

# **Individual-level causes and population-level consequences of variation in fitness in an alpine rodent**

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# Summary

This thesis investigates the stochastic and selective causes of variation in fitness components, and the evolutionary consequences of this variation in a wild rodent population. It shows the contemporary genetic evolution of body mass and decouples classic estimates of selection from adaptive evolution.

The heart of evolutionary biology is understanding the variation in organisms. For over 150 years, researchers have documented the causes of within-species variation and how it contributes to speciation and explains the fit between organisms and their environment. Recently, increasing concerns regarding rapid anthropogenic changes have driven renewed investigation of how wild populations adapt to environmental change. This new focus has revealed the difficulties measuring natural selection, disentangling evolution from plastic changes, and predicting evolutionary trajectories. For instance, there are few robust examples of contemporary evolution in wild populations, casting doubt on the possibility that evolution can rescue populations from rapid environmental change. In this thesis, I investigate the causes of natural selection and evolution in a wild population of snow voles (*Chionomys nivalis*). Thanks to 10 years of intensive individual-based monitoring and genotyping, knowledge of this population includes life-history, morphological data, and a high-resolution pedigree. This population is therefore among the best available worldwide to measure selection and evolution in action.

The population is nevertheless relatively small and recent publications suggest that the evolutionary potential in small populations is effectively cancelled by stochasticity in fitness components. I assess the methods used in those publications and demonstrate that the variation in fitness components is not purely stochastic. Small populations, including these snow voles, show evolutionary potential.

With collaborators, I then compare four common methodological frameworks to disentangle the contributions to phenotypic change of evolution, plasticity, and demography. We identify important discrepancies between the frameworks, partly originating from using different definitions, but also possessing intrinsically different capabilities. Among the considered frameworks only quantitative genetics can measure genetic change.

Applying methods from quantitative genetics to the snow vole population, I demonstrate that body mass evolved adaptively over the study period. I show that phenotypic estimates of selection are not predictive of genetic evolution: neither the mean selection nor its temporal variation are related to the rate of genetic evolution. This demonstrates that the dominant purely-phenotypic method used to measure selection risks measuring variation in nutritional status instead. Nevertheless, I employed quantitative genetics to identify the target of selection and obtain selection estimates in line with the observed genetic change

This thesis establishes contemporary evolution in a wild population and shows that



evolutionary responses to environmental change cannot be reliably estimated nor understood from purely-phenotypic methods; an explicit genetic approach is necessary.



# Zusammenfassung

Diese Doktorarbeit untersucht die stochastischen und selektiven Ursachen der Variation in Fitnesskomponenten und deren evolutionären Konsequenzen in einer freilebenden Nagetierpopulation. Sie zeigt die gegenwärtige, genetische Evolution von Körpermasse und entkoppelt klassische Selektionsschätzungen von adaptiver Evolution.

Das Herzstück der Evolutionsbiologie liegt im Verständnis der Vielfalt von Organismen. Während über 150 Jahren haben Forscher die Ursachen von intraspezifischer Variation dokumentiert, wie sie zur Artbildung beiträgt und zum Zusammenpassen von Organismen mit ihrer Umwelt. Zunehmende Bedenken wegen der schnellen anthropogenischen Veränderungen haben in letzter Zeit eine erneute Erforschung, wie sich freilebende Populationen an Umweltveränderungen anpassen, vorangetrieben. Dieser neue Fokus offenbart die Schwierigkeiten im Messen von natürlicher Selektion, die Entflechtung von Evolution und plastischen Veränderungen und dem Vorhersagen von evolutionären Entwicklungsverläufen. Unter anderem gibt es nur wenige robuste Beispiele von gegenwärtiger Evolution in freilebenden Populationen, die, die Möglichkeit, dass Evolution Populationen bei schnellen Veränderungen der Umweltbedingungen rettet, fraglich erscheinen lässt. In dieser Doktorarbeit erforsche ich die Ursachen natürlicher Selektion und Evolution in einer freilebenden Population von Schneemäusen (*Chionomys nivalis*). Dank 10 Jahren intensivem Individuenbasiertem Monitoring und Genotypisierung, beinhaltet der Erkenntnisstand dieser Population Lebensweise, morphologische Daten und einen hochaufgelösten Stammbaum. Deswegen ist diese Population unter den besten weltweit verfügbaren um Selektion und Evolution in Aktion zu messen.

Trotzdem ist die Population ziemlich klein und neuerliche Publikationen legen nahe, dass das evolutionäre Potential in kleinen Populationen effektiv von Stochastik in Fitnesskomponenten aufgehoben wird. Ich beurteile diese Methoden, die in diesen Publikationen benutzt wurden und demonstriere, dass die Variation in Fitnesskomponenten nicht ausschliesslich stochastisch ist. Kleine Populationen, einschliesslich diese Schneemäuse, zeigen evolutionäres Potential.

Mit Kollaboratoren vergleiche ich dann vier häufig benutzte methodologische Ansätze um die Anteile von Evolution, Plastizität und Demographie an der phänotypischen Veränderung zu entflechten. Wir identifizieren wichtige Unstimmigkeiten zwischen den Ansätzen, die teilweise vom Gebrauch von unterschiedlichen Definitionen, aber auch vom Besitz von intrinsisch unterschiedlichen Fähigkeiten stammen. Unter den in Betracht gezogenen Ansätzen kann nur quantitative Genetik genetische Veränderungen messen.

Durch die Anwendung von quantitativ-genetischen Methoden an der Schneemaus-Population demonstriere ich, dass sich Körpermasse über die Studiendauer adaptiv entwickelt. Ich zeige auf, dass phänotypische Schätzungen von Selektion nicht

genetische Evolution hervorsagen: weder die durchschnittliche Selektion noch die temporale Variation hängen mit der Rate genetischer Evolution zusammen. Das legt dar, dass die dominante, rein phänotypische Methode zur Messung von Selektion stattdessen die Messung von Variation im Ernährungszustand riskiert. Dennoch habe ich quantitative Genetik zur Identifikation von Selektion verwendet und Selektionsschätzungen erhalten, die mit der beobachteten genetischen Veränderung übereinstimmen.

Diese Doktorarbeit weist gegenwärtige Evolution in einer freilebenden Population nach und zeigt dass evolutionäre Reaktionen auf Umweltveränderungen von rein phänotypischen Methoden weder zuverlässig eingeschätzt noch verstanden werden können; ein expliziter, genetischer Ansatz ist notwendig.

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# Chapter 1

## General introduction

*One can't understand everything at once, we can't begin with perfection all at once! In order to reach perfection one must begin by being ignorant of a great deal. And if we understand things too quickly, perhaps we shan't understand them thoroughly.*

— Fyodor Dostoyevsky, *The Idiot* (1868–9)

*Si nous ne trouvons pas des choses agréables, nous trouverons du moins des choses nouvelles. / If we do not find anything very pleasant, at least we shall find something new.*

— Voltaire a.k.a. François-Marie Arouet, *Candide* (1759)

### 1.1 Variation in fitness

#### 1.1.1 The origin of variation in evolutionary biology

The heart of evolutionary questioning is understanding variation among living beings (Lynch and Walsh 1998; Wayne and Miyamoto 2006; Kruuk, Charmantier, and Garant 2014). It is in fact its very starting point. Darwin opens his book *the Origin of Species* with two chapters describing variability in domestic and wild organisms (Darwin 1859). Building on these observations, Darwin then summarizes the evidence showing that variation within species is the fuel generating the astonishing diversity among species, and the striking fit between organisms and their environment.

These great answers immediately opened many more questions about the causes and consequences of variation, some of which remain not fully answered more than 150 years later. In particular, nineteenth century biologists struggled with the sources of variation within species, as is clear in *the Origin of Species* itself: “*Variability is governed by many unknown laws, of which correlated growth is probably the most important. Something, but how much we do not know, may be attributed to the definite action of the conditions of life. Some, perhaps a great, effect may be attributed to the increased use or disuse of parts*” (p. 31 Darwin 1859)<sup>1</sup>. The environment was sometimes thought to play a predominant role, through what would be nowadays called *plastic* effects, and the effect of ageing was acknowledged (Wilkins 2009), but these did not always provide a satisfactory explanation for variation that appears within a population. Further-

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<sup>1</sup> Alternatively, biologists dismissed this within-species variation by considering that species were arbitrary boundaries in a set or continuum of variation. Darwin did not attempt to define species, but by explaining how they originate, he made some definitions indefensible (Wilkins 2009, pp. 129-163).

more, Darwinian arguments build on the observation of this special kind of inherited variation that can appear among siblings of a same litter, clutch, or pod, and that is subsequently transmitted from parent to offspring (Darwin 1859, Chapter 1). The late nineteenth century was utterly ignorant of the sources of inherited variation within species. Only at the beginning of the twentieth century were the laws of inheritance progressively discovered and spread to the scientific community (Dietrich 2006). Four more decades saw these laws formalized into a unified scientific theory to understand variation within populations (R. Fisher 1930), and explained at the molecular level (Oswald, MacLeod, and McCarty 1944; Watson and Crick 1953), thus closing the logical gap in Darwin's argument: Relatives resemble each other because they share similar gene versions on long strands of DNA, a molecule that is copied with high fidelity and transmitted from parent to offspring; There is variation among siblings because of the reshuffling and segregation of parental genes and, on occasions, because DNA mutates.

The understanding of the causes of variation within species and populations has made terrific progresses and now fits elegantly in the broader evolutionary theory (Pigliucci and Müller 2010). Nevertheless, many aspects of the causes of variation are still to be refined or newly explored, especially in natural populations (Kruuk, Charmantier, and Garant 2014). In particular, the relative importance of genes and the environment in the wild remains studied in only a few populations of a few species, taxonomically biased, and concerns a limited set of traits (Lynch and Walsh 1998; Postma 2014). Additional open research questions relate to the consequences of within-species variation. In particular, a lot of attention is paid to how genetic variation translates into adaptive evolution (Brookfield 2016). Any trait that possesses genetic variation can evolve, but it will evolve in an adaptive way only if the trait is subject to selection, be it artificial or natural. Selection occurs when the variation in the trait causes variation in *fitness*. Before we discuss the specificity of the causes and consequences of variation in fitness, we must introduce this difficult concept.

