kappa' | \kappa_1, \kappa_2)$ describes the probability that a mating between parents with genotypes κ_1 and κ_2 produces an offspring with genotype κ'. We can now rewrite the IPM as

$$n(t+1, z', \kappa') = \int G\left(t, z' \middle| z, \kappa\right) S(t, z, \kappa) n(t, s, \kappa) dz +$$

$$+ \sum_{\kappa} P(\kappa' | \kappa_1, \kappa_2) \int D(t, z' | z_1, \kappa_1) R(t, z_1, \kappa_1) n(t, z_1, \kappa_1)\, n(t, z_2, \kappa_2) M(z_1, \kappa_1 : z_2, \kappa_2) dz_1 dz_2$$

Because individuals do not change genotype with age, no function is required to describe how genotypes change within individuals. The probability kernels $D(t, z' | z_1, \kappa_1)$ and $G(t, z' | z_1, \kappa_1)$ capture plasticity. Individual with identical genotypes and body weights at time *t* do not necessarily have the same genotypes and body weights at time *t+1* – the probability functions describes the likelihood of them growing to a specific body weight. Similarly, two reproducing mothers at age *t* with identical genotypes and body weights that mate with males with identical genotypes do not necessarily produce offspring with identical body weights that recruit to the population at time *t+1*. $D(t, z' | z_1, \kappa_1)$ describes the probability distribution of possible offspring weights for such mothers.

Data used to parameterise the integral projection model

We used data collected from 280 radio-collared adult wolves of known sex and their offspring living in Yellowstone between 1998 and 2009 (*16, 17*). Data collection protocols have been published elsewhere and are not repeated here (*16, 17*) – these papers also report existing wolf data. We use the following data to parameterise the IPM reported in the paper:

- Wolf identity: the unique identity of each radio-collared individual
- Wolf year: the wolf year ran from April 1st in year *t*, shortly before birth occurs to March 31st in year *t+1*. Individuals born in year *t* recruit to the population immediately before their first birthday.
- Fate: whether the wolf was known to have survived wolf year *t* (1), died and radio-collared recovered (2) or fate unknown (0: died and not found or left the park and lost from study)
- Age: in years
- Sex
- Coat colour: black or grey
- Genotype at the K locus: coat colour is determined by genotype at the K locus. In Yellowstone wolves there are two alleles at that locus. The black allele is dominant, meaning heterozygotes are black
- Weight on 1st April at year *t* (kg): wolves are caught between January and March in year *t-1*, with weight on April 1st at year *t* estimated by using an individual's residual from a regression line between capture date and body weight and expected weight on April 1st
- Number of recruits: the total number of pups produced by each wolf determined by observation and genotyping. Observational data are collected daily by wolf project staff and volunteers from each pack that could be located
- Predicted weight of recruits on April 1st when aged 1: estimated in the same way as the weight of adults

- Pack Identity: the name of the pack in which the individual lived
- Population size: the total number of wolves known to be living within Yellowstone on April 1st of each year, compiled from observations taken at least weekly of each pack within the park.

Statistical analysis

Our aim was to construct an integral projection model that simultaneously predicted body weight, genotype frequency, population dynamics and life history descriptors (Table 1, main text). Prior to any analyses we devised a strategy to parameterise the IPM to maximize use of the available weight data. First, we assumed that all functions should be linear (or linearized when data were binomially distributed). Non-linear functions would have meant the IPM would have contained additional parameters, complicating its analysis. Second, to ensure the IPM consisted of functions parameterised in similar ways, we retained sex and population size as fixed effects in all statistical models, even if they were statistically non-significant. Similarly, year and pack identity were fitted as random effects in all models. Our justification for retaining non-significant parameters is that all parameters included in the IPM were corrected for the same nuisance factors (see supplementary discussion). Third, if published work had identified associations between body weight and genotype with survival, annual reproductive success, growth and inheritance we used the same functional form in the model (see Growth function below) to retain consistency with previous analyses. We refer readers to references (2,6, 16, 17) for further information and data.

