Why do individuals differ in fitness? Investigating the maintenance of variation in life-history traits

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Genetical evolution,
if we choose to look at it liverishly instead of with fatuous good humor,
is a story of waste, makeshift, compromise and blunder.

The Future of Man, Sir Peter Medawar

Abstract

Within populations, individuals vary in fitness. Why this variation is maintained is central to our understanding of ecology and evolutionary biology. The general consensus is that variation is largely maintained by the resolution of life-history trade-offs, by which limited resources force individuals to invest in one trait to the detriment of another. However, there remains a lack of conclusive evidence that supports this theory. In this thesis, I investigate why variation in life-history traits is maintained. To separate the costs of reproduction from quality effects, I conducted a meta-analysis of the two traits most closely related to fitness; fecundity and survival, and quantified the fitness consequences of brood size decisions. I show the costs of reproduction are limited and cannot explain constraints on fecundity as assumed in life-history theory. I then used a long-term dataset of a closed population of house sparrows (*Passer domesticus*) to separate the genetic and environmental effects of fecundity and survival. I found no evidence of costs of reproduction in terms of survival to the following year in genetic or permanent environmental space. This suggests that individuals are not consistent in their quality. Body temperature in birds is well studied as an indicator of current condition. I used infrared technology to determine whether, as nestlings age, body temperature is genetically driven or derived from the nest environment. I find small heritability of body temperature which decreases throughout the nestling period. I also find the natal environment has carry-over body temperature effects as nestlings age independently of physiological growth. I provide evidence that body temperature is under selection during the nestling period. The work in this thesis highlights how the costs of reproduction are limited and quality effects are variable and constrained to phenotypic space. This work challenges the current mainstream theory that life-history trade-offs drive between-individual variation in fitness.

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Gannets’ Rock, Lundy Island.

Contents

Chapter 1: General Introduction

1.1 The components of fitness

At an individual level, fitness is estimated by determining the relative proportion of genes contributed to future generations in the population. Longevity dictates how many reproductive attempts an individual can have and is, therefore, a crucial life-history trait. The quality of offspring produced throughout an individual's life, and not just their number, also determines its fitness. For each breeding attempt, life-history theory predicts that the number of offspring that is produced should be at the level that maximises lifetime reproductive success (Stearns 1976). The major life-history traits of fecundity and survival are expected to trade-off with each other or with other traits in order to benefit an individual's current situation.

It is not always the most beneficial tactic for an individual to produce more offspring as it has been shown that investing more in fewer offspring can result in a higher offspring recruitment rate (Lack 1947; Kluijver 1951). This is often more commonly the case in females (though not always), especially in species where males provide no parental care (Kölliker et al. 2014). Individuals within a population should reproduce at the rate which balances quality of offspring with quantity in order to produce the highest fitness return. This should mean that comparative (e.g., same age) individuals within the population should move towards reproducing at the same rate as fitness differences that selection can act upon should have been removed (Fisher 1958). It is, therefore, puzzling that all individuals within a population do not reproduce at the population mean, which in theory should be the optimal clutch size for all individuals. By experimentally increasing the number of offspring in a nest, brood manipulation studies have shown that parents are usually able to successfully raise more offspring than they originally produced (Roskaft 1985; Lessells 1986; De Kogel 1997;

Monaghan and Nager 1997). It may be that this strategy optimises the number of offspring over a lifetime. For example, parents may underinvest as an insurance against environmental instability or may be constrained by the amount of resource they can allocate to reproduction in a given year (Roskaft 1985; Burness et al. 2000; Reid et al. 2003).

Survival and lifespan are often used interchangeably, however, they have fundamentally different meanings. Survival, at the population level, refers to the proportion of individuals that are alive at a given age (or given time), often estimated yearly in life-history studies. Longevity or lifespan (used interchangeably) refer to the age to which an individual survives. Both intrinsic and extrinsic factors affect an individual's lifespan and survival, influenced by both genetic predisposition (also influenced by selection pressures) and environmental factors, respectively.

Intrinsically, an individual’s genetics influences not only its predisposed longevity but also its ability to cope with its situation, such as during extreme temperatures and when infected with disease, though the two are not mutually exclusive. However, there is only mixed support for survival being heritable (Table 1.1) and the genetic basis for survival in wild animals is little understood. In addition to this, lifespans can vary even in animals that are genetically identical and in laboratory conditions, as shown in *Caenorhabditis elegans* (Kirkwood and Finch 2002). This demonstrates that it is not just environment and genetics that determines an individual’s lifespan but factors such as mutation, molecular interactions and configuration of organelles as well as random stochasticity (further details in Kirkwood et al. 2005).