### 1.1.2 Variation in the definition of fitness

There has been a great deal written about the concept of fitness, including multiple conflicting definitions, which “*is hardly surprising as every important scientific concept is difficult to understand from first principles, as for instance the notions of space and time, or energy and force*” (p. 1358 Wagner 2010). I will not solve the question of the definition of fitness here, but I will try to make clear how the word is used in this thesis.

To start with, in the past, there has been some confusion on whether fitness is a realized reproductive outcome or a propensity to reproduce (Brandon and Beatty 1984). It is now rather consensual that the concept of fitness is more useful when it is defined as a propensity, that is, as an expected value that cannot be measured directly because of stochasticity (Brandon and Beatty 1984; Price 1996; Krimbas 2004) and we will follow this consensus. Fitness has been defined at the level of the genetic lineage (e.g. Akçay and Van Cleve 2016), of the individual (e.g. Cam and Monnat 2000), of the genotype (e.g. Steiner and Tuljapurkar 2012), or of the population (e.g. Tienterden 2000). A propensity definition partly dissolves the problem of the level of the definition, since the expected reproductive outcome of a genotype is the same as the

expected reproductive outcome of the individuals bearing this genotype, and the expected reproductive outcome of a population is the sum of the expectation of the reproductive outcome of its individuals. Here, we will consider fitness at the level of individuals, because they are the unit most easily observable and the primary target of natural selection.

More confusion on fitness comes from it being alternatively defined as the asymptotic number of descendants or as the contribution to the next generation (Wade 2006). Since we consider fitness at the level of individuals and because most of the work carried out is based on data covering about ten generations only, it is intuitive to consider fitness as the contribution to the next generation. Besides practical considerations, this choice allows for a clear, and conceptually crucial, distinction between selection, inheritance and evolution, that is blurred in asymptotic definitions (R. Fisher 1930; Arnold and Wade 1984). Indeed, imagine the simplistic case of a genetic locus with two non-recombining haplotypes in a closed population. Assuming there is no balancing selection, one of the haplotypes will eventually go to fixation, its asymptotic fitness is one, and the other one will go extinct, its asymptotic fitness is zero. Fixation might have happened due to genetic drift, due to selection on the initial differences between the haplotypes, or due to selection on new mutations. Observing only the starting point (two haplotypes), and the end point (one haplotype), perfectly describes the long-term evolution, but does not reveal its mechanisms. To understand the respective roles of chance (drift), selection, and inheritance (mutations), one must describe the generation-to-generation changes in haplotype frequencies with respect to the allelic state of both haplotypes.

A slightly contentious point is whether fitness should be defined as an absolute number of offspring (Wade 2006) or a relative one (Rousset 2004), that is, whether “relative fitness” is a meaningful phrase or a tautological one. The relative definition avoids appending *relative* to every occurrence of *fitness*, and seems closer to the interest of evolutionary biologists. Nevertheless, the field massively favours the absolute definition, possibly because it has a concrete and observable meaning. For the sake of consistency I attempted to yield to the convention (possibly with some inconsistencies).

Finally, instead of a measure of reproductive success, relative fitness has recently been defined as the amount of information about the environment that populations accumulate by selection (Frank 2012). The first and secondary theorems of natural selection can be rigorously written in term of gain and loss of bits of information about the environment. This shows that populations gain information by selection, and lose it by imperfect transmission or environmental change. I see great conceptual promises in this view, that brings together an intuitive meaning of the word *fitness* and the scientific field of information theory, with all its powerful tools and concepts. An information interpretation of fitness did not directly influence the work and is not necessary to understand it, but it might enlighten some of the results presented here, and evolutionary biology in general. To sum up, we define the fitness of an individual as its expected number of descendant in the next generation.

### 1.1.3 Causes and consequences of fitness variation in the wild

Why is there variation in individual fitness? This question attracts a lot of research attention, because (i) genetic variation in fitness controls the pace of evolution within a population, and because (ii) an intuitive consequence of evolution is the erosion of genetic variation in fitness, thus making the presence of genetic variation in fitness paradoxical (Jones 1987). In this thesis, we only deal briefly with the second point, the fundamental question of appearance and maintenance of genetic variation in fitness, in chapter 2. On the other hand, all the chapters deal with the proximal sources of variation in fitness, and we will consider these proximal sources from two complementary angles.

First in a descriptive approach, one can decompose variation in fitness, i.e. the opportunity for selection, into components of variation. Apart from genetic variation, variation in fitness can also originate from variation in early-life, micro-environment (Turner 2009), or maternal effects (Wolf and Wade 2009). In addition, when working with wild sexual organisms, individual fitness as we defined it cannot be observed directly. Indeed, individuals are unique and their realized reproductive success does not equal their expected reproductive success. Therefore, researchers have to rely on fitness proxies, often realized reproductive success and survival, that contain a large stochastic component. Additive genetic variation in fitness is also the rate of evolution in fitness and sets the maximal rate of evolution (R. Fisher 1930). A variance decomposition approach is therefore useful to ascertain whether variation in fitness proxies is all stochastic and environmental or whether it hides genetic variation in fitness. In doing so, it determines how much adaptive evolution can be expected to happen within a population.

Second, in a more mechanistic approach, one can investigate what characteristics make some individuals fitter than others, that is what traits are under natural selection<sup>2</sup>. The study of natural selection in the wild took-off with the development of regression-based methods to accurately measure its strength and predict its effects (Lande 1979; Lande and Arnold 1983). Under some assumptions, the genetic change in response to selection on a trait is the product of a selection gradient and of additive genetic variation in that trait (Lush 1937). Therefore, by understanding what traits cause variation in fitness, one can predict what traits should evolve, as well as their direction and speed of evolution.

The study of natural selection and adaptive evolution in the wild is very topical in the context of unprecedented rates of environmental changes induced by human activities (Parmesan 2006). Anthropogenic changes provide the opportunity of natural experiments to evolutionary biologists (Altermatt, Ebert, and Altermatt 2016; Brookfield 2016), but also come with societal concerns and an ever increasing urge to better understand and predict how living things respond to the selective pressures imposed by environmental changes (Mc Carty 2001; Shaw and Shaw 2014). This regain of focus

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<sup>2</sup>In this thesis, unless mentioned otherwise, we consider *sexual selection* as part of *natural selection* and of *selection*. Measuring sexual and natural selection separately, would certainly provide a finer understanding of the mechanisms of selection in the study population, but this was beyond the scope of this thesis. Nevertheless, the question was partly explored by García-Navas, Bonnet, Waldvogel, et al. 2016 and García-Navas, Bonnet, Bonal, et al. 2016.

has highlighted the gaps in the understanding of adaptation in natural populations: it is still challenging to predict, or even understand retrospectively, how natural populations respond to selective pressures (Merilä, Sheldon, and Kruuk 2001; Tafani et al. 2013; Shaw and Shaw 2014; Brookfield 2016).

In order to study the evolutionary potential of wild populations and their response to selective pressure, it is necessary to measure genetic parameters. More specifically, one must determine whether the traits under selection are heritable, whether there is heritable variation in fitness and how what is the rate of genetic change for the traits of interest.

## 1.2 Measuring genetic variation

### 1.2.1 Looking up or down? Two philosophies

How to measure and make sense of genetic variation? For over a century, there have been two main approaches (Liedvogel, Cornwallis, and Sheldon 2012), that can grossly be traced back to the scientific controversy that opposed the Mendelians to the biometricalists (Dietrich 2006), and summarized as “bottom-up” and “top-down”. Bottom-up approaches, embodied by candidate gene and genome wide association studies, start from molecular data to infer the phenotypic effects of individual genetic loci. Top-down approaches, encompassed within quantitative genetics, attempt to decompose phenotypic variation into genetic variation and other sources of variation, based solely on phenotypic data and on some knowledge of the relatedness between individuals (Lynch and Walsh 1998). Some pros and cons of both approaches are nicely illustrated by the confrontation of the quantitative genetics of mass with the genotyping of a candidate gene for mass. The former will be further developed in chapters ?? and 2 and we present it in a minimalist nutshell here: using a quantitative genetics *animal model* (Henderson 1950; Kruuk 2004), we estimated additive genetic variation in body mass and lifetime reproductive success. The candidate gene approach is a side project of this PhD that does not appear in the other chapters, and we take the opportunity to present it below.

### 1.2.2 A candidate gene for body mass: insights and limits

We used a candidate gene approach (Fitzpatrick et al. 2005) to uncover a molecular mechanism underlying variation in body mass. To date, the only candidate gene we fully analysed is an intronic region of the gene *lepr*, which codes for the receptor to leptin. Leptin is a hormone known to regulate fat metabolism, energy expenditure and food intake, including in rodents (Houseknecht et al. 1998).

We found a recessive allele (let call the recessive allele *a*, and the dominant allele *A*) associated with lighter individuals (Fig. 1.1A). Homozygotes *aa* were -2.9 g lighter (95% credibility interval [0.6; 5.1]), that is, 8% lighter than the mean. On average, during their lifetime, these *aa* individuals produced one third less offspring than the *AA* individuals (Fig. 1.1B). This strong difference in fitness is however not statistically significant. These results suggest that some of the genetic variation in body mass is due

to food intake and/or fat metabolism, which could not be sensed from the estimation of genetic variances and covariances. Based on such a strong phenotypic effect, *lepr* could be called a major locus, but how much of the genetic variation does it explain?