When predictions were made from our initial model it over-estimated population size and the frequency of gray wolves and under-estimated the frequency of black wolves (see improving model fit). Because our primary objective was to construct an IPM that captured the essential features of the wolf population, we decided to improve

model fit by re-parameterizing reproductive success functions (see below). We chose to re-parameterise this function because naïve estimates of reproductive rates based on the number of recruits divided by the size of the adult population suggested predicted values were too large. We suspect this may have arisen because dominant pairs may be more likely to be targeted for radio collaring as adults. We re-parameterised the annual reproductive success function using probe matching (*32, 33*) (see Improving model fit).

Although re-parameterizing the annual reproductive success function improved the model to allow us to achieve our primary aim of constructing an IPM that performed adequately in describing observed population parameters of the wolf population, the re-parameterisation did not influence our general conclusions (Table S3).

Because our primary aim was to construct an IPM that described observed patterns in the wolves, the appropriate examination of model fit is a comparison of predictions with observation (*32, 34*). However, a lack of model fit could arise if statistical models failed to capture patterns in data, so visual examination of residuals around each statistical model suggested all fits were adequate (results not shown).

We describe the parameterisation of each of the functions below. Table S1 provides functional forms for the functions included in the IPM, and Table S2 provides parameter values. Note that sex and pack identity were included in all statistical analyses as nuisance variables, but were not incorporated into the IPM. Because the focus on the paper is on model predictions rather than data analysis we only provide estimates of parameters used in the IPM and do not report estimates for sex and pack identity in Table S2.

$S(t,z,\kappa)$: association between body size, genotype and survival

Not all wolves were weighed each year they were known to be alive. The lack of weights for these individuals complicates mark-recapture analysis (*35*). In addition,

not all wolves were weighed in all years they were known to be alive. To maximise data we first conducted a mark-recapture analysis to estimate recapture and recovery rates. The log of the ratio of these rates were then used as an offset in generalised linear mixed model to provide unbiased estimates of how body weight, sex, population density, year and pack identity explained variation in whether an individual was seen (1) or not (0) (*36*)

We consequently used a two-step process to maximise available information:

1) From the data file we constructed re-sighting and recovery (whether an individual is found dead) histories for each individual. A re-sighting and recovery history consists of a string of 0s, 1s, or 2s for each individual. A 0 means the animal was not observed. A 1 means the animal was observed, and a 2 means the animal was found dead. We found recovery rates varied between black and gray wolves, while recapture rates did not vary with coat colour. Recapture and recovery rates did not vary with time.
2) We combined the unknown fates with the known mortalities giving them a score of 0 and conducted a generalised linear mixed effect model of the binomial data (1=present in the population, 0=not present in the population). The ratio calculated in step (1) was fitted as an offset to correct for the fact that 0s contained two fates: death and disappearance (*36*). The variance component for year, and regression estimates for population size, body size and genotype were used to construct the IPM.

<u>$R(t,z,\kappa)$: associations between body size, genotype and the number of recruits produced</u>

The distribution of the number recruits produced by an individual was zero inflated. The number of recruits produced conditional on producing at least one recruit, was

approximately normally distributed. We consequently conducted two separate analyses, and multiplied the resulting functions together (*37*).

1) We fitted a generalised linear mixed effects model to the binomial distribution of producing zero recruits (0) or at least one surviving recruit (1). Pack identity and year were fitted as random effects, and population size, sex, body size and genotype as fixed effects. This is the fertility function described in the main text.
2) We next analysed half the number of recruits produced among those individuals that successfully recruited one offspring with a linear mixed effect model. We work with half the number of recruits as both sexes are included in the data file. Pack identity and year were fitted as random effects, and population size, sex, body size and genotype as fixed effects. This is the number of offspring function described in the main text.

The annual reproductive success function is the product of the each of the functions identified in steps 1) and 2).

G(t,z'|z,κ): Growth function

The growth function describes how individuals grow from weight *z* in year *t* to weight *z'* in year *t+1*. Its parameterisation requires two steps: a function describing the expected growth rate, and a function describing the variance around the mean growth rate. To maximize all weight data we examined how mean weight varied with age, and then transformed the age-mean weight function into a function describing mean weight in year *t+1* as a function of mean weight in year *t*. We then fitted this function to data collected from individuals with repeated weights collected in different years, and used the residuals around this function to estimate the variance.