Table 1.1: Heritability of adult survival in wild populations. Studies were found in a literature search on Google Scholar using a keyword search of “heritability of survival” AND “adult survival” AND “wild”. I have also included some additional studies of which I was aware that contained heritabilities of survival.

Taxa	Survival measure	Species	Finding	h^2	Reference
Bird	Lifespan	*Ficedula albicollis*	Yes in males No in females	0.15 ± 0.06 0.00 ± 0.06	Merilä and Sheldon 2000
	Lifespan	*Passer domesticus*	No	0.003 (0.001-0.006)	Schroeder et al. 2012
	Lifespan	*Sterna hirundo*	Yes	0.09 (0.00-0.28)	Vedder et al. 2021a
	Survival	*Cyanistes caeruleus*	No	0.02 (0.00-0.08)	Papaïx et al. 2010
	Lifespan	*Larus novaehollandiae*	No in females Yes in males	0.00 ± 0.00 0.21 ± 0.11	Teplitsky et al. 2009a
Mammal	Lifespan	*Cervus elaphus*	No in females No in males	0.00 ± 0.54 0.006 ± 0.09	Kruuk et al. 2000
	Lifespan	*Ovis aries*	Yes	(Variance = 0.20 (0.05-0.42))	Froy et al. 2021
	Lifespan	*Macaca mulatta*	Yes	0.43 ± 0.08	Blomquist 2010
Fish	Survival	*Salmo salar*	Yes	0.32 ± 0.10	Salte et al. 2010

Extrinsically, factors such as predation, disease and weather conditions can drive population-level mortality rates. In these cases, an animal's death would not be exclusively caused by a decline in condition as the animal ages – i.e., senescence. As such, the causes of mortality are numerous and may be conflated with other, linked causes, such as age and sex. Furthermore, mortality is not constrained to specific time periods, as is the case with fecundity. For these reasons, it was thought that wild animals would never reach senescence because extrinsic factors would cause mortality before animals reached an age at which senescence was detectable and, indeed, there are high levels of pre-senescent mortality in wild populations (Medawar 1952b; Nussey et al. 2008). There is now strong evidence, however, that senescence does occur in wild animals (reviewed in Nussey et al. 2008). Survival, senescence and age-specific fecundity explain why individuals within a population vary in the number of offspring they produce.

1.2 Life-history trade-offs; the hidden costs of reproduction

Selection acts weakly on old individuals, allowing for deleterious germ-line mutations that have late-acting effects to accumulate (Medawar 1952b). Ageing is defined as the reduction in physiological functioning with age (Kirkwood and Austad 2000). However, why individuals age is not fully understood. One explanation is the antagonistic pleiotropy hypothesis, first described by Williams (1957), which is an extension of the mutation accumulation hypothesis devised by Medawar (1952b). The antagonistic pleiotropy hypothesis suggests that, as selection acts more strongly in early life, pleiotropic genes which have beneficial early-life effects will be selected for, even if these genes are detrimental later in life. Another explanation is the disposable soma theory, which states that, as resources are limited, an individual should invest in growth and reproduction in early life, which leads to reduced

investment in somatic maintenance and drives senescence (Kirkwood 1977). These theories have developed into the field of life-history theory, and life-history trade-off theory is commonly cited as the reason why individuals exhibit different fitness returns within a population despite other plausible explanations such as mutation-selection and fluctuating selection (Stearns 1992; Roff 1993). Indeed, it is often argued that when no trade-off is observed that variation in a particular trait must be the result of a trade-off with another trait not being explicitly tested.

Life-history trade-off theory assumes that greater investment in one aspect of life requires the sacrifice of another (Stearns 1992; Roff 1993). As resources are limited, individuals must make trade-offs (van Noordwijk and de Jong 1986). Differences in resource allocation should arise when individuals invest in certain traits at a level which benefits their own situation at a given time. Though trade-offs can occur between many traits, trade-offs have commonly been used to explain why differences in reproductive effort are maintained, both within and between species. Across taxa, species vary in fundamental life-history traits, such as the number of offspring produced and the time spent raising young (Lack 1947; Harvey and Clutton-Brock 1985; Ricklefs 2000a). This between-species variation is maintained because of a trade-off between the number of offspring produced and the effort per offspring. Longer-lived species generally invest more heavily in fewer offspring and shorter-lived species generally produce more offspring but provide little parental care. This concept has also been influential in explaining why individuals within a population vary in fitness-related traits, as it is assumed that individuals trade-off investment in fecundity with survival, through self maintenance (Zera and Harshman 2001a).