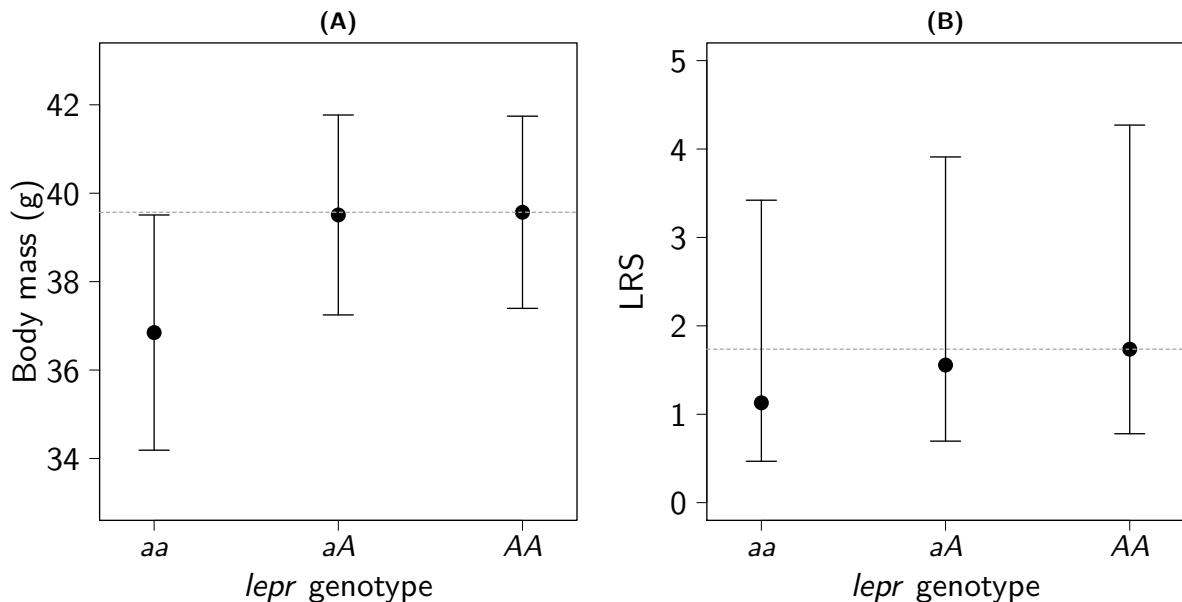


Figure 1.1: Body mass and lifetime reproductive success (LRS) as a function of *lepr* genotypes. (A) Expected body mass of snow voles bearing the three *lepr* genotypes. The expectations and 95% confidence intervals were predicted from a linear mixed model fitted to the 2311 mass measurement of 532 snow voles. The model accounted for sex, age, date of capture and their two-ways interactions, as well as year of capture and multiple measurements of the same individual. (B) Expected LRS of snow voles bearing the three *lepr* genotypes. The expectations and 95% confidence intervals were predicted from a Poisson generalized linear mixed model fitted to the LRS of 611 snow voles. The model accounted for inbreeding coefficient, year of birth and over-dispersion (using an observation-level random effect). For both panels, the dashed horizontal line projects the expected value of genotype AA to ease comparison with *Aa* and *aa*.

Knowing the effect of the three genotypes and the allele frequencies one can compute analytically the additive genetic variances associated with a bi-allelic locus (R. A. Fisher 1941; Lynch and Walsh 1998, p77). Thus, the additive genetic variances associated with *lepr* are  $0.052\text{g}^2$  for body mass and  $0.006\text{pup}^2$  for lifetime reproductive success. For both traits, *lepr* explains about 1% of the additive genetic variation as estimated from an animal model. This is rather large for a single locus given that quantitative traits loci typically explain a fraction of a percent to a few percent of additive genetic variance ( $V_A$ ), when they have a large enough sample size to mitigate Beavis effect (Flint and Mackay 2009; Jensen, Szulkin, and Slate 2014). Still, 1% of  $V_A$  is not sufficient to infer the evolutionary potential of the trait. Finally, genotyping many more markers, for instance using high-throughput sequencing (Goodwin, McPhereson, and McCombie 2016), is unlikely to improve this situation in the snow vole pop-



ulation. Generally, very large sample sizes and high-quality genomic resources are necessary to explain a biologically proportion of genetic variances (Bloom et al. 2013; Jensen, Szulkin, and Slate 2014). For instance 183,727 individuals were necessary to find 180 QTL that jointly explained only 13% of additive genetic variation in human body height (Lango Allen et al. 2010). High-throughput sequencing can also been used in a top-down way, that does not identify causal genetic variants, but instead quantifies the phenotypic variation jointly explained by all the genotyped markers. Thus, 3,925 individuals and 294,831 markers were able to explain 45% of the genetic variation in human height (Yang et al. 2010). This is much better, but given knowledge on the relatedness between individuals, quantitative genetics can estimate all the genetic variation in a phenotype, without any genotyping effort.

To conclude, bottom-up approaches can better unravel the molecular mechanisms underlying phenotypes. By opening the black box of what mechanisms make up a phenotype, they can identify what is most crucial in a phenotype, how it is linked to the environment and what is the target of natural selection (Jong et al. 2014). Moreover, they contribute to building a genotype-phenotype map, a long lasting challenge in evolutionary biology (Kirschner and Gerhart 2010). On the other hand, quantitative genetics lump all the effects of individual genes and their interactions into only a few parameters, non-informative about the underlying genetic architecture (Mackay 2001; Nietlisbach and Hadfield 2015; Huang and Mackay 2016). This summarized estimation provides simple and direct measures of genetic parameters. Quantitative genetics work directly on the phenotype which is the target of selection, and the source of ecological interactions. They therefore provide simple measures of genetic parameters that can directly be interpreted within the ecology of organisms. This thesis is concerned with the genetics and evolution at the level of organisms, in relation to their environment, and accordingly, most of my work relies on quantitative genetics.

## 1.3 This thesis

### 1.3.1 Objectives

In this thesis, I investigate the causes of individual-level variation in fitness, and the consequences of this variation at the population level. This thesis aims at better measuring and better understanding selection and evolution in the wild. It examines the relative importance of stochasticity and selection in shaping reproductive success and survival, disentangles evolutionary from plastic changes and explores the link between selection and evolution. These questions are addressed using a combination of computer simulations and of data from the long-term individual-based monitoring of a snow vole population.

### 1.3.2 Snow voles in Churwalden

The snow vole (*Chionomys nivalis*, Martins 1842) is a medium-sized rodent, its adult body size ranges from 10 to 14 cm, without the tail (5 to 7.5 cm long). Contrary to a widespread intuition, snow voles are not white (Fig. 1.2). Instead, the fur colour of



the upper-parts varies from light to dark taupe grey, sometimes tinted with brown or dark red. The misconception about color highlights that the species could favourably be renamed *rock vole*: it is a rock, rather than a snow, specialist (Luque-larena, López, and Gosálbez 2002) and might be associated with high elevations only because rocky areas are more widespread there. It is sparsely distributed across southern Europe and Asia Minor, from sea level up to 4000 m of elevation (Janeau and Aulagnier 1997).



Figure 1.2: Juvenile (left picture) and adult (right picture) snow voles in their habitat in Churwalden, Switzerland. Juveniles always lack the brown hue generally found in adults. Neither adults nor juveniles are white.

Snow voles excavate burrows under the rocks, but can also use natural clefts between rocks, sometimes carrying small stones to build walls (Niederer 2008). A burrow consists of tunnels connecting chambers, one for the nest and multiple ones to stock dry plants (Janeau and Aulagnier 1997). The species is not known to hibernate and is therefore exposed to harsh winter conditions in its high-elevation range. Adult females actively defend small territories against non-relatives, and tend to form matrilineal clusters of territories, whereas adult males wander, and fight, across large overlapping home-ranges (Luque-larena, López, and Gosálbez 2004; García-Navas, Bonnet, Waldvogel, et al. 2016). The mating system is promiscuous and a same litter can be sired by multiple males. Females normally produce 1 to 4 litters of 1 to 5 pups between May and September. Juveniles generally do not reproduce in their first civil year. Although they can eat flour worms in the lab, there is no evidence that snow voles are not strictly herbivorous in the wild (Janeau and Aulagnier 1997). In the Swiss Alps, snow voles suffer predation from red foxes, stoats, various owls and corvids, and parasitism from fleas, lice and ticks (Janeau and Aulagnier 1997; Martinoli et al. 2001).

The study area is located by the Churer Joch, Churwalden, in the Swiss canton Graubünden (Fig. 1.3; coordinates 46°48' N, 9°34' E), and covers about 5 ha between 1980 m and 2100 m above sea level. It consists of a west-exposed scree interspersed with small coniferous trees and with patches of alpine meadow. The study area is demarcated by extensive meadows to the south and to the north, by a coniferous forest to the west and by cliff to the east (Fig. 1.4).

Another scree offers about 1 ha of favourable habitat, starting 300 m north-east to

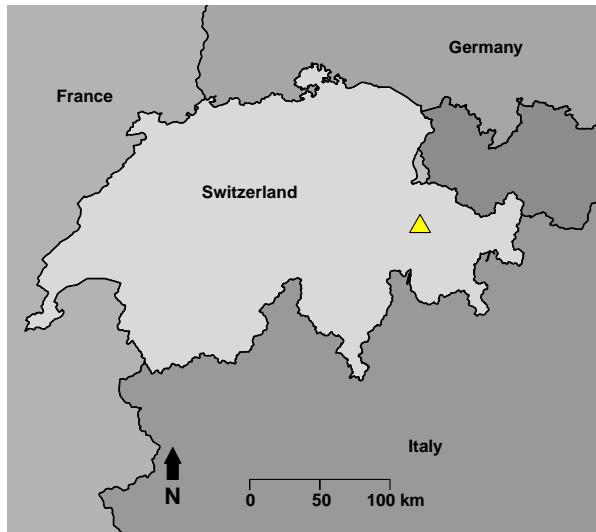


Figure 1.3: Location of the study area in Switzerland. The yellow triangle indicates the study area, with coordinates  $46^{\circ}48' N$ ,  $9^{\circ}34' E$ , by Churwalden, in the Swiss canton Graubünden. Countries are filled with different shades of grey, Austria and Lichtenstein are not labelled.

the monitored area. This area was trapped in 2008 and 2013. The snow vole density was rather low, with on average five captures per night of trapping, versus 18 on the main study area. More habitat favourable to snow voles can be found 2 Km to the south. The study population is moderately isolated and receives 5 to 10 immigrants per year, on a total of 60 to 180 individuals (García-Navas, Bonnet, Waldvogel, et al. 2016).