MacNulty *et al.* (*17*) examined patterns of age and size in Yellowstone wolves. They identified a threshold of below which young, small wolves grew, while above which

wolves grew more slowly. We used their form of model. We modelled body weight as a function of age, sex, population density, genotype at the K locus and pack identity and year as random effects. Our results confirmed the age-specific pattern of (*17*) – we estimated a threshold at 41.7kg. We now transformed predicted age-specific weights onto a scale of expected weight at time *t* against weight at time *t+1*. On this scale we obtained the following equation,

$$E(z(t+1)) = \begin{cases} 41.7, & \bar{z}(t) < 41.7 \\ 22.05 + 0.55E(z(t)), & x \geq 41.7 \end{cases}$$

where $E(z)$ represents the expected body weight in kg. What this means is that individuals <41.7kg are predicted to grow to 41.7kg, while the expected weight of wolves above 41.7kg is determined by the linear regression.

Next, we required a function to describe the variation in growth (*10*). We calculated residuals of body weight from the expected weight function of adults weighed in different years. There was no evidence of patterning in the residuals with body weight, so we squared these residuals and took their mean. Following (*10*) we then constructed the probability kernel using the equation for the growth kernel in Table S1 and the mean of the squared residuals (Table S1).

D(t,z'|z,κ): Inheritance function

To parameterise the body weight inheritance function we fitted a linear mixed effect model of the body weight of recruits at time *t+1* against the body weight of parents at time *t*. There were few data for this regression. Parent sex, recruit sex, population size and body weight were fitted as fixed effects and year was fitted as random effect. To construct the parent-offspring body weight kernel we analysed the square of the residuals around this function as a function of body weight.

To keep the number of model parameters low, and because there was no evidence that either of the probability kernels varied with genotype, we used the same *D(t,z'|z,κ) and G(t,z'|z,κ)* for each genotype.

Improving model fit

An IPM constructed from the parameters identified through the regression analyses described some observed patterns well, but over-estimated mean population size by 15%, and predicted a grey coat frequency of 85% rather than the observed 56%. To improve model fit we allowed annual reproductive success parameters to vary within the standard errors identified from the regression functions and estimated the following parameters from the IPM: mean population size, genotype frequencies of the gray homozygote, mean body weight. These estimates were respectively divided by the observed mean population size, the observed mean frequency of the gray genotype, and observed mean body weight, and 1 subtracted from each. Finally we took the absolute value of these three quantities and summed them. If we define observed population size, mean body weight and gray genotype frequency respectively as $\bar{N}$, $\bar{Z}$ and $\bar{\kappa}$ and expected population size, mean body weight and gray genotype frequency respectively as $\hat{N}$, $\hat{Z}$ and $\hat{\kappa}$ the quantity we calculate is:

$$\rho = \left| \left(\frac{\bar{N}}{\hat{N}} - 1 \right) + \left(\frac{\bar{Z}}{\hat{Z}} - 1 \right) + \left(\frac{\bar{\kappa}}{\hat{\kappa}} - 1 \right) \right|$$

We varied parameter values using the Metropolis algorithm to minimise this quantity. This approach is based on the probe matching method of (*33*). We only explored parameter values within the parameter standard errors estimated from the regression models. Parameter values for the final model are provided in Table S2 and observed and predicted population biology parameters are given in the main text. individual and population level wolf data to examine model fit are available on Dryad: doi:10.5061/dryad.bp23483h

Numerical implementation – running the simulation

To aid analyses we approximate the stochastic density-dependent IPM as a high dimensional stochastic, density-dependent matrix model consisting of a sequence of matrices (*10, 20*). These matrices then determine the dynamics of the bivariate distribution of genotype and body weight when described by a vector.