Studies testing trade-offs with respect to fecundity have commonly done this by experimentally altering an individual's current reproductive investment and attempting to locate areas of reduced function in other areas of the individual’s life-history. As time and

resources are constrained during breeding, it is expected that individuals will sacrifice their own self maintenance in favour of increased reproductive effort. In a meta-analysis on brood-size manipulation experiments, the survival of parents whose clutch sizes were increased or reduced did not differ significantly from control (Santos and Nakagawa 2012). One possible explanation for this is that the trade-off decision was made when the original brood was produced, leaving parents unable to adjust their resource allocation in response to the experiment. If this is the case, then it would be expected that a trade-off instead occurs within the brood. It has been found that increased brood size leads to the offspring having reduced survival, body mass and fecundity (Gustafsson and Sutherland 1988; Smith et al. 1989; Merilä and Wiggins 1995; Charnov and Ernest 2006); however, see (Tinbergen and Sanz 2004). It is also possible that a trade-off was not detected because the costs of reproduction are delayed and instead impact future fitness. A review of early–late-life trade-offs across vertebrate species suggested that increased early-life fitness investments resulted in reduced fitness later in life (Lemaître et al. 2015). However, this review also noted alternative responses in late life in reproduction and longevity within the same study, though no evidence was found that either future reproduction or longevity was more likely to display a trade-off. It is also likely that longer-lived species are more likely to display late-life costs in reproduction as a result of environmental variability and shorter-lived species should exhibit survival costs (Gaillard and Yoccoz 2003; Hamel et al. 2010). This is because longer-lived species gain fitness through extending their longevity, and therefore gaining more reproductive attempts, rather than through maximising current reproductive effort.

It is, therefore, not the case that trade-offs do not exist but rather there is currently no conclusive evidence to suggest that trade-offs drive variation in within-population fitness-related traits. Furthermore, the costs of reproduction do not seem to be generalisable across species or even within species. This, therefore, means that we currently do not understand why variation in fitness-related traits is maintained. Understanding this would

allow us to predict what factors lead an individual to be of higher quality at a given time; such information could have some applied value, such as in conservation programmes.

1.3 Age-specific patterns of fecundity and survival

Not all breeding opportunities are equal. Resource allocation can lead to patterns of reproduction and survival that vary throughout an individual's life. As well as at the individual level, age-specific fecundity varies between species. For example, many longer-lived species delay their first breeding attempt for several years after becoming independent from their parents (Mourocq et al. 2016). At younger ages, many species produce fewer offspring than at older ages (Curio 1983). One possibility is that this arises because birds in poor condition produce fewer offspring and have lower overall survival, perhaps consistent with the high mortality of young adults that has been well documented in birds (Botkin and Miller 1974; Reid 1988). However, increasing reproduction with age has also been shown to occur at the within-individual level, demonstrating that low-quality individuals are not exclusively driving the population-level (between-individual) relationship between age and reproductive output (Bouwhuis et al. 2009; Hammers et al. 2012). Alternatively, as individuals grow older, they gain experience and so are better able to gain resources, high-quality mates or occupy better habitats, which enable them to raise more young (Curio 1983). Between midlife and old age, reproductive output declines (reproductive senescence), and this has largely been attributed to antagonistic pleiotropy (Medawar 1952b; Williams 1957; Bouwhuis et al. 2010; Hammers et al. 2012; Nussey et al. 2013). It is possible that, as individuals age and decrease in condition, they invest less in reproduction due to the accumulation of damage brought about by the number of reproductive attempts they have made (McNamara et al. 2009). However, after a peak of reproductive output and survival rates in mid-life, both fecundity and survival decline into late adulthood, suggesting that experience is only in part

determining age-related fitness patterns. Later age declines are also matched with a decrease in survival probability (actuarial senescence) and various physiological traits towards old age (Nussey et al. 2008). However, some studies have failed to find evidence of senescence, which questions whether senescence is driven by antagonistic pleiotropy. Alternative explanations for observed survival and reproductive senescence include confounding effects of seasonal behaviour (Slade 1995) and populations where extrinsic factors are the main source of mortality (Pistorius and Bester 2002). A counter hypothesis to senescence is the Relative Reproductive Rate Hypothesis, where certain traits lead to increased survival and/or reproduction at older ages than younger (Congdon et al. 2001, 2003).

