Bigger Is Fitter? Quantitative Genetic Decomposition of Selection Reveals an Adaptive Evolutionary Decline of Body Mass in a Wild Rodent Population

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Abstract

In natural populations, quantitative trait dynamics often do not appear to follow evolutionary predictions: Despite abundant examples of natural selection acting on heritable traits, conclusive evidence for contemporary adaptive evolution remains rare for wild vertebrate populations, and phenotypic stasis seems to be the norm. This so-called 'stasis paradox' highlights our inability to predict evolutionary change, which is especially concerning within the context of rapid anthropogenic environmental change. While the causes underlying the stasis paradox are hotly debated, comprehensive attempts aiming at a resolution are lacking. Here we apply a quantitative genetic framework to individual-based long-term data for a wild rodent population and show that despite a positive association between body mass and fitness, there has been a genetic change towards lower body mass. The latter represents an adaptive response to

viability selection favouring juveniles growing up to become relatively small adults, i.e. with a low *potential* adult mass, which presumably complete their development earlier. This selection is particularly strong towards the end of the snow-free season, and it has intensified in recent years, coinciding which a change in snowfall patterns. Importantly, neither the negative evolutionary change, nor the selective pressures that drive it, are apparent on the phenotypic level, where they are masked by phenotypic plasticity and a non-causal (i.e. non-genetic) positive association between body mass and fitness, respectively. Estimating selection at the genetic level thereby enabled us to uncover adaptive evolution in action, and to identify the corresponding phenotypic selective pressure. We thereby demonstrate that natural populations can show a rapid and adaptive evolutionary response to a novel selective pressure, and that explicitly (quantitative) genetic models are able to provide us with an understanding of the causes and consequences of selection that is superior to purely phenotypic estimates of selection and evolutionary change.

Introduction

Given the rapid anthropogenic environmental changes experienced by organisms around the world, there is an increasing need for an ability to understand and predict the evolutionary dynamics of wild populations [1, 2]. Despite good empirical examples of the adaptive evolution of traits with a simple genetic architecture [3–5], the picture is very different for quantitative traits, which in most cases are a function of many genes of small effect [6]. Although these are the traits that are of most interest to evolutionary biologists [7, 8], predictive models of quantitative trait evolution have largely failed when applied to data from wild populations [9].

Although there is an abundant literature showing that both directional selection [10, 11] and heritable genetic variation [12, 13] are common, these pre-requisites of Darwinian evolution rarely allow us to explain evolutionary trends retrospectively, let alone to make predictions for the future [9, 14]. For example, both natural and sexual selection almost universally favour larger and heavier individuals [15]. Furthermore, size-related traits are generally moderately heritable [12, 13], and averaged across the 151 estimates compiled in [13], the heritability of body mass is 0.33 ± 0.02. These levels

of directional selection and heritability generate an expectation of rapid evolution of   16
larger body size. Nevertheless, while species do tend to get larger over geological   17
timescales [16–19], this rate of evolution is orders of magnitude slower than predicted   18
from the strength of selection and heritability observed in contemporary   19
populations [9, 20, 21].   20

On the whole, conclusive evidence for the contemporary adaptive evolution of   21
quantitative traits in wild vertebrate populations is remarkably scarce and elusive [9, 14],   22
and good examples —Trinidadian guppy life-histories [22], human reproductive timing   23
and educational attainment [23, 24], timing of pink salmon migration [25] and big-horn   24
sheep horn size [26]—can be counted on the fingers of one hand. Furthermore, of these   25
studies, those dealing with humans might not be representative of wild populations, [26]   26
reported a response to harvesting-induced, artificial rather than natural selection, and   27
despite considerable effort to uncover any evolutionary consequences of climate   28
change [2, 27–29], only [25] were able to demonstrate an adaptation to climate.   29

Our apparent inability to reconcile predictions of evolutionary change based on   30
estimates of selection and genetic variation with the (lack of) of phenotypic change   31
observed, i.e. the 'stasis paradox' [9], is a major concern in urgent need of a resolution.   32
Given how commonly evolutionary predictions fail to capture observed trait dynamics,   33
some have concluded that there are fundamental problems that prohibit the application   34
of quantitative genetic methods to natural populations [30, 31]. However, there are in   35
fact three theoretically well-developed (quantitative genetic) explanations for this   36
mismatch.   37

First, many studies base their conclusion of evolutionary stasis on the observed   38
phenotypic trend not being significantly different from zero, but they do not explicitly   39
compare the observed change to the change predicted [32]. Hence, at least some cases of   40
phenotypic stasis may be the result of a lack of statistical power. Additionally,   41
evolutionary change does not need to be apparent at the phenotypic level. Instead, it   42
may be masked by phenotypically plastic changes, which may be several-fold larger   43
and/or go opposite to the genetic change [33]. For instance, while a change in the   44
environment may generate selection favouring an increase in the frequency of alleles   45
promoting fat accumulation, at the same time this may create a food shortage, leading   46
to a plastic decrease of fat reserves. As a consequence, an evolutionary trend may be   47

masked by a counteracting plastic change, resulting in "cryptic evolution" [34, 35].   48

Second, the observed rates of phenotypic change (which as outlined above may   49
provide poor estimates of evolutionary change) are typically compared to predictions   50
from the univariate breeder's equation, i.e. the product of selection and heritability,   51
where selection is quantified as the phenotypic covariance between the trait of interest   52
and fitness. In natural systems, selection however rarely acts on traits in isolation [36].   53
If these traits are genetically correlated to the focal trait, they may significantly alter   54
the focal trait's evolutionary trajectory [37, 38]. While the role of genetic correlations   55
among traits within the same individual [39] or between the sexes [40] has received   56
substantial attention, the potential role of genetic constraints resulting from genetic   57
correlations between traits expressed in different life-stages has received far less   58
attention. In particular parent-offspring conflict, e.g. a genetic trade-off between   59
offspring quality and fecundity [41], may constrain the evolution of size [42], with   60
positive directional selection on offspring size counterbalancing selection against   61
investment per offspring on the level of parents [43].   62

Finally, even in the absence of selection on correlated traits, it is challenging to   63
obtain an estimate of the strength of natural selection that is unbiased by the existence   64
of a third, non-genetic variable that influences both the trait and fitness [44]. Although   65
the univariate breeder's equation assumes that the covariance between phenotype and   66
fitness is solely the result of a causal relationship between the two, this assumption is   67
likely to be violated, especially in natural populations [14, 45]. For instance, a trait that   68
plastically responds to food availability, such as body mass, is likely to covary with   69
fitness at the phenotypic level, irrespective of the causal effects of this trait on fitness: 70
individuals that have access to more food are heavier and reproduce more [37, 46].   71
However, because the fitter individuals are not genetically different in terms of body   72
mass, this covariation has no evolutionary consequences, even if body mass is   73
heritable [44].   74