We approximate the distribution n(t,z,κ) as a vector of length 300: **n**(*t*). Elements 1 to 100 describe the number of individuals in body weight classes of width 0.7kg ranging from 0kg to 70kg (e.g. $0 < z' < 0.7$, $0.7 < z' < 1.4$, …, $69.3 < z' < 70$ for genotype AA at the K locus (black wolves). Elements 101 to 200 describe the number of individuals in each of the body size classes for genotype AB (black wolves), while the final 100 elements describe body size classes for genotype BB (grey wolves). The lower and upper body weight values are well beyond observed wolf body mass and the model does not predict individuals of this weight. See (*10, 20*) for further information on integration limits and mesh sizes.

This vector is operated on by four square matrices of dimension 300 at each time step. The first matrix, **S**(*t*), is a diagonal matrix describing the expected survival rate of individuals in each body size-genotype class given the population density and environmental state at time *t*. The values in the matrix are calculated using the mid-point body weight value for each body weight-genotype class from the function $S(t,z,\kappa)$. Specific values at time *t* are determined by population density and a year effect drawn from a normal distribution with mean equal to the overall intercept of the survival function and variance equal to the variance component for the random effect of year.

The second matrix, **R**(*t*), is also a diagonal matrix, and describes the expected number of recruits produced by individuals in each body size-genotype class given the population density and environmental state at time *t*; values are calculated using

the function $R(t,z,\kappa)$. At time *t,* predicted values for the fertility function and the number of offspring produced function are calculated given population size and year effects. The mean of the year effect distribution for the fertility function is the intercept of the function, and the variance is equal to the variance component for year. An equivalent approach is used to generate predictions for the number of offspring produced function. The predicted values for each function are then averaged.

The third matrix, **G**(*t*), describes the probability that an individual in stage class *i* at time *t* that survived to time *t+1* transitioned to stage class *j* at time *t+1* given population size and year effects. Transition rates are calculated from the function $G(t,z'|z, \kappa)$. As with **S***(t)* and **R***(t)* we use the intercept and variance component from the statistical model to determine year effects. Many elements in this matrix are zero, as individuals cannot move between genotype classes.

The fourth matrix, **I**(*t*), describes the probability that a pup born to a mother of body size *z* and genotype κ_1 and father with genotype $\kappa_{2\,1}$ at time *t* that survives to recruit to the population at time *t+1* does so at body size z' and genotype κ. The matrix is calculated from the functions $D(t,z'|z, \boldsymbol{\kappa_1})$, $P(\boldsymbol{\kappa'}|\boldsymbol{\kappa_1}, \boldsymbol{\kappa_2})$ and $M(z_1, \kappa_1{:}z_2, \boldsymbol{\kappa_2})$. Year effects are simulated as they are for the other functions.

Population size at each time step is calculate as $N_t = \Sigma\mathbf{n}(t)$.

In each model run we simulate the population for 700 years and discard the first 200 years, as these include transient dynamics as the population converges from an arbitrary starting population structure to a stationary demographic distribution. We report results from the remaining 500 years of the simulation.

When we perturbed model parameters, we multiplied by them 1.1. Obviously, this increased positive parameters and decreased negative ones. When interpreting

results it is important to note the sign on the parameter (Table S2). In our initial model, year effects across functions were independent. However, when exploring how correlation between year effects impacted results, we conducted two further simulations. One, when year effects were perfectly positively correlated across functions, and one when they were perfectly negatively correlated.

R code to run the model (text file), is available on Dryad: doi:10.5061/dryad.bp23483h.

SUPPLEMENTARY RESULTS

Model parameters included in the IPM and their confidence intervals are displayed in Table S2, and the effects of body weight, genotype and population density on mean survival and annual reproductive success in Figure S1. Observed population size, the frequency of the gray coat colour, the body weight distributions and the strength of fertility and viability selection in each year are displayed in Figure S2. Individual and population level data underpinning this figure are available at doi:10.5061/dryad.bp23483h. Examination of this figure shows that the model captures the mean body weight distribution well, does adequately in capturing mean values of population biology quantities measured over years, but does not capture year-to-year variation in some other population biology quantities.

SUPPLEMENTARY DISCUSSION

Model performance

The model performed well in capturing the expected distribution of body size and mean values of population biology parameters averaged across years (Table 1, main text, Figure S2). Model estimates of temporal variation in population size, mean body weight and the strength of viability and fertility selection under-estimated observation. There are several reasons why this may be the case, some to do with the model structure and parameterisation, and some to do with the wolf population.