The monitoring of this snow vole population was initiated in 2006 by Dr. Peter W. Wandeler. Dr. Erik Postma took the monitoring over in 2012, but the protocol has remained practically unchanged. This thesis contains data collected up to the year 2015. Every year from 2006 to 2016, snow voles were life-trapped multiple times between late May and early October. Traps were set during the day, opened around sunset and checked the next morning. For every snow vole capture<sup>3</sup>, we recorded sex, age, body mass, body length, tail length, date, location and signs of reproductive activity (pregnancy, lactation, swollen scrotum). In addition, all newly-captured snow voles were individually marked and genotyped for 18 microsatellites (Wandeler, Ravaioli, and Bucher 2008). Based on the autosomal microsatellite genotypes, we reconstruct the pedigree of the population. This pedigree is the raw material for most of the work carried out during this thesis. In particular, the pedigree is used to define reproductive

<sup>3</sup>Other species (bank voles, pine voles, wood mice, stoats, black salamanders, slugs...) were released without taking measurements.



Figure 1.4: Distant view of the field site, taken from the west. The trapped area covers about a fifth of the width and a tenth of the height of the picture and is located in the centre. This scree is surrounded by a forest, a cliff and meadows.

success, as well as to estimate the relatedness between all pairs of individuals. These two statistics are essential to estimate selection, fitness and genetic variation.

### 1.3.3 Thesis outline

In natural populations, fitness is generally measured using individual measures of reproductive success and survival. These proxies are not fitness itself and their variation is largely stochastic, leading some authors to doubt that there is any significant variation in fitness in natural populations. Recent methodological developments appeared to support the view that variation in reproduction and survival was purely stochastic, and suggested that the potential for selection and evolution in the wild was largely over-estimated. In chapter ?? I examine these methods and, based on computer simulations, demonstrate that they lack statistical power to detect latent variation in fitness components. Using an alternative approach we show the presence of significant variation in the propensity of reproductive success in the snow vole population, thereby showing some potential for selection and adaptive evolution in this population. We also attempt to clarify some conceptual misunderstandings between the proponents of the two methodological schools.

A similar attempt motivated chapter ??, where, with collaborators from different methodological schools, we review and compare four frameworks to disentangle the causes of phenotypic changes. In particular, these frameworks differ in their estimation of the relative roles of plasticity, demography and genetic change. Based on com-

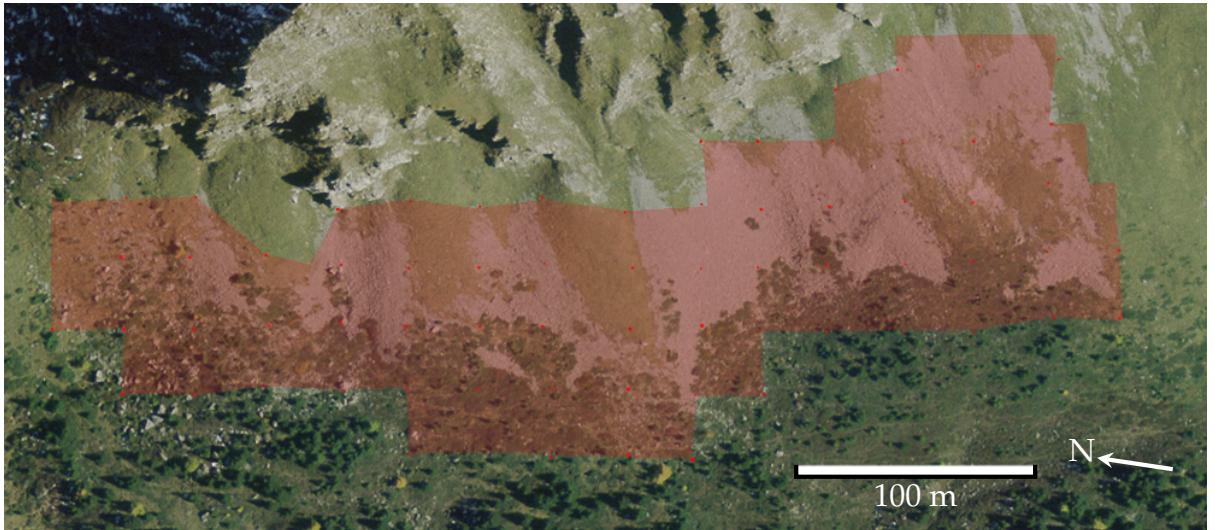


Figure 1.5: Orthophoto of the study site, from 2008. The red shading indicates the approximate area where traps are set.

puter simulations and on mathematical comparisons, we show that the discrepancies between the frameworks primarily originates from different definitions of the components of change. Nevertheless, one of these frameworks, the quantitative genetics *animal model*, stands out as the only framework able to estimate genetic change and the response to selection (that is, the trans-generational consequence of variation in fitness). I relied heavily on this framework for the two next chapters.

In chapter ??, I explore the reasons of the mismatch between apparent phenotypic selection, phenotypic change and genetic change for body mass. I describe one of the first case of contemporary evolution of a quantitative trait in the wild and show that this genetic change is adaptive. Both the evolution and the selective pressure responsible for it are invisible to purely phenotypic approaches, however. Using multivariate animal models, we identify the main component of selection as juvenile viability. I then infer that the target of selection is potential adult mass in juveniles and that selection is related to a recent change in climatic conditions.

The previous chapter considered selection and evolution averaged over the whole study period, without considering their temporal dynamic within the period. The fluctuation of selection is thought to be a major determinant of the rate of evolution, and a process to consider to understand adaptation in the wild. Nevertheless, unbiased measures of the variation of selection are rare and of the coupling between variation in selection and variation in evolution has been largely ignored. Chapter 2 shows that selection fluctuates in the study population, mainly due to variation in fertility selection. The rate of adaptive evolution is, however, remarkably constant, because viability selection, the driver of body mass evolution, does not vary. In this case the fluctuation of selection is evolutionary irrelevant. These two last chapters highlight the dangers of relying on phenotypic estimates of selection to understand the evolutionary dynamics of natural populations.

Finally, in chapter 3, I summarize the progresses made during this PhD on the un-

derstanding of natural populations, and discuss some of the remaining challenges and future working directions.

## References

- Akçay, E. and Van Cleve, J. 2016. "There is no fitness but fitness, and the lineage is its bearer." *Philosophical Transactions of the Royal Society B: Biological Sciences* **371** (1687): 20150085.
- Altermatt, F., Ebert, D., and Altermatt, F. 2016. "Reduced flight-to-light behaviour of moth populations exposed to long-term urban light pollution." *Biology Letters* **12**:20160111.
- Arnold, S. J. and Wade, M. J. 1984. "On the measurement of natural and sexual selection: Theory." *Evolution* **38** (4): 709–719.
- Bloom, J. S., Ehrenreich, I. M., Loo, W. T., Lite, T.-L. V., and Kruglyak, L. 2013. "Finding the sources of missing heritability in a yeast cross." *Nature* **494**:234–239.
- Brandon, R. and Beatty, J. 1984. "The propensity interpretation of 'fitness'. No interpretation is no substitute." *Philosophy of Science* **51** (2): 342–347.
- Brookfield, J. F. 2016. "Why are estimates of the strength and direction of natural selection from wild populations not congruent with observed rates of phenotypic change?" *BioEssays* **38**:1–8.
- Cam, E. and Monnat, J. Y. 2000. "Stratification based on reproductive state reveals contrasting patterns of age-related variation in demographic parameters in the kittiwake." *Oikos* **90** (3): 560–574.
- Darwin, C. 1859. *On the origin of species by means of natural selection, or the preservation of favoured races in the struggle for life*. John Murray, London.
- Dietrich, M. R. 2006. "From Mendel to molecules: A brief history of evolutionary genetics." In *Evolutionary genetics: concepts and case studies*. Edited by C. W. Fox and J. B. Wolf, 3–13. Oxford University Press, Oxford.
- Fisher, R. A. 1941. "Average excess and average effect of a gene substitution." *Annals of Eugenics* **11** (1): 53–63.
- Fisher, R. 1930. *The genetical theory of natural selection*. First. Oxford university press, Oxford, Great Britain.
- Fitzpatrick, M. J., Ben-shahar, Y., Smid, H. M., Vet, L. E. M., Robinson, G. E., and Sokolowski, M. B. 2005. "Candidate genes for behavioural ecology." *Trends in Ecology & Evolution* **20** (2): 96–104.
- Flint, J. and Mackay, T. F. C. 2009. "Genetic architecture of quantitative traits in flies, mice and humans." *Genome Research* **19**:723–733.

- Frank, S. a. 2012. "Natural selection. V. How to read the fundamental equations of evolutionary change in terms of information theory." *Journal of Evolutionary Biology* **25** (12): 2377–2396.
- García-Navas, V., Bonnet, T., Bonal, R., and Postma, E. 2016. "The role of fecundity and sexual selection in the evolution of size and sexual size dimorphism in New World and Old World voles (Rodentia: Arvicolinae)." *Oikos* **early view**.
- García-Navas, V., Bonnet, T., Waldvogel, D., Camenisch, G., and Postma, E. 2016. "Consequences of natal philopatry for reproductive success and mate choice in an Alpine rodent." *Behavioral Ecology* **27** (4): 1158–1166.
- Goodwin, S., McPherson, J. D., and McCombie, W. R. 2016. "Coming of age: ten years of next-generation sequencing technologies." *Nature Reviews Genetics* **17** (6): 333–351.
- Henderson, C. 1950. "Estimation of genetic parameters." *Annals of Mathematical Statistics* **21** (2): 309–310.
- Houseknecht, K. L., Baile, C. A., Matteri, R. L., and Spurlock, M. E. 1998. "The biology of leptin : a review." *Journal of Animal Science* **76**:1405–1420.
- Huang, W. and Mackay, T. F. 2016. "The genetic architecture of quantitative traits cannot be inferred from variance component analysis." *bioRxiv*. eprint: <http://biorxiv.org/content/early/2016/02/26/041434.full.pdf>.
- Janeau, G. and Aulagnier, S. 1997. "Snow vole - *Chionomys nivalis* (Martins 1842)." *IBEX Journal of Mountain Ecology* **4**:1–11.
- Jensen, H., Szulkin, M., and Slate, J. 2014. "Molecular quantitative genetics." In *Quantitative Genetics in the Wild*, edited by A. Charmentier, D. Garant, and L. E. B. Kruuk. Oxford: Oxford University Press.
- Jones, J. S. 1987. "The heritability of fitness: bad news for good genes?" *Trends in Ecology & Evolution* **2** (2): 35–36.
- Jong, M. a. de, Wong, S. C., Lehtonen, R., and Hanski, I. 2014. "Cytochrome P450 gene Cyp337 and heritability of fitness traits in the Glanville fritillary butterfly." *Molecular Ecology* **23** (8): 1994–2005.
- Kirschner, M. W. and Gerhart, J. C. 2010. "Facilitated variation." In *Evolution. The extended synthesis*, edited by M. Pigliucci and G. B. Müller. The MIT press, Cambridge, Massachusetts, USA.
- Krimbas, C. B. 2004. "On fitness." *Biology & Philosophy* **19** (2): 185–203.
- Kruuk, L. E. B. 2004. "Estimating genetic parameters in natural populations using the 'animal model'." *Philosophical Transactions of the Royal Society B: Biological Sciences* **359** (1446): 873–890.
- Kruuk, L. E. B., Charmantier, A., and Garant, D. 2014. "The study of quantitative genetics in wild populations." In *Quantitative Genetics in the Wild*, edited by A. Charmentier, D. Garant, and L. E. B. Kruuk. Oxford: Oxford University Press.