While these difficulties have been discussed previously, and studies regularly note   75
that the mismatch between the observed and predicted response may be attributable to   76
any of them, they are rarely accounted for in an explicit, quantitative manner.   77
Therefore, we here apply a comprehensive analytical framework to long-term   78
individual-based body mass data for a wild rodent population, which appear to show a   79

mismatch between the observed rate of phenotypic change and the predicted rate of   80

genetic change. To resolve this mismatch, we use information on within-population   81

relatedness and individual-level trait measurements [47, 48] to obtain a statistically   82

robust estimate of the direction and rate of genetic change [14, 49, 50]. Subsequently we   83

disentangle the role of genes and the environment in shaping the covariance between   84

body mass and fitness, and identify the likely target of selection. This allows us to   85

directly compare the observed genetic change to a range of evolutionary predictions, and   86

to thereby resolve the stasis paradox and provide a deeper understanding of selection   87

and evolution in this system.   88

Results and discussion   89

Based on ten years of data on an alpine population of snow voles [51, 52] (*Chionomys*   90

nivalis, Martin 1842), we find that relatively heavy individuals both survive better   91

($p = 0.04$) and produce more offspring per year ($p = 0.003$). Assuming causality, this   92

generates strong phenotypic selection favouring heavier individuals (selection differential   93

$S = 0.86$ g, $p < 10^{-5}$; mean adult mass= 41.7 g, standard deviation=5.2 g). In line   94

with other morphological traits [12, 13], variation in body mass has a significant additive   95

genetic component (V_A=4.34 g^2, 95%CI [2.40;7.36]), which corresponds to a heritability   96

(h^2) of 0.21 (95%CI [0.11;0.29]). Similarly, we find significant additive genetic variance   97

in fitness (V_A=0.10; 95%CI [0.06;0.19], $h^2 = 0.06$ 95%CI [0.04;0.12]), measured as   98

relative lifetime reproductive success (rLRS).   99

Given these estimates of selection (S) and heritability (h^2), and after normalizing by   100

a mean generation time of 1.2 years, the breeder's equation ($R = h^2S$) predicts an   101

adaptive evolutionary response (R) in body mass [14, 48], i.e. an increase in the mean   102

breeding value for body mass over time, of 0.17 g/year (95%CI [0.07;0.28]; Fig 2A UBE,   103

Univariate Breeder's Equation). However, over the past nine years (approximately eight   104

generations), the change in mean body mass is not significant and small at best (0.08   105

g/y; 95%CI [-0.02;0.18]; p=0.14) after correcting for changes in demographic structure   106

(i.e. accounting for sex and age effects, see Fig 1A and S1). This apparent mismatch   107

between the predicted increase in body mass based on the breeder's equation and the   108

absence of a strong and statistically significant phenotypic change would appear to   109

provide yet another example of the stasis paradox [9].   110

To test whether the predicted positive genetic trend, i.e. an increase in breeding   111
values, is being masked by the effects of phenotypic plasticity [9, 35], we directly   112
estimated the additive genetic covariance between mass and fitness. Based on the   113
Robertson-Price's equation, this provides an unbiased estimate of the rate of genetic   114
change per generation [14, 53, 54]. Contrary to the predicted evolutionary increase in   115
body mass, this estimate of genetic change in mass is strongly negative and highly   116
significant ($p_{\text{MCMC}} < 0.001$; Fig 2A GCPE, Genetic Change Price Equation). When   117
normalized by a mean generation time of 1.2 years, this provides a rate of evolutionary   118
change of -0.29 g/year (95%CI [-0.55; -0.07]) or approximately 7,300 Darwins (the   119
natural logarithm of the proportional change in trait value over a million years [55]),   120
which is in line with other estimated rates of "micro-evolution" (e.g. between 3,700 and   121
45,000 Darwins in the Trinidadian guppies [56]). Importantly, this rate of evolution is   122
unlikely to have happened solely through genetic drift ($p_{\text{MCMC}} < 0.001$; Fig S2 and 123
S3) [50], and therefore most likely reflects a response to selection favouring genetically 124
lighter individuals.   125

The above result was confirmed by an independent estimate of the rate of evolution   126
using best linear unbiased predictors (BLUPs) of breeding values for mass: Taking into   127
account the non-independence of BLUPs and sampling variance [49, 50], we find that   128
predicted breeding values have likely declined over the past nine years (-0.07 g/year,   129
95%CI $[-0.16; 0.01]$, p_{MCMC}=0.06; Fig 1B & Fig 2A TPBV, Trend in Predicted   130
Breeding Values), and this despite the BLUPs approach being potentially biased   131
towards the phenotypic trend [49] (i.e. in this case towards zero). This negative trend,   132
combined with the fact that the phenotypic mean has either remained constant or has   133
shown a slight increase (see above), implies that the plastic component of body mass   134
must have increased. Although the cause of this increase remains unknown, population   135
size has declined over the study period (Fig S1), which may have resulted in an increase   136
in the per-capita resource availability (i.e. density dependence). Alternatively, the   137
absolute food availability or quality may have improved. Interestingly, although these   138
environmental changes may be coincidental, they may also be a direct result of a change   139
in the selection regime or the evolutionary change toward smaller size [35, 57].   140

As the phenotypic selection differential ($\sigma_P(m, \omega)$), i.e. the phenotypic covariance 141

between body mass m and fitness ω, measured as relative lifetime reproductive success (rLRS)) is equal to the sum of the additive genetic and environmental covariances between mass and fitness ($\sigma_A(m,\omega)$ and $\sigma_E(m,\omega)$, respectively [14, 53, 54, 58, 59], it follows that because $\sigma_P(m,\omega)$ is positive and $\sigma_A(m,\omega)$ is negative, the environmental covariance must be large and positive (Fig 2B LRS). In other words, while environmental conditions that make voles heavy (for instance abundance of food or lack of parasites) also make them successful at reproducing and surviving, there is no causal *positive* relationship between breeding values for mass and fitness. It is this difference in sign between $\sigma_A(m,\omega)$ and $\sigma_E(m,\omega)$ that represents an extreme violation of the assumption of the breeder's equation that $\sigma_A(m,\omega)/V_A = \sigma_E(m,\omega)/V_E$ [44]. Hence, our initial prediction of evolution based on phenotypic estimates of selection was wrong, illustrating how these may provide severely biased predictions of the evolutionary trajectories of wild populations. But *why* is evolution taking place in a direction that is opposite to apparent phenotypic selection?