First, this is a model - it is a simplification of the real world, so we would not expect it to predict everything perfectly. The model assumes linear(ized) functions, and does not incorporate within and between pack dynamics, age-structure or any traits other than body weight or genotype. These processes, and others, are likely to impact the population dynamics, life history and character distributions to some extent. We did not incorporate these processes into the model, as our aim was not to construct a sim-Yellowstone, but rather to show that the dynamics of genes, phenotypes, life history and population size can be investigated using only four types of function. Second, the failure of the model to capture temporal variation in mean body size and viability and fertility selection may be related. The strength of selection within a year depends upon the variation in body weight. If the distribution of body weight varies with time – and the observed mean does vary – then the strength of selection may also vary. The greater variation observed in data compared to prediction could arise if the strength of density-dependence or the slope of the associations between body weight and survival, reproduction, growth or inheritance varies with time (*38*). An IPM could be constructed so that all parameters, rather than just function intercepts, vary with time. However, this would complicate the model substantially.

The wolf re-introduction occurred in 1995 and 1996 (*39*). The population then showed a period of growth, before flattening off, and then decreasing. When a population increases from small size before fluctuating around carrying capacity, transient dynamics can persist for many years (*40*). The profound changes that have occurred in Yellowstone in recent decades (*11, 13, 14*) further suggest that the system is in a state of transience. In contrast to the observed population, the predicted dynamics are from the model run at equilibrium. This difference between model and observation could also contribute to the mis-match in Figure S2.

Data requirements for parameterising an IPM

All IPMs model the dynamics of continuous characters. Such characters may be morphological, like body weight, behavioural, like personality, or biochemical, like immune response – essentially anything that is continuous that can be measured on an individual. IPMs can be extended to incorporate multiple continuous characters, or a combination of continuous and discrete characters. Discrete characters can be genotype, age, sex or even social rank (*18, 31*).

In their simplest, IPMs consist of functions that describe how a single character influences survival, reproduction, development and inheritance. They can easily be extended to include functions describing how the character(s) influence immigration and emigration. All that is required to construct an IPM are equations for the functions that constitute it. These can be estimated from data that biologists routinely collect (*10*). In general, it is easiest to estimate these functions from data collected from individuals that are monitored over their lifespan. However, it may be possible to estimate functions from individuals that are not marked, using laboratory experiments.

The more data that are available, the greater the confidence one should have that functions can be identified and parameterised that accurately reflect reality. As with any population modelling approach, if data are limited the model may fail to provide an accurate characterisation of the system. However, despite this, perfect population coverage is not required. Biases in data, caused by a failure to always observe or capture all individuals within a population, or through sampling error, can be statistically corrected. For example, mark-recapture analysis (*35*) is widely used to estimate unbiased survival estimates from sparse data, and Bayesian methods are now frequently used to correct for bias, sampling and observation error, especially when data are scarce (*41*). Statistical models can be as complicated or as simple as the modeller desires, and terms fitted into statistical models do not need to be

incorporated into IPMs – they can simply be fitted into statistical models to provide unbiased estimates of parameters that will be included in the IPM. IPMs can also be constructed to incorporate both fixed and random effect estimates (as we have done).

To date IPMs have only been published for animal and plant systems where extensive data exist across multiple years (*18, 21, 42, 43*). This does not preclude IPMs being constructed for systems where data are sparser. IPMs are a fabulous modelling tool, which are straightforward to parameterise, but their utility does not make them a substitute for collecting data. On the contrary, our results suggest that accurate prediction will require data that accurately captures how both biotic and abiotic environmental drivers impact character-survival, character-reproduction, character-development and character-inheritance functions.

Various choices need to be made when parameterising IPMs. The regression methods we used to identify survival, reproductive success and inheritance functions that we use are standard (*10, 18, 20*). However, the approach we used to identify the growth function is new. The reason we chose this approach was to allow us to use weights from wolves that were not captured in consecutive years. Age-mean body weight functions exist for several species when repeated measures on individuals may be scare; we consequently suspect that our approach provides the potential for IPMs to be fitted to species where individual-based data are less widespread.

