

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

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von

Timothée Bonnet

aus
Frankreich

Promotionskomitee

Prof. Dr. Lukas Keller (Vorsitz)
Dr. Erik Postma (Leitung der Dissertation)
Prof. Dr. Ben Sheldon
Prof. Dr. Barbara Tschirren
Prof. Dr. Arpat Ozgul
Dr. Jarrod Hadfield
Dr. Marc Kéry

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timotheebonnetc@gmail.com

Photos: Timothée Bonnet

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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

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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.

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

1.1 Variation in fitness

1.1.1 The origin of variation in evolutionary biology

Understanding the causes of variation among living beings is at the heart of evolutionary questioning (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, but also 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)¹. Of course, the effect of ageing was acknowledged and plastic responses to the environment were thought to be predominant (Wilkins 2009). Ageing and the environment does not always provide a satisfactory explanation for variation that appears within a population, however. Furthermore, Darwinian

¹ 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).

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 is evolvable, but it can 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. Tienderen 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). 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. I feel like the relative definition is really closer to the interest of evolutionary biologists and avoids appending *relative* to every occurrence of *fitness*. Nevertheless the field massively favours the absolute definition and 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). I see great conceptual promises in this view, that brings together an essentialist use of the word *fitness* and the scientific field of information theory. 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 attracted 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 will not deal with the second point, the fundamental question of appearance and maintenance of genetic variation in fitness, but rather with its proximal sources. We will consider these proximal sources from two complementary angles.

First in a descriptive approach, one can decompose variation in fitness into components of variation, without mention of the underlying mechanisms. 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 selection². 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 (McCarty 2001; Shaw and Shaw 2014). This regain of focus 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-

²In 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.

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 ?? 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 latter 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 the molecular mechanisms 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, meaning that it could very well be the result of chance alone. 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?

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 0.052g^2 for body mass and 0.006pup^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 population. 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 quan-

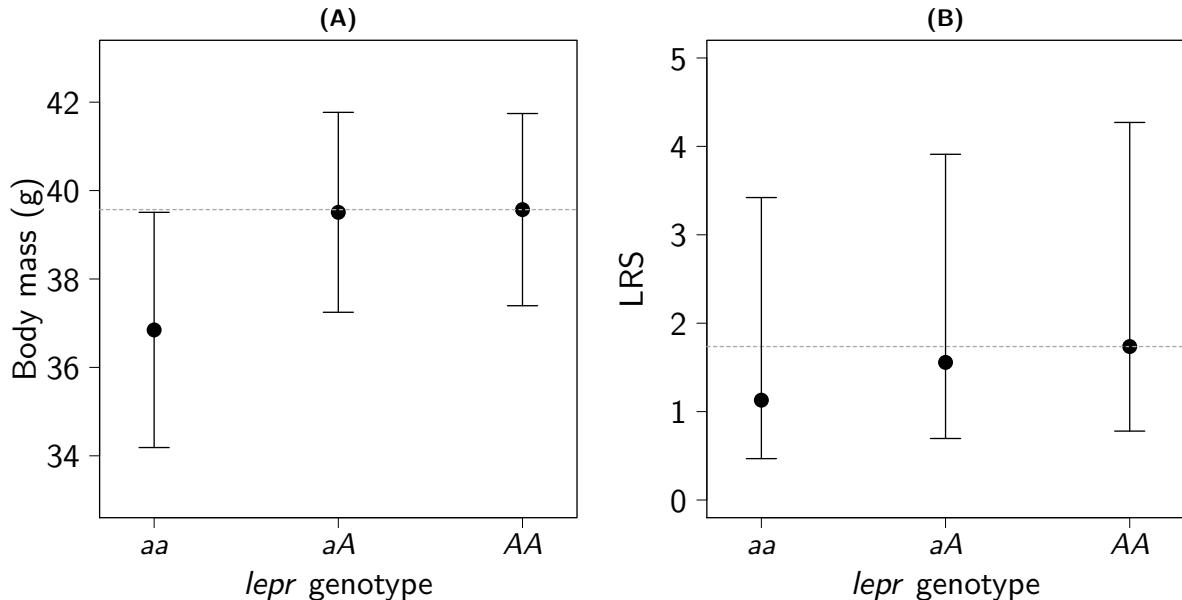


Figure 1.1: Body mass and lifetime reproductive success (LRS) as a function of *lepr* genotypes. (A) Expected body mass of snow voles bearing the three *lepr* genotypes. The expectations and 95% confidence intervals were predicted from a linear mixed model fitted to the 2311 mass measurement of 532 snow 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*.

tifies 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 proximal 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).

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





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.

and corvids, and parasitism from flees, lices 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 consist 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. reffig:landscape).