## Chapter 1 General introduction

- Lande, R. and Arnold, S. J. 1983. "The measurement of selection on correlated characters." *Evolution* **37** (6): 1210–1226.
- Lande, R. 1979. "Quantitative genetic analysis of multivariate evolution, applied to brain: body size allometry." *Evolution*: 402–416.
- Lango Allen, H., Estrada, K., Lettre, G., Berndt, S. I., Weedon, M. N., Rivadeneira, F., Willer, C. J., et al. 2010. "Hundreds of variants clustered in genomic loci and biological pathways affect human height." *Nature* **467** (7317): 832–8.
- Liedvogel, M., Cornwallis, C. K., and Sheldon, B. C. 2012. "Integrating candidate gene and quantitative genetic approaches to understand variation in timing of breeding in wild tit populations." *Journal of Evolutionary Biology* **25** (5): 813–23.
- Luque-larena, J. J., López, P., and Gosálbez, J. 2002. "Microhabitat use by the snow vole *Chionomys nivalis* in alpine environments reflects rock-dwelling preferences." *Canadian Journal of Zoology* **80**:36–41.
- . 2004. "Spacing behavior and morphology predict promiscuous mating strategies in the rock- dwelling snow vole, *Chionomys nivalis*." *Canadian Journal of Zoology* **82**:1051–1060.
- Lush, J. 1937. *Animal breeding plans*. Ames, Iowa: Iowa State College Press.
- Lynch, M. and Walsh, B. 1998. *Genetics and Analysis of Quantitative Traits*. Sinauer Associates.
- Mackay, T. 2001. "The genetic architecture of quantitative traits." *Annual Review of Genetics* **35**:303–309.
- Martinoli, A., Preatoni, D. G., Chiarenzi, B., Wauters, L. a., and Tosi, G. 2001. "Diet of stoats (*Mustela erminea*) in an Alpine habitat:The importance of fruit consumption in summer." *Acta Oecologica* **22** (1): 45–53.
- Mc Carty, J. P. 2001. "Ecological Consequences of Recent Climate Change." *Conservation Biology* **15** (2): 320–331.
- Merilä, J., Sheldon, B. C., and Kruuk, L. E. B. 2001. "Explaining stasis: microevolutionary studies in natural populations." *Genetica* **112**:199–222.
- Niederer, A. 2008. "Das Verhalten der Schneemaus." PhD diss., Universität Basel.
- Nietlisbach, P. and Hadfield, J. D. 2015. "Heritability of heterozygosity offers a new way of understanding why dominant gene action contributes to additive genetic variance." *Evolution*: 1–16.
- Oswald, T., MacLeod, C., and McCarty, M. 1944. "Studies on the chemical nature of the substance inducing transformation of pneumococcal types: induction of transformation by a desoxyribonucleic acid fraction isolated from pneumococcus type III." *Journal of Experimental Medicine* **79** (2).
- Parmesan, C. 2006. "Ecological and evolutionary responses to recent climate change." *Annual Review of Ecology, Evolution, and Systematics*: 637–669.

- Pigliucci, M. and Müller, G. B. 2010. *Evoution. The extended synthesis*. The MIT press, Cambridge, Massachusetts, USA.
- Postma, E. 2014. "Four decades of estimating heritabilities in wild vertebrate populations: improved methods, more data better estimates?" In *Quantitative Genetics in the Wild*, edited by A. Charmentier, D. Garant, and L. E. B. Kruuk. Oxford: Oxford University Press.
- Price, P. W. 1996. *Biological evolution*. Saunders College Publishing, Philadelphia, PA.
- Rousset, F. 2004. *Genetic structure and selection in subdivided populations*. Princeton, New Jersey, USA.
- Shaw, R. G. and Shaw, F. H. 2014. "Quantitative genetic study of the adaptive process." *Heredity* **112** (1): 13–20.
- Steiner, U. K. and Tuljapurkar, S. 2012. "Neutral theory for life histories and individual variability in fitness components." *Proceedings of the National Academy of Sciences, USA* **109** (12): 4684–4689.
- Tafani, M., Cohas, A., Bonenfant, C., Gaillard, J.-M., and Allainé, D. 2013. "Decreasing litter size of marmots over time: a life history response to climate change ?" *Ecology* **94** (3): 580–586.
- Tienderen, P. H. van. 2000. "Elasticities and the link between demographic and evolutionary dynamics." *Ecology* **81** (3): 666–679.
- Turner, B. M. 2009. "Epigenetic responses to environmental change and their evolutionary implications." *Philosophical Transactions of the Royal Society B: Biological Sciences* **364** (1534): 3403–18.
- Wade, M. J. 2006. "Natural selection." In *Evolutionary genetics: concepts and case studies*. Edited by C. W. Fox and J. B. Wolf, 399–413. Oxford University Press, Oxford.
- Wagner, G. P. 2010. "The measurement theory of fitness." *Evolution* **64** (5): 1358–76.
- Wandeler, P., Ravaioli, R., and Bucher, T. B. 2008. "Microsatellite DNA markers for the snow vole (*Chionomys nivalis*)." *Molecular Ecology Resources* **8**:637–639.
- Watson, J. and Crick, F. 1953. "Genetical implications of the structure of deoxyribonucleic acid." *Nature* **171** (4361): 964–967.
- Wayne, M. L. and Miyamoto, M. M. 2006. "Genetic variation." In *Evolutionary genetics: concepts and case studies*. Edited by C. W. Fox and J. B. Wolf, 399–413. Oxford University Press, Oxford.
- Wilkins, J. S. 2009. *Species. A history of the idea*. Berkeley, California, USA: University of California Press.
- Wolf, J. B. and Wade, M. J. 2009. "What are maternal effects (and what are they not)?" *Philosophical Transactions of the Royal Society B: Biological Sciences* **364** (1520): 1107–15.

## *Chapter 1 General introduction*

Yang, J., Benyamin, B., McEvoy, B. P., Gordon, S., Henders, A. K., Nyholt, D. R., Madden, P. A., et al. 2010. "Common SNPs explain a large proportion of the heritability for human height." *Nature Genetics* **42** (7): 565–569.

# Chapter 2

## Fluctuating selection

*All entities move and nothing remains still.*

— Heraclitus cited by Plato, *Cratylus* (4-5th century BCE)

*It is impossible to live in the past, difficult to live in the present and a waste to live in the future.*

— Frank Herbert, *Dune* (circa 1965)



# Chapter 3

## General discussion

*It is difficult to understand the universe if you only study one planet.*

— Miyamoto Musashi, *A Book of Five Rings* (circa 1645)

*The world is always full of the sound of waves. The little fishes, abandoning themselves to the waves, dance and sing, and play, but who knows the heart of the sea, a hundred feet down? Who knows its depth?*

— Eiji Yoshikawa, *Musashi* (1935)

### 3.1 Overview

In this thesis, I investigated the causes and consequences of variation in fitness in a wild population. I showed that the variation in proxies for individual fitness is not purely stochastic, but is underlain by variation in latent fitness (chapitre ??). Besides, the variation in latent fitness has an additive genetic component, showing the presence of natural selection and of adaptive evolution in the snow vole population (chapter ?? and 2). I explored ways to decompose the causes of phenotypic changes and identified the animal model from quantitative genetics as a convenient tool to estimate evolution (chapter ??). Using this tool in various ways, I showed that body mass was an important contributor of variation in fitness proxies (chapter ??), but not in a consistent way over time (chapter 2). Nevertheless, body mass was a consistent contributor to variation in genetic variation for fitness (chapters ?? and 2), and therefore, body mass evolved over the study period.

Below, I will discuss further the insight brought by this thesis and the remaining challenges, in understanding the causes of phenotypic variation and the response of wild populations to environmental change.

### 3.2 The causes of phenotypic variation

This thesis brings some new knowledge about the causes of variation in fitness and other phenotypes, but there is still much more to learn. Causality can be refined *ad infinitum* unless, perhaps, once phenotypic variation is modelled in term of quantum interactions between fundamental particles. Below, I comment on two promising directions that were only touched upon in this thesis, but have the potential to improve the predictive understanding of the causes and consequences of fitness variation. These

are the effect of an individual's gene on other individuals, and the study of the molecular basis of genetic variation through genomics.

### 3.2.1 The effect of others' genes

Using quantitative genetics, I decomposed the phenotypic variation of morphological and life-history traits into components related to additive genetic effects, maternal effects or permanent environments. This decomposition was sufficient to measure the rate of evolution of the direct genetic effects (chapter ??), that is, the direct action of an individual's genes on its own body. Nevertheless, an individual's genes have effects reaching out beyond its body, to the environment, including other individuals (Dawkins 1982), whether it is through interactions between individuals (indirect genetic effects, e.g. maternal effects, McAdam, Garant, and Wilson 2014), or through the pleiotropic action of genes expressed in kin at different life-stages (e.g. genetic conflicts, Trivers 1974).