Individuals can experience a marked reduction in reproductive functioning in the year before death, known as a terminal decline (Coulson and Fairweather 2001). Though this effect in itself is not senescence, as the terminal effect is more severe than would be predicted by senescence alone, senescence may inflate terminal declines. Furthermore, terminal declines have been detected before senescence is otherwise detectable (Rattiste 2004). Poor performance in the year preceding death is likely a result of poor condition caused by disease, parasite load or accumulated genetic damage surpassing a threshold for which the animal can function normally (Ricklefs 2000b). Alternatively, individuals can increase reproductive output in the year prior to death; this is known as terminal investment (Clutton-Brock 1984; Bonneaud et al. 2004; Hanssen 2006). The theory behind this is that, as individuals cannot invest in self-maintenance in a way that will lengthen their lifespan, they instead invest heavily in reproduction.

Individuals within a population vary in their pattern of ageing through age-specific changes in resource allocation (Vaupel et al. 1979). This is driven by the environmental conditions an individual experiences throughout its life and leads to different reproductive peaks at different

ages between individuals (Schroeder et al. 2012; Marasco et al. 2018a). Similarly, individuals may adjust the timing of key life-history events based on their current and past experiences, such as their age at first breeding (Kruger 2005). The degree of stress an individual experiences and metabolically challenging activities, leading to oxidative damage, may have long-term life-history implications and be a driver of individual differences in ageing (Monaghan 2008; Monaghan et al. 2009; Pérez et al. 2009; Speakman and Selman 2011; Selman et al. 2012). These factors not only shape an individual's ageing pattern but also determine how well an individual will perform in relation to the population at a given time.

1.4 Individual quality

In addition to life-history trade-off theory, which predicts negative correlations between life-history traits, some studies have reported positive correlations between fitness-related traits, suggesting instead that variation in life-history traits is caused by differences in individual quality (Pettifor et al. 1988; Cam et al. 1998; Beauplet et al. 2006; Weladji et al. 2008; Hamel et al. 2009a; McLean et al. 2019). van Noordwijk and de Jong (1986) argue it is variation in both the acquisition and allocation of resources that determines whether correlations between fitness-related traits are positive or negative. However, the idea of individual quality has received comparatively less attention than life-history trade-off theory. Individual quality can be defined as a trait or a collection of traits that lead to an individual’s phenotype producing a higher relative fitness return than the population mean (Wilson and Nussey 2010). Furthermore, Wilson and Nussey (2010) argue that quality should also be repeatable. The importance of increased relative fitness returns in defining quality is key, as individuals may have alternative age-specific fitness outputs, without differing in their lifetime fitness (Marasco et al. 2018a). As such, if quality is only investigated through one trait, a

high-quality individual may be wrongly or misleadingly defined because it is not of consistently higher quality for other traits.

In an evolutionary context, the idea of individual quality is often dismissed, as high-quality individuals should be selected for, eventually leading to no or little variation in fitness-related traits. A further complicating factor is that life-history traits are influenced by the environment in early life and/or maternal effects and, therefore, it is difficult to separate genetic variation from early-life conditions. Though cross-fostering (where offspring from one brood are swapped with another) is used as an experimental attempt to solve this issue in studies of birds, the effects of incubation and early-hatching experience are often overlooked (Nord and Nilsson 2011; Hadfield et al. 2013). Furthermore, phenotypes can vary over the course of an individual's life due to ageing and environmental influences, driving variation in their fitness over their life (Nussey et al. 2007; Wilson et al. 2008).

It may be that high-quality individuals are not consistent in their quality, meaning that in one specific environment individuals with specific phenotypes will outperform others, but in other environments these phenotypes will underperform. If this is the case, separating quality effects from differences in reproductive patterns of ageing will be challenging and studies aiming to determine quality need to define the context in which quality is being applied. For example, if older individuals produce more offspring, does this mean that older individuals are of higher quality than younger individuals? As discussed by Tuljapurkar et al. (2009), quality could be produced by dynamic heterogeneity, where quality is driven by stochasticity, and therefore quality should be determined by comparing the size of the life-history trait to a null model.