Indirect selection may be acting on body mass through one or more traits genetically correlated to mass [36, 38]: a positive selective pressure on a negatively correlated trait (or a negative selective pressure on a positively correlated trait) would indirectly select for lower mass. However, the genetic correlations among the three morphological traits for which we have data—body mass (m), body length (b) and tail length (t)—are all positive (estimates and 95%CI: $\rho_{m,b} = 0.79$ [0.06; 0.93]; $\rho_{m,t} = 0.40$ [0.01; 0.66]; $\rho_{t,b} = 0.56$ [−0.04; 0.85]), and direct selection is positive for two of them and only slightly negative for one of them (see table S1). Hence, the predicted response based on the multivariate breeder's equation (Fig 2A MBE, Multivariate Breeder Equation) is very similar to that based on its univariate counterpart (Fig 2A UBE), as well as to that based on a multivariate breeder's equation constraining the correlations to zero (Fig 2A $\text{MBE}_{\rho=0}$). Therefore, the genetic correlations among the traits considered do not constrain the evolution of body mass and do not explain the trend towards lower body mass. To test whether parent-offspring conflict between size and fertility might be constraining the evolution of size, we furthermore estimated the genetic correlation between juvenile size and adult annual reproductive success, which should be negative for it to provide a constraint [43]. Although we were not able to incorporate all sources of uncertainty into the estimation of this correlation (see Methods), our best estimate

was 0.21, 95%CI $[-0.24; 0.74]$), which argues against a major role for a trade-off between fertility and offspring size in driving the observed evolution toward smaller sizes.

To pursue the possibility that the counter-intuitive direction of evolution was due to selective pressures directly acting on body mass, we first tested which fitness component is negatively associated with genes for being heavy. Computing sex- and age-specific genetic covariances between mass and fitness components revealed that whereas the genetic covariances between mass and both relative annual reproductive success and adult survival are close to zero in both sexes (Fig 2B), the genetic covariance between mass and over-winter survival is negative in juveniles (-0.98 [-2.44;-0.18] on a logit scale, p_{MCMC}=0.01). Because the genetic correlation between juvenile and adult mass is positive ($r_A = 0.88$; 95%CI [0.39;1]) and significantly different from 0 (p=0.004) but not 1 (p=0.35), selection on juvenile mass can shape genetic variance for mass at all ages, and thereby contribute to the observed negative genetic change [60]. While this shows that negative viability selection of juvenile mass is responsible for the genetic change toward smaller individuals, how come survival is higher for heavier phenotypes *and* lighter genotypes?

Juvenile mass covaries positively with both within- and between-year survival ($p = 0.009$ and $p = 1.3 \times 10^{-6}$, respectively). However, juveniles can only be captured when they first leave their burrow, at an age of approximately three weeks [61], when they weigh 12 to 20 g. They may however continue to be captured until the end of the season, when they can reach weights of up to 50 g. Because of growth, mass measurements are therefore not directly comparable among juveniles of different ages. Indeed, at least part of the positive phenotypic relationship between juvenile mass and survival probability is likely to be mediated by the fact that both increase with age [62]. In addition, viability selection introduces non-random missing data, which further biases estimates of viability selection on mass [62, 63]. Therefore, the positive phenotypic association between juvenile body mass and survival is unlikely to be the result of an among-individual covariation of survival probability and mass, and hence provides a poor representation of selection.

The (co)variance decompositions presented above have the advantage that they do not make causative statements. For example, a genetic covariance describing the rate of evolution has a self-contained, tautological, meaning and does not make any assumptions

with respect to its causes [64]. However, if we are to identify the target of juvenile ²⁰⁶ viability selection, we must adopt a more traditional hypothesis testing framework. This ²⁰⁷ is somewhat complicated by our relatively poor understanding of snow vole biology, ²⁰⁸ much of which takes place below the rocks or the snow, and the impossibility to carry ²⁰⁹ out manipulative experiments in a natural setting. Although this means that the ²¹⁰ scenario detailed below is in part speculative, it is well-supported by our data. ²¹¹

We hypothesised that juveniles with a low *potential adult* mass, i.e. juveniles that ²¹² will grow up to be relatively small adults (*if* they survive), require less time to reach ²¹³ their adult size. Furthermore, when the period favourable for growth is limited, and ²¹⁴ juveniles that have not completed development before the arrival of winter pay a ²¹⁵ survival cost, for example due to trade-offs between growth and vital physiological ²¹⁶ processes [65, 66], survival selection will favour juveniles that need less time to reach ²¹⁷ their adult size. This scenario generates selection for small size, especially for juveniles ²¹⁸ born toward the end of the season (Fig 3A and Fig S4), and gives rise to a negative ²¹⁹ genetic association between mass and survival. On the phenotypic level this negative ²²⁰ selection would however be masked by the positive within-individual age-related ²²¹ association between mass and survival (see above). ²²²

Although, as we emphasised above, inferring a causal relationship between a trait ²²³ and fitness based on their covariance requires great care, we set out to obtain an ²²⁴ estimate of viability selection that is unbiased by growth and non-random missing data ²²⁵ due to mass-dependent mortality occurring after the first capture [62]. To this end we ²²⁶ used a Bayesian model to simultaneously infer birth dates and growth curves for all ²²⁷ juveniles observed at least once. Although we cannot account for viability selection ²²⁸ acting before the first capture, this model enabled us to quantify viability selection on ²²⁹ age-corrected juvenile mass—i.e. asymptotic or predicted adult mass—, and thereby ²³⁰ compare all individuals at the same developmental stage (adulthood), irrespective of ²³¹ their fate. Inferred birth dates revealed that snow fallen during the preceding winter is a ²³² major ecological factor constraining the onset of reproduction in spring, with ²³³ reproduction starting on average 40 days after the snow has melted (SE 4.5, ²³⁴ $p = 4 \times 10^{-5}$) (Fig 3B). As a consequence, juveniles only have a limited amount of time ²³⁵ to grow and reach their adult mass before the return of winter. As growth rate and ²³⁶ predicted adult mass are only weakly (and negatively) correlated (correlation −0.077; ²³⁷

95%CI [−0.150; −0.002]), juveniles with a smaller adult mass on average require less ₂₃₈ time to complete development. The strength of survival selection acting on predicted ₂₃₉ adult mass was slightly negative when averaged over all years and the complete ₂₄₀ reproductive season (p_{MCMC}=0.13), but interacted strongly and significantly with the ₂₄₁ number of days between birth and the first snowfall of that year (interaction = 0.0025, ₂₄₂ 95%CI [0.0001; 0.0048] p_{MCMC}=0.008; for other effects see table S2). This implies that ₂₄₃ individuals born closer to the first snowfall are more strongly selected for a low adult ₂₄₄ mass, and that the length of the snow-free period in a given year determines the total ₂₄₅ selection experienced by the population in that year. ₂₄₆