Indirect genetic effects could be an important component shaping selection and evolution in the snow vole population. Indeed, in the snow voles, genes within an individual are likely to affect the phenotype of another individual during at least two types of situations. First, related females tend to form clusters of territories, and the presence of kin could suppress reproduction in subordinate females (García-Navas et al. 2016). Moreover, as in all placental mammals, maternal effects on offspring phenotypes are prevalent from pregnancy to weaning. Maternal effects have been studied extensively in natural populations (Wolf and Wade 2009), but estimations of the genetic component of maternal effects remain scarce (McAdam, Garant, and Wilson 2014). Nevertheless, genetic maternal effects could provide extra evolutionary potential in addition to that of direct genetic variation (McGlothlin and Galloway 2014; McAdam, Garant, and Wilson 2014; Mcfarlane et al. 2015). In the snow vole population, preliminary analyses showed the presence of additive genetic maternal effects for body mass (results not shown). Genetic maternal effects for mass could therefore be subject to selection and evolve adaptively. In chapter ??, maternal genetic effects are not explicitly modelled, and their evolution is assigned to phenotypic plasticity. A full account of body-mass evolution should measure this evolution in addition to that of direct additive genetic effects.

Besides indirect genetic effects, the effect of others' genes matters for evolution in the case of genetic conflicts, that is, genetic trade-off between traits expressed in different individuals. For four decades, genetic conflicts between parents and offspring have been thought to be a major constraint on the evolution of size (since Trivers 1974), but the idea resisted empirical tests despite behavioural studies showing patterns consistent with it (Kölliker et al. 2015). Kölliker et al. 2015 demonstrated that a genetic trade-off between offspring number and offspring size constrains the evolution of size in earwigs (*Forficula auricularia*, Linnaeus 1758). Moreover, Rollinson and Rowe 2015 presented qualitative evidence suggesting that this constraint is widespread among animals and could be a general explanation for the evolutionary stasis of size. In chapter ?? we briefly explored the possibility that a genetic conflict constrains the evolution of body mass, and found qualitative evidence that it is not the case. The snow vole study system is not an ideal to test this hypothesis, however. First, we do not capture

all juveniles—some die or emigrate before their first year—and cannot measure litter size accurately. Because mass is under selection in juveniles, selective disappearance is likely to blur the trade-off signal (Hadfield, Wilson, and Kruuk 2011). Second, the size-number genetic trade-off is best described as an explanation of evolutionary stasis of size or mass, but mass is evolving in the snow vole population (chapter ??), making it more difficult to formulate an expectation for the genetic covariance between mass and litter size. Finally, it is in theory possible to measure the genetic trade-off using quantitative genetics, but nor the exact model to fit nor the modelling tools are published yet (Hadfield 2012; Rollinson and Rowe 2015). An experimental approach remains the only option to quantitatively test for a size-number genetic trade-off (Kölleiker et al. 2015), and such an approach appears impossible in a wild population such as Churwalden’s snow voles.

### 3.2.2 Molecular basis of genetic variation

During this PhD, on several occasions, Dr Erik Postma and myself, considered using high-throughput genome sequencing (van Dijk et al. 2014) to sequence the snow vole population retrospectively (tissue is kept in  $-80^{\circ}$  freezers for most of the individuals trapped in the last ten years). As of yet, we did not obtain the funding necessary, and I ran out of time to carry out work in the laboratory and to develop a bio-informatic pipeline. As I discussed in chapter 1, molecular approaches to measuring selection and evolution are in general inferior to quantitative genetic approaches. Nevertheless, individual-based genomic data could bring complementary insights to my empirical chapters.

To start with, individual-based genomic data could marginally improve the estimation of quantitative genetic parameters (Bérénos et al. 2014) by: (i) allowing the use of realized relatedness in animal models, instead of the relatedness expected from the pedigree; and (ii) providing some relatedness information about individuals with unknown parents (for which there is no information at all in a pedigree). More importantly, individual-based genomic data would allow the identification of some of the genetic loci underlying phenotypic variation and quantitative evolution. This task is generally a challenging one in small populations (Wellenreuther and Hansson 2016), but the snow vole population presents three rare advantages that would ease it considerably.

First, at least one trait, body mass, has been evolving during the last decade, and some adaptive molecular evolution must have happened. The search for the molecular basis of evolution would therefore start with the knowledge that there is something to find, and with indications on what functional types of genes are likely to be involved. Second, in natural populations, it is difficult to show that evolution at a genetic locus is due to selection and not only due to drift, because there is in general no null-expectation for the effect of drift under complex demographics and mating patterns. A pedigree provides such a null expectation. Simulating the random dropping of alleles down our pedigree would result in a null distribution of changes in allele frequencies against which to test for the effect of selection on each genetic locus. This method was successfully employed to show contemporary adaptive evolution at 67 genetic loci in a wild population of Florida scrub-jays (Nancy Chen, Evolution confer-

ence, 2016, Austin, USA). Third, thanks to the availability of life-history data, it would be possible to correlate the allelic variation of the evolving loci to success and failure in various life-stages. Therefore, the combination of genomic and life-history data can pinpoint when selection occurs in life, and what kind of molecular mechanism selection acts on. Altogether, individual-based genomic data could therefore refine not only our molecular understanding of phenotypic variation, but also provide clues regarding the ecological nature of selection.

### 3.3 Predicting responses to environmental change

Anthropogenic environmental change has triggered research aiming at understanding and predicting the response of natural populations to environmental change (Parmesan 2006; Chevin, Collins, and Lefèvre 2012; Smallegange and Coulson 2013; Charmantier and Gienapp 2014), but massive challenges hinder this research agenda. Already, the retrospective study of phenotypic and demographic responses often remains inconclusive (Merilä, Sheldon, and Kruuk 2001; Mc Carty 2001; Charmantier and Gienapp 2014; Brookfield 2016) and, at the moment, prospective prediction seems out of reach in most cases. During my PhD, I confronted three challenges that must be tackled to improve the predictive abilities of evolutionary ecology. Below, I discuss the problems with measuring selection, predicting the response to selection, and integrating evolutionary and demographic responses.

#### 3.3.1 Measuring selection in the wild

For over 150 years, natural selection has been known to cause the match between organisms and their environment, and biologists have attempted to understand its causes and mechanisms. More recently, the study of selection assumed a more applied goal as researchers hope to predict the response of natural populations to the selective pressures imposed by environmental change (Chevin, Lande, and Mace 2010; Coulson, Tuljapurkar, and Childs 2010; Merilä and Hendry 2014). The principle of natural selection is very simple: in a given environment, individuals with a phenotype that favours survival and fertility contribute more to the next generation. Given the level of research attention on such a simple process, it can be surprising to see how slowly the understanding of natural selection has developed, and how difficult its study remains. For most of the 20th century, the main brake to progresses was the lack of an unified framework to quantify selection in natural populations (Wade 2006). Such a framework progressively emerged, starting with covariance-based methods (Robertson 1966; Price 1970) which efficiently measure the total effect of selection. The most influential breakdown was the popularization of regression-based methods (Lande 1979; Lande and Arnold 1983) which measure the proportional effect of selection per unit of phenotypic variation, and allows to decompose selection into the direct and indirect effects of selection on multiple traits (Broodie III, Moore, and Janzen 1995). Since then, these methods have provided thousands of estimates of selection in natural populations (Kingsolver et al. 2001; Stinchcombe et al. 2008; Kingsolver et al. 2012), thus showing several general patterns. For instance, directional selection is stronger

and more common than suggested by early evolutionists, whereas stabilizing selection appears to be rare, while fertility selection is generally stronger than viability selection (Kingsolver et al. 2012). The abundance of estimates of selection should not be mistaken for a good understanding of natural selection, however. The estimation of selection through regression-methods faces at least three difficulties that might severely hamper their significance and explain the general absence of response to selection (Merilä, Sheldon, and Kruuk 2001; Brookfield 2016).

First, to obtain an unbiased measure of selection, fitness should be regressed on the trait of interest. Since, fitness is rarely observable directly, fitness proxies must be used instead. Many estimates of selection are computed on fitness components, for instance fertility and survival (Kingsolver et al. 2012). In this case, the estimation of selection can be biased in the presence of a trade-off between fitness components: a certain phenotype might increase survival but decrease fertility, so that the net selection on the trait is null, despite covariation with fitness components (Thompson et al. 2011; Kingsolver et al. 2012; Brookfield 2016). Fortunately, this bias appears to be minor in general, with the exception of body mass (Kingsolver and Diamond 2011). For the empirical part of this thesis (chapter ?? and 2), I used fitness proxies that attempted to include all fitness components in order to avoid a bias. Thus, I used lifetime reproductive success when measuring selection within a generation, and annual reproductive success plus twice survival when measuring selection within a year. These fitness proxies are imperfect since we do not capture all juveniles and a trade-off between early juvenile survival and reproduction could bias the selection estimation (Hadfield 2008). Still, estimates of evolution using Price equation (that is, selection on the genotype) or using the trend in BLUPs for breeding values (that is, not using any information about selection nor fitness) agree qualitatively with my corrected estimates of selection (chapter ?? and 2), suggesting that my proxies for fitness are adequate.

Second, it is possible to estimate the total effect of selection on a trait with selection differentials, but it is much more difficult to disentangle the causal selective effect of a trait from the indirect selection due to other traits. In theory, it is possible to disentangle direct and indirect selection by including all the traits under selection in the analysis (Lande and Arnold 1983). In natural populations, however, it is impossible to know *a priori* what traits are under selection, and often it is impossible to measure all relevant traits (Brookfield 2016; Hadfield 2008). Furthermore, as more traits are included in a selection analysis, the statistical power to detect significant selection on any one trait decreases (Mitchell-Olds and Shaw 1987). I did detect significant indirect selection on body mass, but genetic correlations between the traits considered were such that the prediction of evolution was not affected by the inclusion of indirect selection (chapter ??). Only three traits were tested, however, and we cannot exclude that body mass is not under indirect selective pressure. The evolution of body mass could be driven by selection on an unmeasured trait. Nevertheless, this problem is irrelevant to the measures of total selection and evolution, on which chapters ?? and 2 rely.