As predicted, there are numerous examples of adult characteristics which are indicative of quality, such as antler length in red deer stags (Schmidt et al. 2001) and plumage-colour

dimorphism in birds (Owens and Hartley 1998). As an extension of this, indicators of an individual's quality, especially in early life, could be used to predict individual future success, in a way that would be invaluable, for example, in conservation programmes. Despite numerous examples of how early-life conditions can affect future fitness (Burness et al. 2000; Hamel et al. 2009b; Bouwhuis et al. 2010; Hammers et al. 2013; Marasco et al. 2018a), there is a lack of studies that separate early-life effects from genetic quality. However, it is difficult to separate genetic and phenotypic quality from developmental conditions (Hadfield et al. 2013). This is particularly true in wild systems, where it is difficult to track individuals throughout the entirety of their lifespan, where early-life conditions are likely to vary substantially and detailed data on environmental conditions during early life are often lacking.

Individual quality has been somewhat overlooked as a driver of between-individual variation in fitness traits, largely due to the assumption that quality cannot drive performance or else selection would act upon it. Research is therefore needed on whether quality can be located in genetic space or if quality is driven by the permanent environment effect. Furthermore, we need more research on what factors lead an individual to be of higher quality. For example, if quality is not heritable, then is it a combination of past and present environmental conditions that determine quality?

1.5 Thesis outline

In this thesis, I investigate whether the costs of reproduction are driving variation in fitness-related traits within a population. I largely focus on the two most important life-history traits; fecundity and survival. I use wild birds as a model system to study this, across studies in a meta-analytic context and in an intensive long-term field study using the isolated house sparrow (*Passer domesticus*) population on Lundy Island. The reason for this focus is, first, because birds are globally distributed and have a range of life-history strategies, and, secondly, they have comparatively shorter life spans to our own, meaning that we can study multiple generations over a relatively short period of time. Birds, further, have a distinct reproductive output (an egg) in which a large proportion of the offspring development occurs extrinsically to the mother, meaning that it is relatively easy to, in part, separate genetic and phenotypic effects. In the context of lifespan biology, birds and bats present a distinct comparative group of species showing a longer lifespan than predicted for their body mass and metabolic rate, attributed to their ability to fly, and have attracted considerable interest for this reason alone (Speakman and Król 2010). Students of life-history have studied birds more than mammals, longitudinally as they are relatively easy to observe and can be banded to aid identification. For these reasons, birds are well studied globally, providing a wealth of information, and provide a key model system to study life-history evolution within and between species.

In Chapter 2, I conduct a meta-analysis of the trade-off between two traits closely linked to individual fitness - fecundity and survival - in wild birds. I use data on parental survival given the brood size, of both natural and experimentally altered brood sizes, to separate the costs of reproduction from quality effects. My results show that brood enlargements lead to reduced parental survival but this is directly opposite to the effect of phenotypic quality, where birds who naturally produced larger broods also survived better. I also quantify

parental effort for the brood size raised on a per-egg basis, and standardised the parental effort across species and determined whether the effect was equal across species with different life histories and found that species with small clutches are more severely affected by brood manipulations. I also use the effect size to quantify the fitness constraints on parental clutch size decisions. This shows that the benefit of producing larger brood sizes is offset by increased mortality.

In Chapter 3, I use long-term data from a closed population of wild house sparrows to study why high-quality individuals are not being selected for. I separate the genetic and phenotypic effects of fecundity and survival using genetic pedigree data. I quantify fecundity in various ways: as the number of broods produced in a year, the total annual egg production and the total annual chick production. First, I study age-specific patterns of fecundity and survival and show after an initial increase, fecundity declines throughout an individual's life, however, longer-lived individuals produce more offspring annually. I then use these findings to build a bivariate model of fecundity and survival, which determines whether there is a correlation between these effects in the genetic and permanent environment space. I find no evidence that reproduction is costly in terms of survival and instead provide evidence that variation in reproduction is maintained by quality, which is inconsistent across an individual's life.

In Chapter 4, I study the drivers of body temperature during the nestling period. I first determine whether there is any heritable variation in body temperature at different ages throughout the nestling period. I find small heritable variation in body temperature, though this trend reduced as the nestlings aged. I also explore, through a cross-fostering experiment, whether variation in body temperature, as the individual ages, is driven by the natal environment or by the rearing conditions. I find, after endothermy development, that variation in body temperature is driven more by the natal environment than the rearing environment. I also show that there is a carry over effect of the natal environment.