At our field site, the length of the snow-free period in the years 2008 to 2014 has ₂₄₇ been significantly shorter than during the preceding seven years (Fig 3C). The latter ₂₄₈ coincides with a period of exceptionally high snowfall, low temperatures and a long ₂₄₉ duration of snow cover, across the Swiss Alps [67]. Our model estimates that in 2006 ₂₅₀ and 2007, when the snow-free period was long, most juveniles reached their adult mass ₂₅₁ before the first snowfall, and there was hence no selection on predicted adult mass ₂₅₂ ($\beta = -0.002$, SE= 0.0006, p_{MCMC}=0.47, Fig 3D; D; S4). However, in all subsequent ₂₅₃ years, the snow-free period was much shorter, and there was selection for a lower ₂₅₄ predicted adult mass ($\beta = -0.10$, SE=0.0008, p_{MCMC}=0.009). This suggests that the ₂₅₅ shortening of the snow-free season, and thereby selection for lower predicted adult mass, ₂₅₆ is a novel phenomenon that the population is currently in the process of adapting to. ₂₅₇ Assuming that the additive genetic variation in potential adult mass is similar to the ₂₅₈ additive genetic variation in mass (4.34 g^2, see above), the breeder's equation ₂₅₉ ($R = \beta V_A$) predicts a response to selection on potential adult mass between −0.72 and ₂₆₀ −0.24 g/year (from 2008 and 2014) which is in line with the observed rate of body mass ₂₆₁ evolution (−0.29 g/year [−0.55; −0.07] from 2006 to 2014). ₂₆₂

Model complexity and data availability prohibit rigorously disentangling genetic and ₂₆₃ environmental sources of variation in predicted adult mass among individuals and over ₂₆₄ time. Therefore we cannot rule out the possibility that the selective pressure we ₂₆₅ identified is not causative [68], nor can we rule out other selective forces contributing to ₂₆₆ the observed evolution. Nevertheless, the hypothesis presented here is consistent with ₂₆₇ observations: The cohort born in 2013 had an estimated adult mass that was 1.02 g ₂₆₈ smaller than the cohort born in 2006 (p=0.05). This decline in mass is predicted to ₂₆₉

have increased population-level juvenile survival by 2.5%, and may have contributed   270
positively to population recovery (Fig S1).   271

Conclusion   272

We have exploited a case of apparent evolutionary stasis to gain a deeper insight into   273
the evolutionary dynamics of natural populations, and the selective pressures that shape   274
them. Whereas estimates of selection and evolution based on phenotypic data alone can   275
easily mislead our understanding of the selective and evolutionary processes in natural   276
populations, a quantitative genetic framework applied to individual-based long-term   277
data allows us to unravel evolutionary and environmental changes over time, and to   278
obtain unbiased estimates of selection. This has resolved a case of apparent evolutionary   279
stasis, providing a comprehensive empirical demonstration of contemporary adaptive   280
evolution in response to a climatic fluctuation.   281

Methods   282

Snow vole monitoring.   Monitoring of the snow vole population began in 2006 and   283
the present work uses data collected until the fall of 2014. The snow vole monitoring   284
was authorised by the *Amt für Lebensmittelsicherheit und Tiergesundheit*, Chur,   285
Switzerland. The study site is located at around 2030m above sea level, in the central   286
eastern Alps near Churwalden, Switzerland (46°48' N, 9°34' E). The site consists of   287
scree, interspersed with patches of alpine meadows and surrounded by habitat   288
unsuitable for snow voles: a spruce forest to the West, a cliff to the East and large   289
meadows to the North and South. In accordance with it being considered a   290
rock-dwelling specialist [61], at our study site snow voles were almost never captured   291
outside of the rocky area. Given that it is ecologically fairly isolated, we were able to   292
monitor the whole population. Trapping throughout the whole study area took place   293
during the snow-free period, between late May and mid-October. One trapping session   294
consisted of four trapping nights. Analyses presented here are based on a total of two (in   295
one year), three (in three years) or five (in five years) trapping sessions per season. All   296
newly-captured individuals weighing more than 14 g were marked with a subcutaneous   297

passive transponder (PIT, ISO transponder, Tierchip Dasmann, Tecklenburg).   298

Additionally, an ear tissue sample was taken (maximum 2 mm in diameter) using a   299

thumb type punch (Harvard Apparatus) and stored in 100% ethanol at $-20°$C. DNA   300

extracted from these samples was genotyped for 18 autosomal microsatellites developed   301

for this population [69], as well as for the *Sry* locus to confirm the sex of all individuals.   302

Finally, another Y-linked marker as well as a mitochondrial marker was used check for   303

errors in the inferred pedigree (see below). An identity analysis in CERVUS v.3.0 [70]   304

allowed us to identify animals sampled multiply, either because they lost their PIT, or   305

because at their first capture as a juvenile they were too small to receive a PIT. Over   306

the study period we obtained 3382 captures of 937 individuals (see Fig SI5). All the   307

analyses were carried out in R [71]. Specific packages are referenced below.   308

Pedigree inference.   Parentage was inferred by simultaneously reconstructing   309

paternity, maternity and sibship using a maximum likelihood model in   310

MasterBayes [72]. Parentage was assigned using a parental pool of all adults present in   311

the examined year and the previous year, assuming polygamy and a uniform genotyping   312

error rate of 0.5% for all 18 loci. As it is known that in rare cases females reach sexual   313

maturity in their year of birth [61], we matched the genotypes of all individuals against   314

the genotypes that can be produced by all possible pairs of males and females. We   315

retrieved the combinations having two or less mismatches (out of 18 loci) and ensured   316

that parental links were not circular and were temporally consistent. This way, we   317

identified eight young females as mothers of animals born in the same year, with a   318

known father but a mother not previously identified. All of these females were relatively   319

heavy ($>$33 g) at the end of the season and their home-ranges matched those of their   320

putative offspring. Finally, the pedigree was checked using a polymorphic Y-linked locus   321

developed for this population [73], as well as a fragment of the mitochondrial DNA   322

control region, amplified using vole specific primers [74]. There were no inconsistencies   323

between the transmission of these three markers and the reconstructed pedigree.   324

Although the study periods spans over 7.5 times the mean generation time, the final   325

pedigree had a maximum depth of 11 generations (one for each of the nine years of   326

monitoring, plus one for the unobserved parents of siblings that could be inferred in the   327

first year, plus one due to the reproduction of a few females in the same year they were   328

born) and a mean depth of 3.8 generations. The pedigree consists of 987 individuals 329