Another scree, called Wolfgruoben, offers about 1 ha of favourable habitat, starting 300 m north-east to the monitored area. Wolfgruoben 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 capture³, 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

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

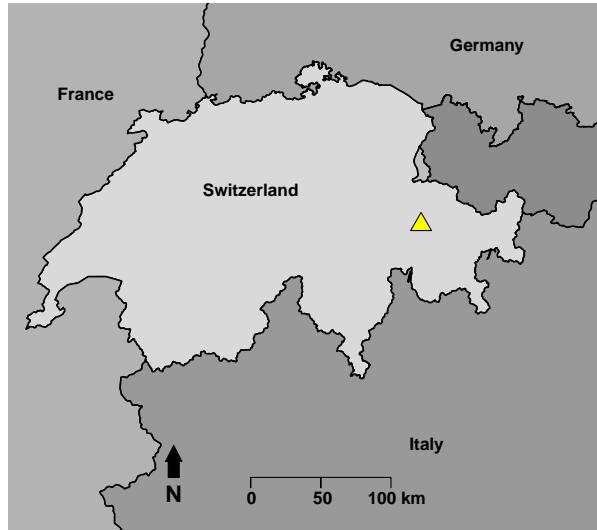


Figure 1.3: Location of the study area in Switzerland. The yellow triangle indicates the study area, with coordinates $46^{\circ}48' N, 9^{\circ}34' E$, by Churwalden, in the Swiss canton Graubünden. Countries are filled with different shades of grey, Austria and Lichtenstein are not labelled.

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 estima-



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.

tion of the relative roles of plasticity, demography and genetic change. Based on computer 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 ?? shows that selection fluctuates in the study population, mainly due to variation in fertility

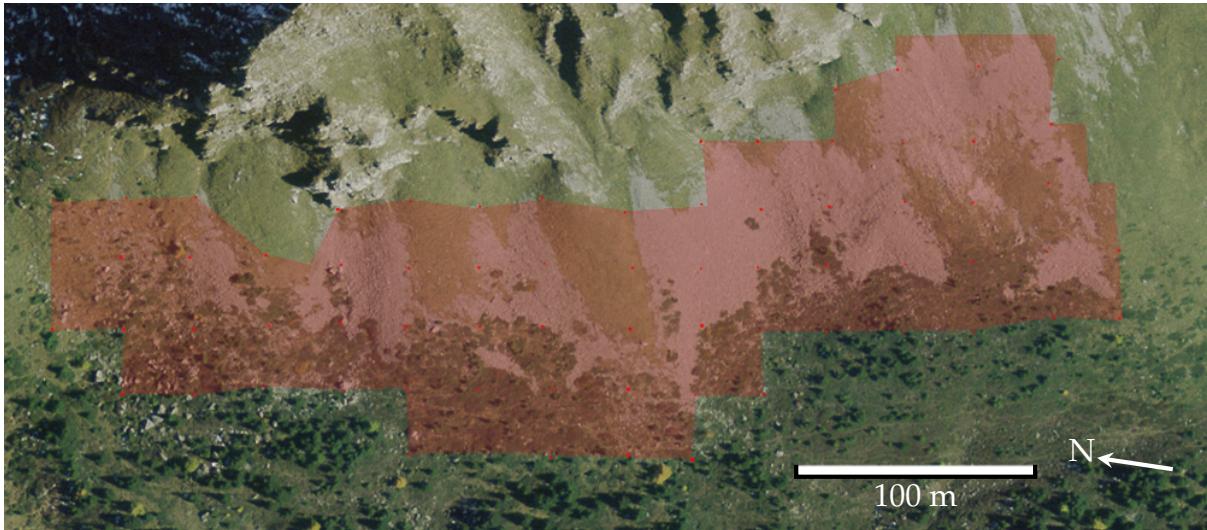


Figure 1.5: Orthophoto of the study site, from 2008. The red shading indicates the approximate area where traps are set.

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 2**, I summarize the progresses made during this PhD on the understanding 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**:201160111.
- 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.

Chapter 1 General introduction

- 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. Charmantier, 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 and 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. Charmantier, D. Garant, and L. E. B. Kruuk. Oxford: Oxford University Press.
- 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.

Chapter 1 General introduction

- Lynch, M. and Walsh, B. 1998. *Genetics and Analysis of Quantitative Traits*. Sinauer Associates.
- Mackay, T. 2001. "The genetic architecture of quantitative traits." *Annu Rev Genet* **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*)." *Mol Ecol Res* **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.
- 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." *Nat Gen* **42** (7): 565–569.

Chapter 2

General discussion

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

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

2.1 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 ??). 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 ??). Nevertheless, body mass was a consistent contributor to variation in genetic variation for fitness (chapters ?? and ??), 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.