Subsequently, I go on to test if selection is acting upon body temperature and find there is selection of warmer nestlings which is independent of selection for body mass.

Finally, in Chapter 5, I discuss the conclusions drawn from my findings in this thesis. My thesis presents new quantitative evidence that variation in fitness is not strongly driven by life-history trade-offs as previously thought. I instead provide evidence that it is differences in individual quality that drives variation in fitness, though, individuals are not consistent in quality over their lives.

1.6 Contributions made to this thesis

The basis of this research is a long-term study of the breeding behaviour of a population of house sparrows. Many others have contributed to the collection of this data since its establishment in the 1990's.

My own field work, involving taking thermal images of the nestling house sparrows, was assisted by Photini Knoyle, Elizabeth Larner and Michelle Ross.

Eleanor Wellman and Jake Hogger-Gadsby assisted in extracting body temperature measurements from raw thermal images.

Joel Pick provided important guidance and advice on statistical analysis for the analysis in Chapters 3 and 4.

I confirm that the thesis is otherwise my own work. I am aware of the University's Guidance on the Use of Unfair Means (www.sheffield.ac.uk/ssid/unfair-means). This work has not previously been presented for an award at this, or any other, university.

Chapter 2: The optimal clutch size revisited: separating the effects of individual quality from the costs of reproduction

2.1 Summary

Life-history theory, central to understanding diversity in morphology, behaviour and senescence, states that traits have evolved through optimisation of trade-offs in investment. Despite considerable study, there is only mixed support for trade-offs between the two traits most closely linked to fitness, reproduction and survival, questioning the theory's general validity. Several factors, including variation in phenotypic quality, explain why costs of reproduction might not be apparent. Therefore, we used a meta-analysis to separate the effects of quality from the costs of reproduction across studies of parental investment and subsequent parental survival in birds. Experimental enlargement of broods caused reduced parental survival. However, this effect was small and similar in magnitude to the effect of variation in phenotypic quality, which was directly opposite: parents that produced larger clutches survived better. In addition, the effects of brood manipulation were stronger in species with small clutches, suggesting negative effects on parental survival are only detectable when brood size is forced outside its natural range. Using the estimated effect sizes for parental survival, given the brood size raised, we quantified the fitness consequences of parental decisions about brood size, and revealed a similar constraint: the reproductive benefits of larger clutches exactly offset their parental survival costs, whereas reduced brood sizes caused fitness losses due to lowered reproductive success. Our results provide the first quantitative evidence, across species, that differences in individual quality are the main driver of variation in fitness related traits within a population.

2.2 Introduction

Across taxa, we see a wide variety of life histories, such as in the number of offspring and time spent raising young (Lack 1947; Harvey and Clutton-Brock 1985; Ricklefs 2000a). The central idea in life-history theory is that resources are finite, resulting in trade-offs, meaning that greater investment in one aspect of life requires the sacrifice of another (Stearns 1976; van Noordwijk and de Jong 1986; Kirkwood and Rose 1991; Lemaître and Gaillard 2017). As reproduction is considered to be one of the most energetically demanding life stages, it is expected that within-species variation in offspring production will be driven by the cost of producing and raising young. It is thought that the fitness costs of reproduction are largely incurred as a detriment to survival, also explaining the fast–slow life-history continuum between reproduction and lifespan across species (Kirkwood and Rose 1991). As reproduction and survival are the two components of life-history most closely related to fitness, this central trade-off has been the subject of much theoretical and empirical research.

Even though future detriment to parental survival is the most intuitive and prevalent explanation for why birds do not produce larger clutches, reductions in future reproduction (see Lemaître et al. 2015 for a review of early-late life trade-offs) and offspring quality (reducing the fitness return to breeders) (Gustafsson and Sutherland 1988; Smith et al. 1989; Merilä and Wiggins 1995; Charnov and Ernest 2006) have also been reported. Interestingly, the studies that have quantified the components of fitness in brood size manipulation studies have concluded that only when all fitness costs are combined do these result in balancing selection for the current most common brood size in the population (Daan et al. 1990; Verhulst and Tinbergen 1991). In the field of ageing, life-history theory has been highly influential, as framed in the disposable soma theory, in explaining the relationships between reproduction and ageing. Investing heavily in reproduction should increase

senescence, by reducing investment in combating the negative effects of ageing (Medawar 1952a; Williams 1957; Kirkwood 1977). Conversely, investing too little may lead to a failed or inefficient reproductive attempt and therefore waste resources (Parejo and Danchin, 2006).