(more than the number of captured individuals because the pedigree contained the 330

unobserved parents of some full-siblings that could be reliably inferred), 458 full-sibling 331

pairs, 3010 half-sibling pairs, 764 known maternities and 776 known paternities, so that, 332

excluding the base population, 86% of the total parental links were recovered. 333

Traits. The recapture probability from one trapping session to the next was 334

estimated to be 0.924 (SE 0.012) for adults and 0.814 (SE 0.030) for juveniles using 335

mark-recapture models. Thus, with three trapping session a year, the probability not to 336

trap an individual present in a given year is below 10^{-3}. Not surprisingly, no animal 337

was captured in year y, not captured in $y+1$, but captured or found to be a parent of a 338

juvenile in $y+2$ or later. Therefore, capture data almost perfectly matches over-winter 339

survival. However, as is almost always the case in these type of studies, we are unable to 340

separate death from permanent emigration. Importantly however, as both have the same 341

consequences on the population level, this does not affect our evolutionary predictions. 342

Annual and lifetime reproductive success (ARS and LRS, respectively) were defined 343

as the number of offspring attributed to an individual in the pedigree, either over a 344

specific year or over its lifetime. 56 individuals born of local parents were not captured 345

in their first year, but only as adult during the next summer, probably because they 346

were born late in the season and we had only few opportunities to catch them. This 347

means that we miss a fraction of the juveniles that are not observed in their first year 348

and die, or emigrate, during the following winter. We acknowledge that this means that 349

our measures of ARS and LRS partly conflate adult reproductive success and the 350

viability of those juveniles that were never observed, but our measures are the most 351

complete measures of reproductive success available in this system. 352

In order to estimate total selection, we used relative LRS (ω) as proxy for 353

fitness [36], where $\omega_i = \frac{\text{LRS}_i}{\frac{1}{N_{s,t}}\sum_{j=1}^{N_{s,t}}\text{LRS}_{j,t}}$. Here, $N_{s,t}$ is the number of individuals of 354

same sex as the focal individual i, present in the cohort t, so that $\frac{1}{N_{s,t}}\sum_{j=1}^{N_{s,t}}\text{LRS}_{j,t}$ is 355

the sex-specific, cohort-specific mean of LRS. The latter is required as the mean LRS 356

differs between males and females due to imperfect sampling [14]. We used 357

cohort-specific means in order to account for variations in population growth rate. In 358

addition, to estimate the viability and fertility component of total selection, we used 359

ARS and survival, standardized by their sex- and year-specific means.   360

Generation time was defined as the mean age of parents at birth of their   361
offspring [75] and used to convert the predictions and estimations of evolution into   362
grams per year.   363

Mass (m) was measured to the nearest gram with a spring scale. Both body length   364
(b), measured from the tip of the nose to the base of the tail, and tail length, measured   365
from the tip to the base of the tail (c), were measured to the closest mm with a calliper   366
while holding the animal by the tail.   367

Selection.   Selection differentials were estimated using bivariate linear mixed models,   368
as the individual-level covariance between fitness (i.e. relative LRS for total selection,   369
relative ARS for fertility selection and relative survival for viability selection) and mass   370
(corrected for sex, age and cohort). However, while this provides the best estimate of the   371
within-generation change in trait mean due to selection [36], because the distribution of   372
fitness is not Gaussian, the credibility intervals produced by these models are not exact.   373
Hence, the statistical significance of selection was tested using a univariate   374
over-dispersed Poisson generalized linear mixed model (GLMM) in which LRS was   375
modelled as a function of individual standardized mass and including sex and age as   376
covariates and cohort as a random effect. Note that the latter estimates the effect of   377
mass on a transformed scale, and therefore cannot be directly used to quantify an effect   378
of selection on the original scale measured in grams [76]. The significance based on the   379
basis of the GLMM was confirmed by non-parametric bootstrapping. Similarly, we   380
tested for the significance of selection through ARS only, using an over-dispersed Poisson   381
GLMM including sex as a fixed effect, and year and individual as random effects.   382

The estimation of survival selection is facilitated by the fact that the year-to-year   383
individual recapture probability is effectively 1. Therefore, selection on year-to-year   384
survival was tested for by a binomial GLMM. This model included sex, age and their   385
interaction as fixed effects, and year as a random effect.   386

In order to integrate the uncertainty in the estimation of selection with the   387
uncertainty in the estimation of heritability when predicting the rate of evolution,   388
selection differentials and gradients were also obtained from the multivariate animal   389
model presented below.   390

Quantitative genetic analyses.   We used uni- and multivariate animal models to   391
estimate additive genetic variances, covariances and breeding values [48, 77, 78] with   392
`MCMCglmm` [79]. All estimations were carried out in a Bayesian framework in order to   393
propagate uncertainty when computing composite statistics such as heritabilities and   394
rates of genetic change [58]. All estimates provided in the text are posterior modes and   395
credibility intervals are highest probability density intervals at the 95% level. All the   396
animal models were run for 1,300,000 iterations with a burnin of 300,000 and a thinning   397
of 1,000, so that the autocorrelations of each parameter chain was less than 0.1.   398
Convergence was checked graphically and by running each model twice.   399

Univariate models: We first carried out univariate model selection, fitting models   400
without an additive genetic effect, to determine which fixed and random effects to   401
include. Based on AICc [80], and fitting the models by Maximum of Likelihood in   402
`lme4` [81], we obtained a model that predicts the mass $m_{i,t}$ of individual i at time t by: 403
age, as a factor (juvenile or adult); sex as a factor; the interaction between age and sex;   404
Julian dates and squared Julian dates (in order to correct for population-level seasonal   405
variation in mass: individuals of all sexes and ages tend to be heavier in the middle of   406
summer and lighter in spring and fall, possibly as a result of food abundance), which   407
were centered and divided by their standard deviations in order to facilitate   408
convergence; the interaction between age and Julian date; the interaction between sex   409
and Julian date; the three way interaction between age, sex and Julian dates; a random   410
intercept for individual, i.e. permanent environment; and a random intercept for year.   411
The inclusion of year accounts for non-independence of observation within years, while   412
the permanent environment random effect accounted for the non-independence of   413
repeated measurements made on the same individual [82].   414