IPM parameterisation also requires decisions about which terms to include in regression functions. Either the same terms can be retained in all statistical functions, as we have done, regardless of their significance, or minimum adequate statistical models can be identified for each function. The choice of approach will depend upon why the model is being constructed. The reason we retained

statistically non-significant terms in models is that their impacts may combine across functions to generate significant dynamical impacts. For example, insignificant density effects in each function could combine to generate significant density-dependence at the population level (*38*). If non-significant density effects had been removed from each function in such scenario a density-independent model would have been constructed even though density-dependence may be detectable in the analysis of the population dynamics.

Model re-parameterization

Re-parameterisation of the IPM improved model fit, and it may be possible to improve it further through the use of alternative statistical methodologies – for example, by using state-space models to estimate all model parameters simultaneously or through extending the IPM to incorporate additional aspects of wolf biology. In future work we will explore these issues further. However, importantly, the results we have obtained via altering parameter values reveal that key conclusions reported in the paper are likely to hold across model parameterisations. For example, both parameterisations of the model predicted maintenance of the genotype through heterozygote advantage, so our general biological insights are not influenced by the model parameterisation. However, the relative frequencies of the genotypes do differ between parameterisations.

Fixed and random effects of the environment

Most previous stochastic structured population models have either incorporated the dynamics of specific environmental drivers (*44*) or have considered the environment as consisting of a set of discrete states – typically based on observation of different year types (*28, 45*). (*20*) discuss how the environment could be treated as a continuous quantity, proposing an approach similar to that which we implement. The

advantage of this approach is fewer parameters need to be estimated from statistical models.

The environment can be treated as either a continuous or a discrete entity. Some environmental drivers are clearly continuous, including variation in the weather. Other environmental drivers will be discrete, including catastrophic events like volcanic eruptions. Fortunately the Yellowstone caldera has remained intact during the course of the wolf study! The choice of whether to model the environment is discrete or continuous will depend upon whether key environmental drivers are continuous or discrete.

SUPPORTING ONLINE FIGURES

Figure S1. Effects of density-dependence on the association of genotype and body weight with the probability of survival and the number of offspring produced from the functions used in the IPM. The left-hand panels are evaluated at mean density, the right-hand panels at mean body weight. The upper lines represent black heterozygotes, the grey lines represent grey wolves and the lower black lines represent the black homozygote.