Brood size manipulations in birds in natural conditions have provided arguably the best experimental paradigm to test the survival costs of reproduction. Experimental increases in brood size result in increased parental effort, suggesting that parents can typically cope with increased reproductive demands (Roskaft 1985; Lessells 1986; De Kogel 1997; Monaghan and Nager 1997; Fontaine and Martin 2006). The reason why birds do not produce larger clutches is thus not generally one of constraint, but is predicted to have evolved as a trade-off with the consequent fitness costs. However, costs of parental investment are not always detected and the current estimate across studies suggest only a small and variable effect (Reznick 1985; Zera and Harshman 2001a; Santos and Nakagawa 2012; Cohen et al. 2020). A lack of survival costs of reproduction means that costs must arise elsewhere or alternatively that individuals may differ in quality. Individuals may each be operating at their own maximum reproductive effort determined by their phenotypic condition, local or temporal genetic adaptation, and by the surrounding environment (Charnov and Krebs 1974; Pettifor et al. 1988; Wilson and Nussey 2010; Cohen et al. 2020). The relative importance of the trade-off between reproduction and survival – central to life-history theory and the biology of ageing – therefore remains unclear. In addition, the effects of individual quality on this trade-off, although suggested, have not been investigated on a quantitative level (van Noordwijk and de Jong 1986; Descamps et al. 2016; Cohen et al. 2020).

Here, we present a meta-analysis that distinguishes between quality effects and the costs of reproduction. To do this we tested how parental annual survival in birds is affected by the brood size cared for in two different contexts: first in brood manipulation studies and, second, in observational studies of natural variation in clutch size. We expressed changes in

survival on a per-egg basis, which allows for a quantitative comparison across studies. We find that quality is associated with higher survival chances, and that this effect is opposite but equal in magnitude to the costs of reproduction. The survival trade-off for offspring production within a population is therefore offset by differences in quality, potentially constraining the evolution of higher reproductive effort. Our analysis also uniquely allowed a quantitative comparison across species as survival risk was expressed on a per-egg basis. We transformed the response variable, scaling for variance and mean, given that a per-egg increase in clutch size does not equate to the same proportional increase in parental effort for all species equally. Our findings suggest that species that generally lay smaller clutches are affected more severely by brood size manipulations. This provides evidence that trade-offs are only detected when an individual is forced to perform outside its individually optimum level.

To determine the evolutionary consequences of the effect sizes we estimated using meta-analysis, we projected the fitness consequences for a change in brood-size life-history strategy. We found that the effects on parental survival translate into negligible fitness costs, with a relatively flat fitness landscape, suggesting that birds underproduce in terms of brood size, given the absence of fitness costs. This conclusion fits with our comparative finding that suggests only manipulated brood sizes beyond the natural range incur substantial survival costs.

Our results therefore suggest that, in wild populations, parental survival costs are only, at most, a small component of the total fitness costs of investing parental effort. Our results do suggest that a cost of reproduction can be detected when an organism is pushed to the extreme of its reproductive capacity. We therefore infer that, though the survival–parental care trade-off does exist within species, it is too minimal to explain why variation in clutch size is maintained within a population. In addition, our work shows that differences in

individual quality counterbalance the trade-off between survival and reproduction and as such constrain reproductive effort and maintain clutch size variation in a population.

2.3 Methods

We conducted a meta-analysis with the aim of distinguishing between quality effects and reproductive costs when considering within-population variation in clutch size. We tested how parental survival to the following year in birds is affected by the brood size cared for in two different contexts; first, in brood manipulation studies and secondly, in observational studies; we compared these approaches to separate the costs of reproduction from quality effects.

2.3.1 *Study sourcing & inclusion criteria*

We used the following inclusion criteria as set out in Santos & Nakagawa (2012): the study must be on a wild population; the study must detail the effect of variation in the number of raised young (hereafter referred to as clutch size for simplicity) on parental survival to the following year and the study must provide sample sizes. We did not make a requirement for each sex to have been investigated separately and instead included mixed-sex groups, in addition to the separate sexes, where parental survival was reported for both parents combined. Excluded studies and the grounds for their removal are given in the supplementary information (Supplementary Information S2.1).