We then fitted an animal model by adding a random intercept modelling variance   415
associated with mother identity [78], and a random intercept modelling additive genetic   416
variance. Although it was not included in the best models, we kept inbreeding   417
coefficient (estimated from the pedigree) as a covariate, because leaving it out could   418
bias the later estimation of additive genetic variation [83]. Nevertheless, animal models   419
fitted without this covariate gave indistinguishable estimates. The univariate animal   420
model for body mass can be written as: 421

$$m \sim bX + D_1 a + D_2 \mu + D_3 p + D_4 y + Ir. \tag{1}$$

Here X, D_1, D_2, D_3 and D_4 are design matrices relating observations to the   422

parameters to estimate, b is a matrix of fixed effects, a, μ, p and y are random effects   423

accounting for the variance associated with breeding value, mother, permanent   424

environment (i.e. individual repeatability) and year, respectively.   425

The most important aspect of this model is that a, the matrix of breeding values,   426

follows a multivariate normal distribution: 427

$$a \sim MVN \left(0, A \times \sigma_A^2(m)\right) \tag{2}$$

where A is the relatedness matrix describing the relatedness among all individuals, and   428

$\sigma_A^2(m)$ is the additive genetic variance in body mass. For univariate animal models for   429

each random effect we used a vague proper prior with the variance parameter set to 1   430

and the degree of belief set to 0.002.   431

Multivariate models: Univariate animal models can be expanded to multivariate   432

models in order to estimate genetic correlations, genetic gradients and genetic   433

differentials between body mass (m), body length (l), tail length (t) and fitness (ω).   434

$$[m, l, t, \omega] \sim bX + D_1 a + D_2 \mu + D_3 p + D_4 y + Ir. \tag{3}$$

The fixed part of the model matches that used for each trait in univariate models. We   435

used parameter-expanded priors for the variance components in order to facilitate   436

convergence of parameters that were close to zero [79]. The working parameter prior   437

was normally distributed, with a mean of 0 and a variance of 1000. For ω, the residual   438

variance was set to zero in the model, so that the residual variance in fitness is   439

estimated by the permanent environment random effect. Although fitness is measured   440

only once per individual, this allows the estimation of the covariance between fitness   441

and the non-additive genetic repeatable component of trait variation, that the   442

environmental component of selection [14]. We assumed a Gaussian distribution for 443
fitness so that covariances between traits and fitness could be directly interpreted as 444
selection differentials [76]. 445

The matrix of breeding values ($\boldsymbol{a}$) here follows a multivariate normal distribution: 446

$$\boldsymbol{a} \sim MVN\left(\boldsymbol{0}, \boldsymbol{A} \otimes \boldsymbol{G}\right) \qquad (4)$$

where $\boldsymbol{A}$ is the relatedness matrix describing the relatedness among all individuals, and 447
$\boldsymbol{G}$ is the G-matrix, containing the additive genetic variances and covariances among all 448
traits. 449

$$\boldsymbol{G} = \begin{pmatrix} \sigma_A^2(m) & \sigma_A(m,l) & \sigma_A(m,t) & \sigma_A(m,\omega) \\ \sigma_A(m,l) & \sigma_A^2(l) & \sigma_A(l,t) & \sigma_A(l,\omega) \\ \sigma_A(m,t) & \sigma_A(l,t) & \sigma_A^2(t) & \sigma_A(t,\omega) \\ \sigma_A(m,\omega) & \sigma_A(l,\omega) & \sigma_A(t,\omega) & \sigma_A^2(\omega) \end{pmatrix}. \qquad (5)$$

For any trait z, $\sigma_A(z,\omega)$ is the genetic differential, that is, the predicted rate of 450
evolutionary change according to Robertson's secondary theorem of natural selection, or 451
Price equation applied to genetic variation [14,53,54,58]. The Price equation is generally 452
presented as a prediction of evolutionary change over the next generation, but it has also 453
been used as a description of change [64,84,85]. We use this prediction retrospectively, 454
as an estimation of the mean evolutionary change that has occurred during the study 455
period, which makes the assumption that ω is a good measure of fitness, because when 456
"real fitness" is used, the equation is a mathematical tautology, i.e. it is exact [64]. A 457
deviation from this perfect fitness measure could come from random Mendelian 458
segregation or systematic meiosis distortion. Our results were robust to the use of an 459
annualized measure of fitness (annual reproductive success plus twice survival), and to 460
standardizing fitness across all individuals, within years, within cohorts, or within sexes. 461

For two traits z and y, the genetic correlation is $\frac{\sigma_A(z,y)}{\sigma_A(z)\sigma_A(y)}$. The vector of selection 462
differentials on the three traits ($\boldsymbol{S}$) was estimated as the sum of the vectors of 463
covariances between traits and ω in the variance-covariance matrices for $\boldsymbol{a}$, $\boldsymbol{p}$, $\boldsymbol{m}$ and $\boldsymbol{r}$; 464
which was equivalent to the selection differential computed in the paragraph on 465
selection above. Let $\boldsymbol{G'}$ be a subset of $\boldsymbol{G}$ excluding the column and the row that 466

contain ω. The vector of selection gradients on the three traits ($\boldsymbol{\beta}$) was estimated as $(\boldsymbol{G'} + \boldsymbol{P'} + \boldsymbol{R'})^{-1}\boldsymbol{S}$, where $\boldsymbol{P'}$ and $\boldsymbol{R'}$ are the equivalent of $\boldsymbol{G'}$ for permanent environment effects and for residuals, respectively [38, 58]. The prediction of the multivariate breeder's equation is obtained by $\Delta\overline{\boldsymbol{Z'}} = \boldsymbol{G'}\boldsymbol{\beta}$. In order to visualise the effect of the genetic correlations on the predictions of evolution, we also applied a multivariate breeder's equation that assumes the genetic correlations to be zero. To this end we multiplied the $\boldsymbol{G'}$ matrix by the identity matrix [38]: $\Delta\overline{\boldsymbol{Z'}} = (\boldsymbol{G'} \times \boldsymbol{I})\boldsymbol{\beta}$. We excluded the among-year level covariance from the selection differential and gradients, because (i) covariation between mass and fitness at the level of year does not correspond to selection as it does not occur among individuals, and (ii) due to the standardization of relative fitness at the level of cohorts, the among year variance and covariances involving ω were effectively zero ($\sigma_Y^2(\omega) < 10^{-8}$).

To investigate the potential role of parent-offspring conflict, we estimated the genetic correlation between parental ARS and offspring mass using a bivariate animal model. For juvenile mass, we used predicted adult mass (i.e. age-corrected juvenile mass; see below). We were not able to incorporate the uncertainty in the estimation of predicted adult mass in this model and used the mean of the posterior distribution of each individual. The credibility intervals derived from this model are therefore only indicative. The model included sex as fixed effect, and random additive genetic, individual identify (i.e. permanent environment effects), maternal identity and year effects.