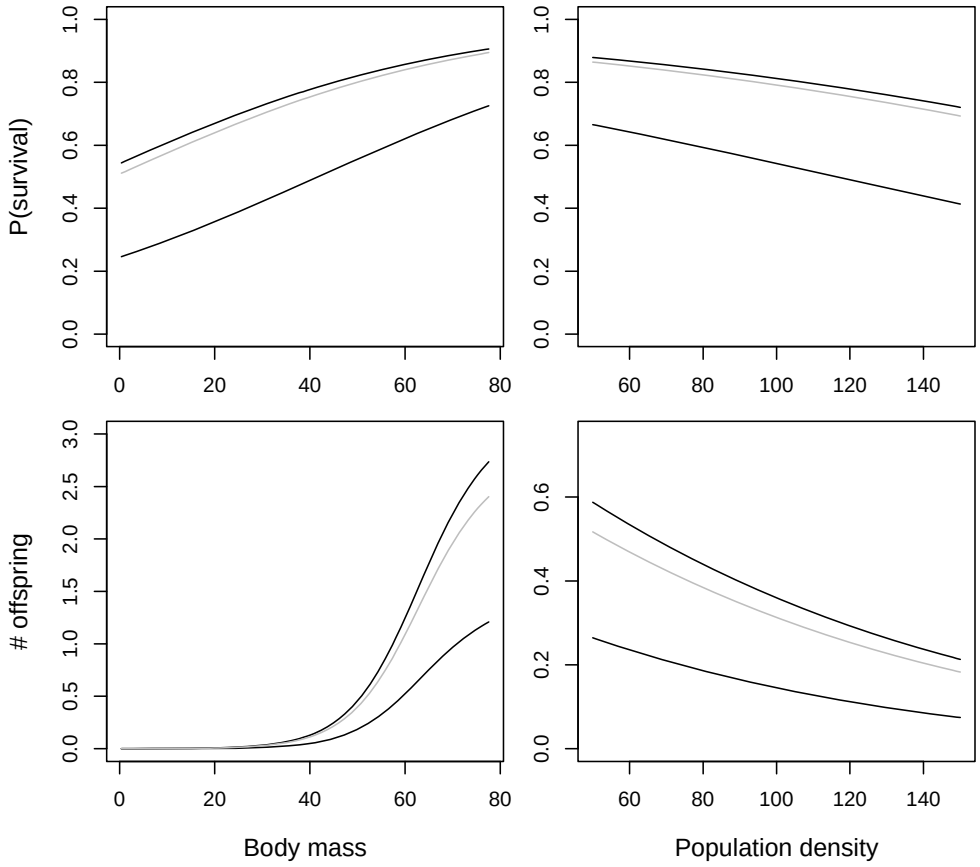
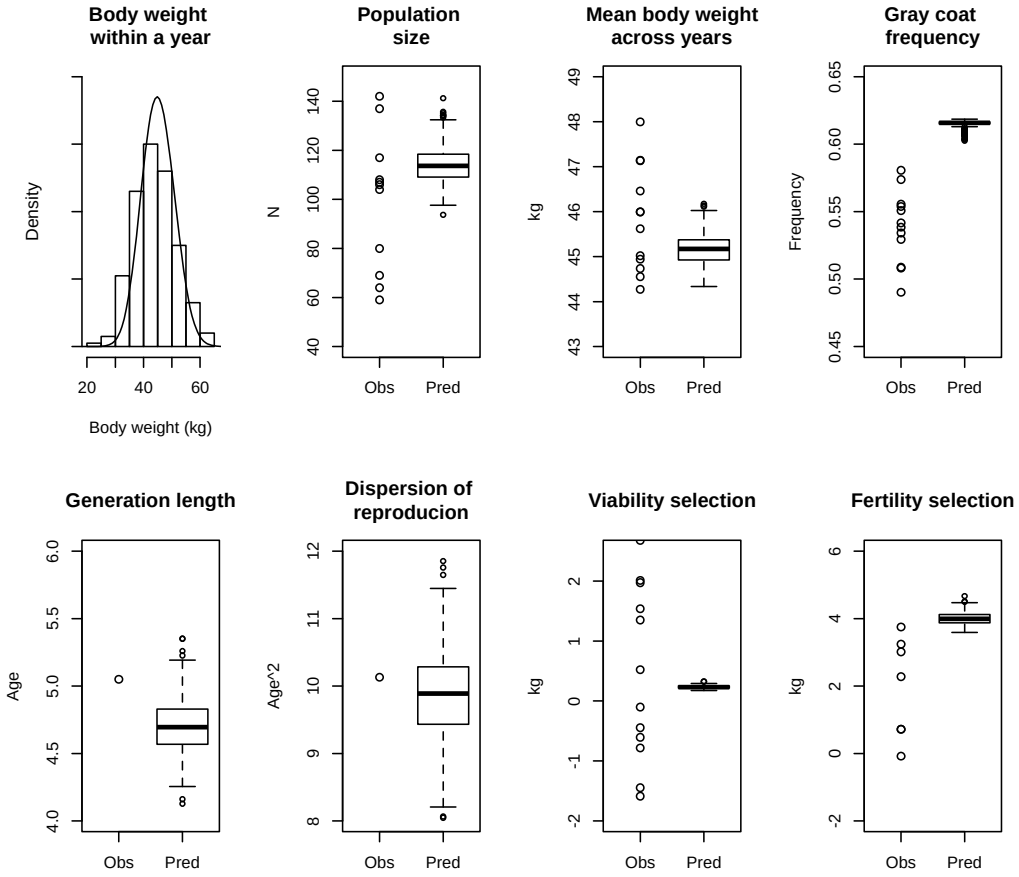

P(survival)
# offspring
Body mass
Population density
0.0
0.2
0.4
0.6
0.8
1.0
0.5
1.5
2.0
2.5
3.0
0
20
40
60
80
100
120
140

Figure S2. Examination of model fit – comparison of observation and prediction.