Third, covariance-based and regression-based methods to estimate phenotypic selection essentially measure the statistical association between traits and relative fitness. Selection must however be a causal association, be it direct or indirect. If the statistical association is entirely mediated by an environmental covariance between

traits and fitness, there is no selection and no possibility of genetic response to selection (Price and Liou 1989; Rausher 1992). Body mass, the main trait analysed in this thesis, is likely to be very sensitive to this source of bias. Indeed, a favourable environment—for instance food rich and lacking parasites—is likely to lead to larger mass, high survival, and high fertility. Accordingly, phenotypic estimates of natural selection on mass and size are overwhelmingly positive, but mass does not evolve has predicted from its heritability (Blanckenhorn 2000; Kingsolver et al. 2012). In the snow voles, an excess of environmental covariance does underlie the apparent selection on mass (chapter ??). A solution to the problem is the experimental manipulation of the trait of interest. This can break the link between phenotype and individual quality and reveals the causal action of phenotype on fitness components (e.g. Tinbergen and Sanz 2004; Tscharren and Richner 2006). Still, experimental manipulation is no without its own limitations. Thus, manipulations are work intensive, time consuming and must be designed carefully in order to manipulate the trait of interest without affecting any other trait. Moreover, manipulations cannot easily be applied to all traits. The approach has been widely used to study selection on brood size, but it is not clear to me how one could manipulate body mass in a controlled way (that is, without accidentally affecting other traits). Instead of an experiment, my approach to the challenge of environmental covariation has been to use quantitative genetics to identify the target of natural selection (chapter ??). After having shown on-going adaptive evolution, I decomposed phenotypic selection into an additive genetic and an environmental component, for various fitness components. I found that only juvenile viability selection showed an additive genetic component, and according to the Robertson-Price identity, was the source of adaptive evolution. Understanding the mechanism of this selection and measuring its strength was then a matter of hypothesis testing. This approach could be used on other systems provided the presence of adaptive evolution. Nonetheless, it requires sufficient phenotypic and relatedness data to fit bivariate animal models. In addition, in the snow vole a single fitness drove evolution, but multiple fitness components could be involved, thus complicating the analysis. Finally, identifying the right fitness component(s) does not guarantee that the phenotypic mechanism of selection can be identified. A good understanding of the biological system will be necessary to formulate a reasonable hypothesis for the cause of selection. The testability of this hypothesis will also depend on data availability and quality, and will be subject to the limits of hypothesis testing approaches: there is always a risk of false positive, equal to the significance level chosen for the test, and a correlation does not prove causation.

### 3.3.2 Evolutionary response

Once a measure of phenotypic selection is obtained, it is straightforward to formulate a prediction of genetic response based on the breeder's equation and on a heritability estimate (Lush 1937; Falconer and Mackay 1996). We have already seen (chapter ?? and 2) that such a prediction is often unreliable in natural populations, however. Estimates of selection might not correspond to causal selection, and unmeasured selection acting on genetically correlated traits might constrain evolution. I have shown that estimating the genetic component of selection, or the rate of evolution, can test whether

selection has been measured appropriately to be predictive (chapter ??).

Nevertheless, most attempts to understand the evolutionary response to environmental change do not measure genetic parameters. Thus, the alarming lack of evidence for evolutionary responses to climate change probably originates primarily from a lack of tests for genetic change (Charmantier and Gienapp 2014; Gienapp and Brommer 2014; Merilä and Hendry 2014; Crozier and Hutchings 2014). Ignoring the genetic properties (e.g. the heritability) of the trait of interest (e.g. Forcada and Hoffman 2014; Coulson and Clegg 2014; Traill, Schindler, and Coulson 2014) easily leads to underestimating, or incorrectly dismissing, the potential to respond to selection and the actual evolutionary response (Nietlisbach and Hadfield 2015; Chevin 2015; Pigeon et al. 2016). Similarly, the evolutionary potential of small populations was dismissed by population matrix simulations that ignored genetic-based arguments (see chapter ??). Moreover, methods based on phenotypic covariances do not distinguish between the presence and the absence of heritable variation, and cannot be used alone to predict an evolutionary response (chapter ??).

Therefore, all the chapters of this thesis illustrate that a genetic approach, be it based on quantitative genetics or population genetics, is necessary to measure evolution, and can more reliably identify the selective causes and the constraints shaping adaptation. Attempts to understand the evolutionary dynamics of natural populations based on phenotypic observations only (e.g. Smallegange and Coulson 2013) are a gamble, that might work on special occasions, but is unlikely to be reliable in general.

### 3.3.3 Demographic response to environmental change

This thesis is almost exclusively concerned with traits and their evolutionary dynamics. In the context of understanding the response of natural population to environmental change, such an investigation is legitimate. Whether a trait distribution changes through demographic, plastic, or genetic mechanisms has different consequences on the fate of the population (Chevin and Lande 2010). Nevertheless, for most applications, and to the eyes of the society, it is unimportant whether animal and plant populations respond to climate change primarily through migration, through plastic changes, or through evolution. The primary motivation of this research is to ascertain whether populations will persist or go extinct, and how managers can affect the outcome.

This question is primarily a demographic one. The evolutionary approach that was mine during this PhD is not sufficient to ascertain the fate of the snow vole population, but it might be a useful first step. Indeed, it is now widely acknowledged that evolutionary processes can act on the same time scale as ecological ones, and that they can significantly affect demographics (Hairston et al. 2005; Ellner, Geber, and Hairston 2011; Chevin, Lande, and Mace 2010; Turcotte, Reznick, and Hare 2011). For instance, theory and laboratory experiments support the existence of *evolutionary rescue*, that is, adaptive genetic change within a population that prevent the population extinction (Gonzalez et al. 2013; Schiffers et al. 2013). Still, empirical evidences of evolutionary rescue in the wild remain extremely limited (Vander Wal et al. 2013).

In chapter ??, I inferred that the genetic response to selection tended to increase mean juvenile survival over the study period. All other things being equal, evolution

therefore had a positive demographic effect and contributed to the recovery of population size. It is, however, unclear to me how to quantify the demographic effect of evolution. To the best of my knowledge, an appropriate methodological framework is still lacking. Indeed, traditional demographic models used to predict population resilience ignore individual heterogeneity and genetic change (Kendall et al. 2011; Vindenes and Langangen 2015; Plard et al. 2016). On the other hand, quantitative genetic studies focus on estimating rates of evolutionary change, but mostly ignore their possible consequences for the dynamics of populations (Coulson, Tuljapurkar, and Childs 2010; Chevin, Collins, and Lefèvre 2012). It is now acknowledged that the integration of evolutionary and demographic aspects is crucial for predicting trait dynamics, population resilience and viability (Schoener 2011; Pelletier et al. 2012; Chevin, Collins, and Lefèvre 2012; Merilä and Hendry 2014). But only in the last year have publications proposed methods that could start to address this question in the wild (Vindenes and Langangen 2015; Coulson et al. 2015; Childs, Sheldon, and Rees 2016), and these should certainly been followed up.

## 3.4 General conclusion

Natural selection is a potent force that shapes the evolution of natural populations, but its causes and consequences can be blurred by the complexity of natural populations. Understanding the process of adaptation requires to isolate selection from the stochasticity in fitness components, to disentangle evolution from other drivers of phenotypic change, and to mechanistically link genetic change to selective pressures. Being able to do so, thanks to an individual-based monitoring including genetic relatedness, I provided a rare example of contemporary adaptive evolution. More examples of evolution in action certainly await to be described, and will make our understanding of the response to environmental change more general and more predictive. This thesis, however, shows in several ways that crucial insight is more likely to come from studies that explicitly study the genetic aspects of selection and phenotypic changes.

## References

- Bérénos, C., Ellis, P. a., Pilkington, J. G., and Pemberton, J. M. 2014. "Estimating quantitative genetic parameters in wild populations: a comparison of pedigree and genomic approaches." *Molecular Ecology* **23**:3434–3451.
- Blanckenhorn, W. 2000. "The evolution of body size: what keeps organisms small?" *Quarterly Review of Biology* **75** (4): 385–407.
- Broodie III, E. D., Moore, A. J., and Janzen, F. J. 1995. "Visualizing and quantifying natural selection." *Trends in Ecology & Evolution* **10** (8): 313–318.
- Brookfield, J. F. 2016. "Why are estimates of the strength and direction of natural selection from wild populations not congruent with observed rates of phenotypic change?" *BioEssays* **38**:1–8.

- Charmantier, A. and Gienapp, P. 2014. "Climate change and timing of avian breeding and migration: evolutionary versus plastic changes." *Evolutionary Applications* 7 (1): 15–28.
- Chevin, L.-M. 2015. "Evolution of adult size depends on genetic variance in growth trajectories: A comment on analyses of evolutionary dynamics using integral projection models." *Methods in Ecology and Evolution* 6 (9): 981–986.
- Chevin, L.-M., Collins, S., and Lefèvre, F. 2012. "Phenotypic plasticity and evolutionary demographic responses to climate change: taking theory out to the field." Edited by A. Hoffmann. *Functional Ecology* 27 (4): 967–979.
- Chevin, L.-M. and Lande, R. 2010. "When do adaptive plasticity and genetic evolution prevent extinction of a density-regulated population?" *Evolution* 64 (4): 1143–1150.
- Chevin, L.-M., Lande, R., and Mace, G. M. 2010. "Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory." *PLoS Biology* 8 (4): e1000357.
- Childs, D. Z., Sheldon, B. C., and Rees, M. 2016. "The evolution of labile traits in sex- and age-structured populations." *Journal of Animal Ecology* 85 (2): 329–342.
- Coulson, T. and Clegg, S. 2014. "Population biology: Fur seals signal their own decline." *Nature* 511:414–415.
- Coulson, T., Plard, F., Schindler, S., Ozgul, A., and Gaillard, J.-M. 2015. "Quantitative genetics meets integral projection models : unification of widely used methods from ecology and evolution." *arXiv*. <http://de.arxiv.org/abs/1509.01351v1>.
- Coulson, T., Tuljapurkar, S., and Childs, D. Z. 2010. "Using evolutionary demography to link life history theory, quantitative genetics and population ecology." *Journal of Animal Ecology* 79 (6): 1226–1240.
- Crozier, L. G. and Hutchings, J. A. 2014. "Plastic and evolutionary responses to climate change in fish." *Evolutionary Applications* 7 (1): 68–87.
- Dawkins, R. 1982. *The extended phenotype*. Oxford Universtiy Press, Oxford, U.K.
- Ellner, S. P., Geber, M. A., and Hairston, N. G. 2011. "Does rapid evolution matter? Measuring the rate of contemporary evolution and its impacts on ecological dynamics." *Ecology Letters* 14 (6): 603–614.
- Falconer, D. S. and Mackay, T. F. C. 1996. *Introduction to quantitative genetics*. 4th ed. Edited by Longman. Essex, U.K.
- Forcada, J. and Hoffman, J. 2014. "Climate change selects for heterozygosity in a declining fur seal population." *Nature* 511:462–465.
- García-Navas, V., Bonnet, T., Waldvogel, D., Camenisch, G., and Postma, E. 2016. "Consequences of natal philopatry for reproductive success and mate choice in an Alpine rodent." *Behavioral Ecology* 27 (4): 1158–1166.