To decompose selection components into environmental and genetic covariances (as presented in Fig 2B) we fitted models like the one described in equations (1-3), but replacing fitness (ω) by the appropriate fitness components: relative ARS or relative survival; and taking the appropriate subset of the data: adult males ($\mathrm{ARS}_{\circlearrowleft}$), adult females ($\mathrm{ARS}_{\circlearrowleft}$), adults ($\phi_{\mathrm{Ad}}$) or juveniles ($\phi_{\mathrm{Juv}}$). To estimate readily interpretable environmental and genetic covariances between body mass and fitness we again assumed a Gaussian distribution of fitness components [76].

Test of genetic correlations: We used `ASReml-R` [86, 87] to test the genetic correlation between mass in adults and in juveniles against 1 and 0, by considering them as two separate traits. We first ran an unconstrained model and then reran it with the genetic correlation parameter set to 0.99 (and not exactly to 1 because `ASReml` cannot

invert matrices with perfect correlations), or 0 respectively. The fit of the unconstrained   498

model was then compared to that of the two constrained models using a likelihood ratio   499

test with one degree of freedom [88].   500

Birth date and growth prediction: Using the Bayesian programming   501

environment JAGS [89], we fitted a multivariate Bayesian model to mass measurements   502

of all 613 juveniles with mass data, and to their overwinter survival. The model   503

simultaneously estimated individual growth curves—that is onset of growth (although   504

this is referred to as "birth date" hereafter, this actually is the projected time when   505

mass was zero, i.e. at conception), individual growth rates and asymptotic masses (i.e.   506

predicted adult mass) of all juveniles—and the effect of predicted adult mass on   507

overwinter survival. The model clustered juveniles from the same mother born in the   508

same year into litters (see e.g. [90] for a similar approach), assuming a maximum of five   509

litters per year and assuming that successive litters are at least 20 days apart [61].   510

Preliminary model selection, assuming no differences in predicted adult masses among   511

individuals, selected a monomolecular growth model (ΔDIC > 80) over Gompertz and   512

logistic models, as defined in [91]. The model accounted for measurement error in mass,   513

assuming that the standard deviation of the errors was that observed in animals   514

measured multiple times on the same day (2.05g). In order to estimate the overall   515

viability selection on predicted adult mass, we performed within the model a logistic   516

regression of year-to-year survival on sex and predicted adult mass. In order to test for   517

the effect of the length of snow free period on the selection on predicted adult mass, we   518

reran the full model including time until the first snow fall and its interaction with   519

predicted adult mass in the logistic regression. We use the estimates of these two   520

models to predict the survival probability as a function of predicted adult mass for   521

every year, or for groups of years, depending on the distribution of birth dates and on   522

the timing of the first snow fall.   523

Three MCMC chains were run for 6,300,000 iterations, with a burnin of 300,000 and   524

a thinning of 6,000. Convergence was assessed by visual examination of the traces, and   525

by checking that $\hat{R} < 1.01$. Convergence was not achieved for the litter affiliations of 25   526

individuals as well as for one predicted adult mass, thus introducing a bit more   527

uncertainty in the estimations. The fit of the model was assessed using posterior   528

predictive checks on the predictions of individual masses (p=0.46) and survival 529
probabilities (p=0.49). The `JAGS` code for this model can be found at 530
https://github.com/timotheenivalis/SelRepSel. 531

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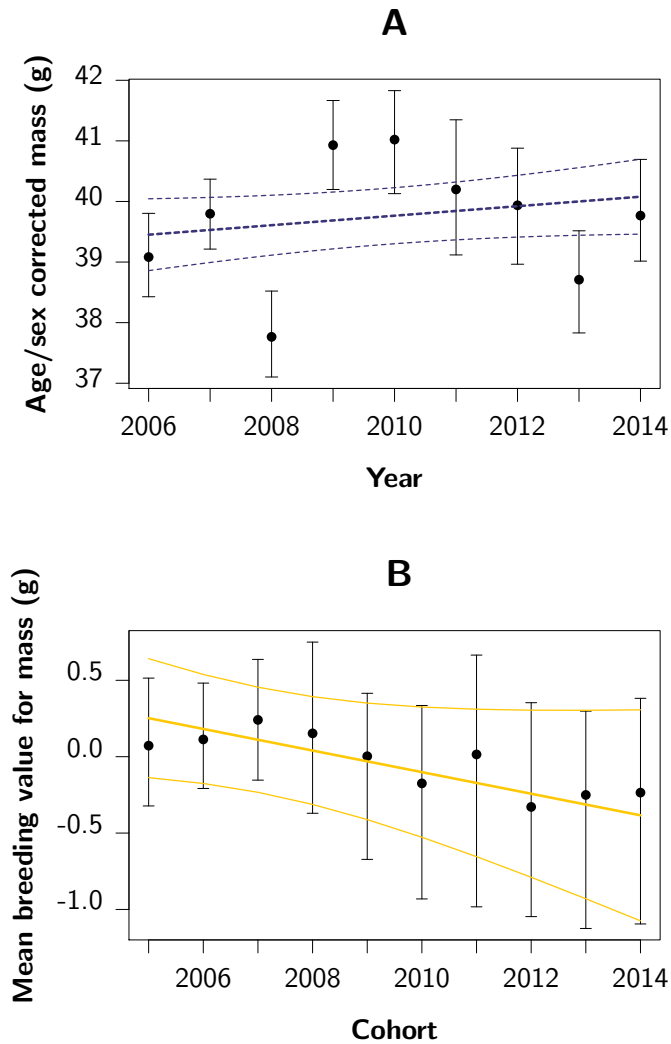   model. Oecologia. 2012 May;169(1):143–53.   763