SUPPORTING ONLINE TABLES

Table S1. Form of the functions used to construct the IPM and parameter values. The functions μ_i and Ω_i are linear functions of body weight, Z, genotype, κ, population density, N, and year effect of the form $\phi(\beta_0, \sigma^2) + \beta_1\kappa + \beta_2 Z + \beta_3 N$. $\phi(\beta_0, \sigma^2)$ is the distribution of year effects with mean β_0 and variance σ^2. μ_i describes the mean effect of variables on survival, fertility, growth and body weight inheritance, while Ω_i is calculated by analysing the square of the residuals around μ_i. Parameter values are given in table S2. Note that sex and pack identity were included in statistical analyses but were not modelled within the IPM

Function	Expression
Survival functions	$S(t,z,\kappa) = \frac{1}{1+e^{-\mu_s}}$
Recruitment function	$R(t,z,\kappa) = \frac{\mu_r}{1+e^{-\mu_b}}$
Body weight inheritance kernel	$D(t,z'\|z,\kappa) = \frac{1}{\sqrt{2\pi\Omega_d}} e^{\frac{-(z-\mu_d)^2}{2\Omega_d{}^2}}$
Body weight growth kernel	$G(t,z'\|z,\kappa) = \frac{1}{\sqrt{2\pi\Omega_g}} e^{\frac{-(z-\mu_g)^2}{2\Omega_g{}^2}}$

Table S2. Parameter values for the functions in Table S1 with their approximate 95% confidence intervals estimated as +/-1.96*standard errors. These are parameter values use in the baseline model.

Function μ	AA intercept β0	AB intercept β1	BB intercept β1	Body mass β2	Density β3	sqrt(Variance component) σ^2
Survival	-0.237 (-0.856,0.382)	1.059 (-0.073,0.894)	0.964 (-0.063,1.990)	0.027 (0.004,0,040)	-0.012 (-0.016,-0.008)	0.100
Reproduce or not	-7.489 (-3.209,-11.769)	-7.489 (-3.209,-11.769)	-7.489 (-3.209,-11.769)	0.136 (0.090,0.182)	-0.010 (-0.007,-0.013)	0.224
# Offspring	1.468 (0.1566,2.780)	3.470 (2.588,4.353)	3.033 (1.949,4.118)	0.007 (-0.009,0.016)	-0.006 (-0.002,0.002)	
Growth rate (<41.7kg)	41.655 (36.187,,47.123)	41.655 (36.187,,47.123)	41.655 (36.187,,47.123)	0.000 NA	-0.004 (-0.010,0.002)	0.316
Growth rate (41.7kg+)	22.040 (16.572,27.508)	22.040 (16.572,27.508)	22.040 (16.572,27.508)	0.550 (0.33,0.77)	-0.004 (-0.010,0.002)	
Inheritance Ω	38.616 (32.44,44.79)	38.616 (32.44,44.79)	38.616 (32.44,44.79)	0.063 (0.009,0.117)	-0.005 (-0.41,0.36)	0.224
Growth rate variance	19.830 (12.191,27,469)	0.000 NA	0.000 NA	0.000 NA	0.000 NA	0.000
Inheritance variance	22.251 (14,139,36.920)	0.000 NA	0.000 NA	0.000 NA	0.000 NA	0.000

Table S3. The effect of improving model fit on results. The table describes how perturbing mean intercepts for the original parameterisation (ORIG) and the improved parameterisation (IMP) changes the mean value of population biology parameters in relation to mean population biology parameters from the unperturbed models. Plus symbols (+ and ++) refer to cases when means of the perturbed population biology parameter distribution are larger for the unperturbed distribution, while minus signs (- and --) identify the converse. Two symbols represent significance at $p<0.05$ on a t-test, single symbols represent differences at $p<0.5$ and = represent differences at $p>0.5$. Improving model fit did not substantially impact our conclusions: there were only eight cases when significant levels changed (green cells); the direction of change was never altered.

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