### Chapter 3 General discussion

- Gienapp, P. and Brommer, J. E. 2014. "Evolutionary dynamics in response to climate change." In *Quantitative genetics in the wild*, edited by A. Charmentier, D. Garant, and L. E. B. Kruuk, 254–273. Oxford: Oxford University Press, Oxford.
- Gonzalez, A., Ronce, O., Ferriere, R., and Hochberg, M. E. 2013. "Evolutionary rescue: an emerging focus at the intersection between ecology and evolution." *Philosophical Transactions of the Royal Society B: Biological Sciences* **368** (1610): 20120404.
- Hadfield, J. D. 2008. "Estimating evolutionary parameters when viability selection is operating." *Proceedings of the Royal Society of London B: Biological Sciences* **275** (1635): 723–734.
- . 2012. "The quantitative genetic theory of parental effects." In *The evolution of parental care*, 267–284. Oxford Univ. Press, Oxford, U. K.
- Hadfield, J. D., Wilson, A. J., and Kruuk, L. E. B. 2011. "Cryptic evolution: does environmental deterioration have a genetic basis?" *Genetics* **187** (4): 1099–113.
- Hairston, N. G., Ellner, S. P., Geber, M. A., Yoshida, T., and Fox, J. A. 2005. "Rapid evolution and the convergence of ecological and evolutionary time." *Ecology Letters* **8** (10): 1114–1127.
- Kendall, B. E., Fox, G. A., Fujiwara, M., and Nogeire, T. M. 2011. "Demographic heterogeneity, cohort selection, and population growth." *Ecology* **92** (10): 1985–1993.
- Kingsolver, J. G., Hoekstra, J. M., Berrigan, D., Vignieri, S. N., Hill, C. E., Hoang, A., Gibert, P., and Beerli, P. 2001. "The strength of phenotypic selection in natural populations." *The American Naturalist* **157** (3): 245–261.
- Kingsolver, J. G. and Diamond, S. E. 2011. "Phenotypic selection in natural populations: what limits directional selection?" *The American Naturalist* **177** (3): 346–57.
- Kingsolver, J. G., Diamond, S. E., Siepielski, A. M., and Carlson, S. M. 2012. "Synthetic analyses of phenotypic selection in natural populations: lessons, limitations and future directions." *Evolutionary Ecology* **26** (5): 1101–1118.
- Kölliker, M., Boos, S., Wong, J. W., Röllin, L., Stucki, D., Raveh, S., Wu, M., and Meunier, J. 2015. "Parent-offspring conflict and the genetic trade-offs shaping parental investment." *Nature Communications* **6**:6850.
- Lande, R. and Arnold, S. J. 1983. "The measurement of selection on correlated characters." *Evolution* **37** (6): 1210–1226.
- Lande, R. 1979. "Quantitative genetic analysis of multivariate evolution, applied to brain: body size allometry." *Evolution*: 402–416.
- Lush, J. 1937. *Animal breeding plans*. Ames, Iowa: Iowa State College Press.
- Mc Carty, J. P. 2001. "Ecological Consequences of Recent Climate Change." *Conservation Biology* **15** (2): 320–331.

- McAdam, A. G., Garant, D., and Wilson, A. 2014. "The effects of others' genes: maternal and other indirect genetic effects." In *Quantitative Genetics in the Wild*, edited by A. Charmentier, D. Garant, and L. E. B. Kruuk. Oxford: Oxford University Press.
- Mcfarlane, S. E., Gorrell, J. C., Coltman, D. W., Humphries, M. M., Boutin, S., and Mcadam, A. G. 2015. "The nature of nurture in a wild mammal's fitness." *Proceedings of the Royal Society B: Biological Sciences* **282**:20142422.
- McGlothlin, J. W. and Galloway, L. F. 2014. "The contribution of maternal effects to selection response: An empirical test of competing models." *Evolution* **68** (2): 549–558.
- Merilä, J., Sheldon, B. C., and Kruuk, L. E. B. 2001. "Explaining stasis: microevolutionary studies in natural populations." *Genetica* **112**:199–222.
- Merilä, J. and Hendry, A. P. 2014. "Climate change, adaptation, and phenotypic plasticity: The problem and the evidence." *Evolutionary Applications* **7** (1): 1–14.
- Mitchell-Olds, T. and Shaw, R. G. 1987. "Regression analysis of natural selection: statistical inference and biological interpretation." *Evolution* **41** (6): 1149–1161.
- Nietlisbach, P. and Hadfield, J. D. 2015. "Heritability of heterozygosity offers a new way of understanding why dominant gene action contributes to additive genetic variance." *Evolution*: 1–16.
- Parmesan, C. 2006. "Ecological and evolutionary responses to recent climate change." *Annual Review of Ecology, Evolution, and Systematics*: 637–669.
- Pelletier, F., Moyes, K., Clutton-Brock, T. H., and Coulson, T. 2012. "Decomposing variation in population growth into contributions from environment and phenotypes in an age-structured population." *Proceedings of the Royal Society of London B: Biological Sciences* **279** (1727): 394–401.
- Pigeon, G., Festa-Bianchet, M., Coltman, D. W., and Pelletier, F. 2016. "Intense selective hunting leads to artificial evolution in horn size." *Evolutionary Applications*: in press.
- Plard, F., Gaillard, J.-M., Coulson, T., and Tuljapurkar, S. 2016. "Des différences, pourquoi? Transmission, maintenance and effects of phenotypic variance." *Journal of Animal Ecology* **85** (2): 356–370.
- Price, G. R. 1970. "Selection and covariance." *Nature* **227**:520–521.
- Price, T. and Liou, L. 1989. "Selection on clutch size in birds." *The American Naturalist* **134** (6): 950–959.
- Rausher, M. D. 1992. "The measurement of selection on quantitative traits: biases due to environmental covariances between traits and fitness." *Evolution* **46** (3): 616–626.
- Robertson, A. 1966. "A mathematical model of the culling process in dairy cattle." *Animal Production* **8**:95–108.

### *Chapter 3 General discussion*

- Rollinson, N. and Rowe, L. 2015. "Persistent directional selection on body size and a resolution to the paradox of stasis." *Evolution* **69** (9): 2441–2451.
- Schiffers, K., Bourne, E. C., Lavergne, S., Thuiller, W., and Travis, J. M. J. 2013. "Limited evolutionary rescue of locally adapted populations facing climate change." *Philosophical Transactions of the Royal Society B: Biological Sciences* **368** (20120083): 20120083.
- Schoener, T. W. 2011. "The newest synthesis: understanding the interplay of evolutionary and ecological dynamics." *Science* **331** (6016): 426–429.
- Smallegange, I. M. and Coulson, T. 2013. "Towards a general, population-level understanding of eco-evolutionary change." *Trends in Ecology & Evolution* **28** (3): 143–148.
- Stinchcombe, J. R., Agrawal, A. F., Hohenlohe, P. a., Arnold, S. J., and Blows, M. W. 2008. "Estimating nonlinear selection gradients using quadratic regression coefficients: double or nothing?" *Evolution* **62** (9): 2435–40.
- Thompson, D. J., Hassall, C., Lowe, C. D., and Watts, P. C. 2011. "Field estimates of reproductive success in a model insect: behavioural surrogates are poor predictors of fitness." *Ecology Letters* **14** (9): 905–913.
- Tinbergen, J. M. and Sanz, J. J. 2004. "Strong evidence for selection for larger brood size in a great tit population." *Behavioral Ecology* **15** (4): 525–533.
- Traill, L. W., Schindler, S., and Coulson, T. 2014. "Demography, not inheritance, drives phenotypic change in hunted bighorn sheep." *Proceedings of the National Academy of Sciences, USA* **111** (36): 13223–13228.
- Trivers, R. L. 1974. "Parent-offspring conflict." *American Zoologist* **14**:249–264.
- Tschirren, B. and Richner, H. 2006. "Parasites shape the optimal investment in immunity." *Proceedings of the Royal Society B: Biological Sciences* **273** (1595): 1773–1777.
- Turcotte, M. M., Reznick, D. N., and Hare, J. D. 2011. "Experimental assessment of the impact of rapid evolution on population dynamics." *Evolutionary Ecology Research* **13**:113–131.
- van Dijk, E. L., Auger, H., Jaszczyzyn, Y., and Thermes, C. 2014. "Ten years of next-generation sequencing technology." *Trends in Genetics* **30** (9): 418–426.
- Vander Wal, E., Garant, D., Festa-Bianchet, M., and Pelletier, F. 2013. "Evolutionary rescue in vertebrates: evidence, applications and uncertainty." *Philosophical transactions of the Royal Society of London B: Biological sciences* **368** (1610): 20120090.
- Vindenes, Y. and Langangen, Ø. 2015. "Individual heterogeneity in life histories and eco-evolutionary dynamics." *Ecology Letters* **18** (5): 417–432.
- Wade, M. J. 2006. "Natural selection." In *Evolutionary genetics: concepts and case studies*. Edited by C. W. Fox and J. B. Wolf, 399–413. Oxford University Press, Oxford.

- Wellenreuther, M. and Hansson, B. 2016. "Detecting Polygenic Evolution: Problems, Pitfalls, and Promises." *Trends in Genetics* **32** (3): 1–10.
- Wolf, J. B. and Wade, M. J. 2009. "What are maternal effects (and what are they not)?" *Philosophical Transactions of the Royal Society B: Biological Sciences* **364** (1520): 1107–15.



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