A

B

Figure 1 Temporal variation in mass and estimated breeding values for mass. (**A**): Year-specific mean mass corrected for age, sex and date of measurement, with 95%CI. (**B**): Cohort-specific mean estimated breeding value for mass with their 95%CI and the trend in breeding value with 95%CI. Note the different scaling of the y-axes in **A** and **B**.
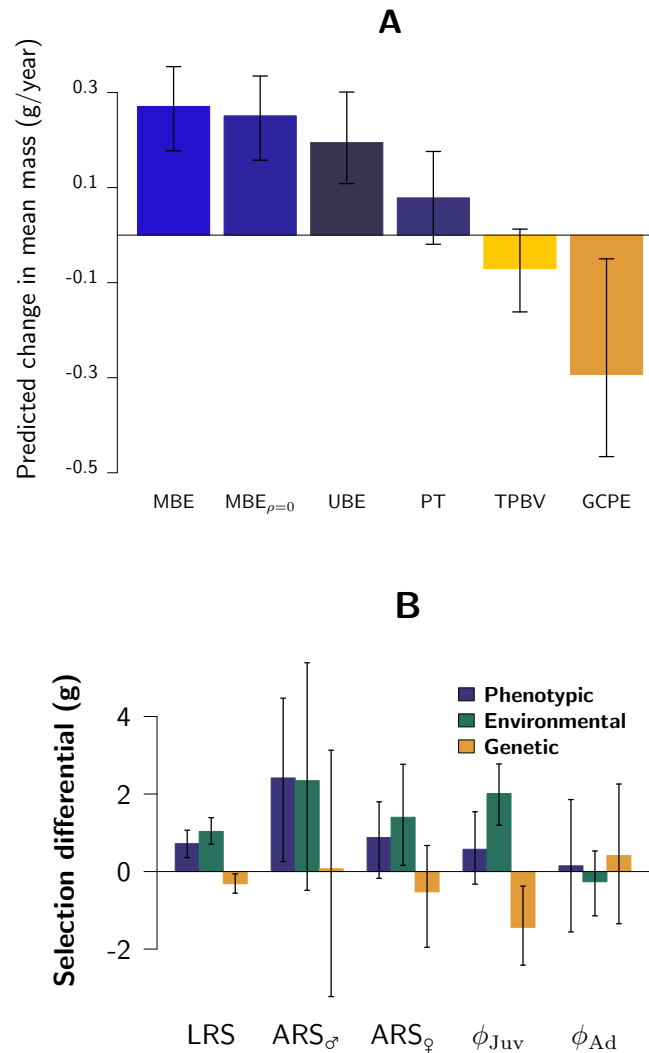
Figure 2 Predicted and observed rates of evolutionary change. (**A**): Rates of evolutionary change predicted by (from left to right) the breeder's equation in its multivariate form (MBE), the multivariate breeder's equation while constraining the genetic correlations to zero (MBE$_{\rho=0}$), and the univariate breeder's equation (UBE), followed by the phenotypic trend (PT), the trend in predicted breeding values (TPBV) and the genetic change estimated by the Price equation (GCPE). (**B**): Phenotypic, genetic and environmental selection differential for total selection (LRS), fertility selection in males (ARS$_{\male}$) and females (ARS$_{\female}$), viability selection in juveniles (ϕ_{Juv}) and in adults (ϕ_{A}). Both panels show posterior modes, with vertical lines indicating 95%CI.
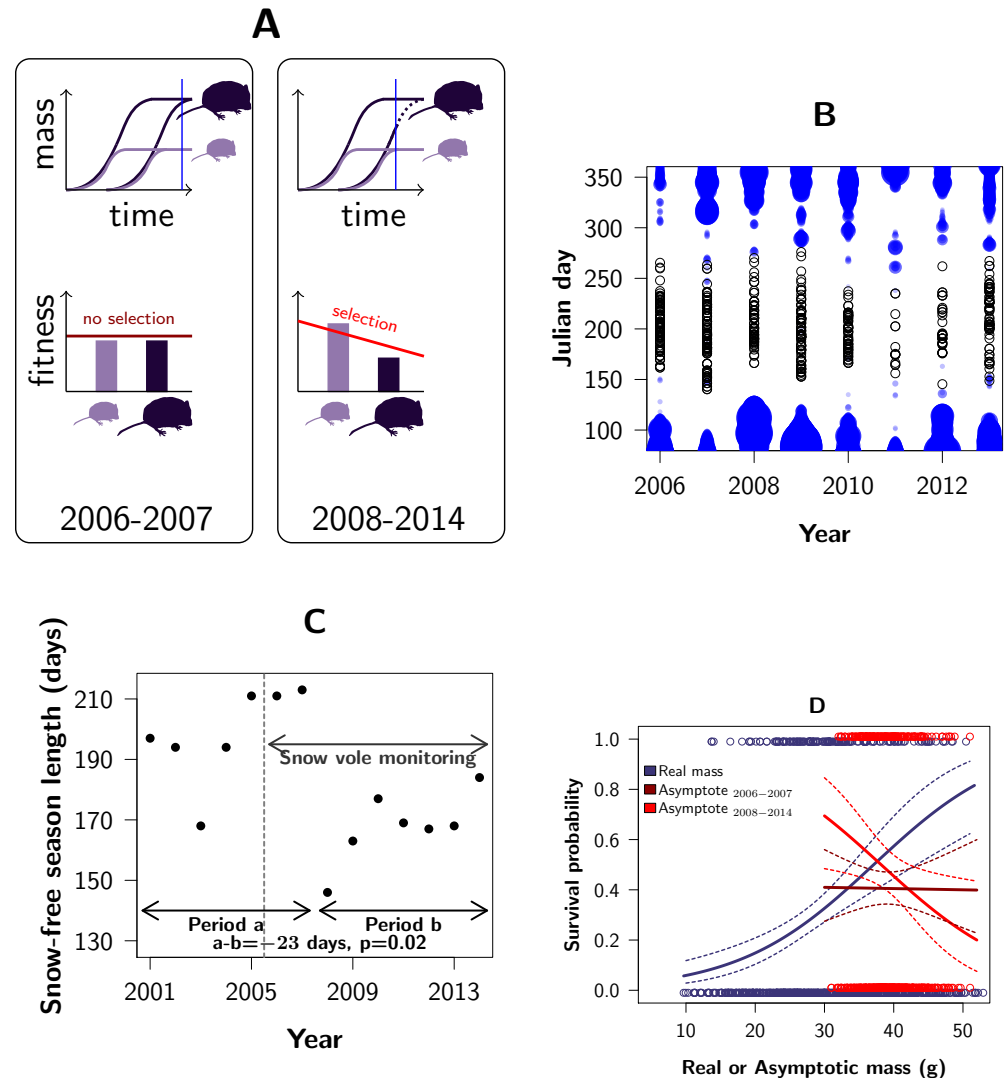
Figure 3 Snow-free season, timing of reproduction and selection for potential adult mass. (**A**) Hypothetical scenario generating selection for lower body mass: In years with short summers (2008-2014), juveniles born late and having a large potential adult mass are still growing at the onset of winter and therefore less likely to survive (blue vertical line). This results in selection for individuals with a low potential adult mass, despite mass covarying positively with survival on a within-individual level due to variation in age. (**B**) Births (black dots) only occur during the snow free season (the depth of the snow cover is indicated by the thickness of the blue dots), (**C**): which in 2008-2014 has been shorter than in the preceding 7 year. Therefore, (**D**) despite a positive phenotypic selection on body mass (blue), predicted adult mass was selectively neutral in 2006-2007 (brown), and was negatively selected in 2008-2014 (red).