2.2 The causes of phenotypic variation

2.2.1 Indirect genetic effects

In this thesis, I have 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 actions 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), or through the pleiotropic action of genes expressed at different life-stages (e.g. genetic conflicts).

Indirect genetic effects are an emerging question in evolutionary quantitative genetics (**OUHUIHOU**) and could be an important component shaping selection and evolution in the snow vole population. Indeed, genes within an individual are likely to affect the phenotype of another individual during at least two types of situations. Snow voles tend to inherit territories from mother to daughter and to form clusters of territories with sisters (García-Navas et al. 2016), but the presence of kin might suppress reproduction in subordinate females. Moreover, as in all placental mammals, maternal effects on offspring phenotypes are prevalent from pregnancy to weaning. Maternal effects have been studied extensively (Wolf and Wade 2009), but only recently have researchers started to measure the genetic variance in maternal effects (**ejfkj**). These genetic maternal effects could provide extra evolutionary potential in addition to that of direct genetic variation (McGlothin and Galloway 2014; Mcfarlane et al. 2015). Preliminary analyses showed the presence of additive genetic maternal effects for body mass in the snow vole population. Genetic maternal effects could be subject to selection and could evolve adaptively, and a full account of body-mass evolution should probably measure this evolution in addition to that of direct additive genetic effects. In chapter ??, maternal genetic effects are not explicitly modelled, and their evolution is assigned to phenotypic plasticity.

Thus, genetic conflicts, that is, genetic trade-off between traits expressed in different individuals (Trivers 1974), are thought to be a major constraint on evolution. For four decades, the existence of genetic parent-offspring remained untested, despite suggestive evidence from behavioural studies (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 earrings. Rollinson and Rowe 2015 then 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 suggestive 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, 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 (Kölliker et al. 2015), and such an approach appears impossible in a wild population such as Churwalden's snow voles.

2.2.2 Molecular basis of genetic variation

Improve QG estimates by solving problem of realized relatedness and immigrants being treated as part of the base population.

GWAS limited in small populations, but combination with a pedigree improves power greatly. Find causal genetic variants responsible for evolution cf scrub jays.

2.2.3 Origin and maintenance of variation in fitness

Still, our study is a snapshot, too short to expect significant loss of genetic variation. What would happen if the observed selective pressures would carry on? Fluctuating selection might contribute to the maintenance of genetic variation New mutations certainly contribute, but probably not sufficient in general (charlesworth). Population structure.

2.3 The response of wild populations to environmental change

2.3.1 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. 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. Therefore, 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 the appearance of 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 measures 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), 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 challenges 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. Fitness is rarely observable directly, and 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 (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 ??), I used fitness proxies that attempted to include all fitness components in order to avoid such biases. 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. Still, the estimation of evolution using Price equation (that is, selection on the genotype) and that using the trend in BLUPs for breeding values (that is, not using any information about selection nor fitness) agree qualitatively (chapter ??), suggesting that the generational proxy for fitness is 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). This thesis did not bring 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 any direct 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 ?? 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 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 (Blanckenhorn 2000; Kingsolver et al. 2012) and an excess of environmental covariance does underlie the apparent selection on mass in the snow voles (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 thought 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). 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: the rate of false positive is equal to the significance level chosen, and a correlation does not prove causation.

2.3.2 Genetic response to environmental change

Predictions based on phenotypic estimates of selection (chapter ??) (chapter ??)

2.3.3 Demographic response to environmental change

Within-population variation in fitness is relevant to the demographic response to environmental changes, either through the direct effects of phenotypic variation (including genetic variation) on demographic rates (Kendall et al. 2011; Vindenes and Langangen 2015; Plard et al. 2016), or indirectly, following genetic changes (Chevin, Lande, and Mace 2010; Turcotte, Reznick, and Hare 2011; Schiffrers et al. 2013).

2.4 Conclusion

References

- 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.

Chapter 2 General discussion

- 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.
- Dawkins, R. 1982. *The extended phenotype*. Oxford University Press, Oxford, U.K.
- 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.
- 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.
- 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., Gilbert, 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." *Evol Ecol* **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.
- 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.
- Mitchell-Olds, T. and Shaw, R. G. 1987. "Regression analysis of natural selection: statistical inference and biological interpretation." *Evolution* **41** (6): 1149–1161.
- 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.
- 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.
- 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; international journal of organic 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.
- Trivers, R. L. 1974. "Parent-offspring conflict." *Am Zool* **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.
- Vindenes, Y. and Langangen, Ø. 2015. "Individual heterogeneity in life histories and eco-evolutionary dynamics." *Ecology Letters* **18** (5): 417–432.

Chapter 2 General discussion

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.

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.