We firstly extracted data from the brood manipulation studies used in Santos and Nakagawa (2012). We then searched the literature to include studies published after the Santos & Nakagawa (2012) paper (methods described in Supplementary Information S2.2).

In addition to brood manipulation experiments, we extracted data from studies that tested for variation in parental survival in relation to natural variation in clutch size (observational studies). We aimed to pair each species included in the brood manipulation studies with an observational study, to make sure that effects of quality were estimated across a similar range of species, to facilitate a more direct comparison. Where there was no equivalent study in the same species, we attempted to include a study of a congener. In most cases, observational data were obtained from either the same paper as the one describing brood manipulations or via searching for other papers by the same authors. If this failed to produce observational data, a search was conducted following the same protocol as for the brood manipulation experiments, but also specifying species, genus and/or common name in the search. Any additional brood manipulation studies found via this search were also included in the meta-analysis. We ceased the literature search on the 3rd May 2019.

From the literature search, 78 individual effect sizes from 46 papers were incorporated into our analysis (20 observational and 58 experimental studies). While extracting these studies, we also made note of the average (reported as either the mean or mode) clutch size, the within-species standard deviation in clutch size, and the longevity of the species. We first tried to extract this information from the paper containing the study but if the information was missing we then searched other published literature with the aim of finding the information for a comparable population (i.e., similar latitude). Largely this information came from other papers of the same population found by searching the literature of the authors of the original study. If this failed we performed a key-word search in Google Scholar with the species name and “average clutch size” or “longevity” OR “lifespan” respectively.

2.3.2 *Extracting effect sizes*

We used raw data to obtain an effect size by performing a logistic regression to give the log odds ratio for parental survival, given the clutch size (i.e., positive values indicate an increased chance of survival). Clutch size was averaged (mean) if a single estimate of survival was reported for multiple clutch sizes. 'Year' was included as an explanatory variable to correct for between-year variation in adult survival where data were presented for multiple years.

We transformed the explanatory variable (clutch size) in addition to the raw clutch size. This allowed us to directly compare species with different life-history traits. Therefore, we were able to quantitatively compare an increase in parental effort given that a per egg increase in clutch size does not equate to a per unit increase in parental effort for all species equally. We standardised the clutch size by the mean of the species and by the within-species standard deviation in clutch size. For species that have no within-species variance in clutch size, we used a value of 0.01 for the standard deviation in clutch size to prevent issues in calculations when using zero. We also measured the clutch size as a proportion of the mean. We, therefore, have a clutch size measure in three ways; a raw increase in clutch size, a standardised clutch size and a proportional clutch size.

2.3.3 *Meta-analysis*

We ran a single model for each clutch size transformation to determine the cost of survival given an increase in parental effort using the *metafor* package (Viechtbauer 2010) in R 3.3.2 (R Development Core Team 2009). From these models we were also able to directly compare the effect size of brood manipulation studies and observational studies. In these meta-analytic models phylogeny was included to correct for shared ancestry. The phylogeny

included was obtained using BEAST to measure a distribution of 1000 possible phylogenetic trees of the focal 30 species extracted from BirdTree (Rubolini et al. 2015). We also included species, phylogeny and the studies' reference as random effects in the model. From these models, we calculated the proportion of variance explained by the phylogenetic effect.

We then tested the effect of the species average clutch size on the effects of the relationship between parental survival and clutch size. We ran a single model with species average clutch size as an interaction with treatment (brood manipulation or observational). The clutch size was adjusted by the combined average clutch size of all species used in the meta-analysis subtracted from the species mean clutch size for each study. Species and reference were also included as random effects to correct for similarity of effect sizes within species and studies.

The difference in survival for the different sexes was modelled for each clutch size measure. Brood manipulation studies and observational studies were analysed in separate models. Sex was modelled as a categorical moderator (41 female studies, 27 male studies and 10 mixed studies). Species and reference were random effects, and a phylogenetic correlation was included in the model. The results of this can be found in Supplementary Information S2.3 and S2.4.

2.3.4 *Publication biases and heterogeneity*

Much of the data used in this analysis were taken from studies where these data were not the main focus of the study. This reduces the risk that our results are heavily influenced by a publication bias for positive results. A funnel plot for the survival against raw clutch size model can be found in Supplementary Information S2.5.

We found significant heterogeneity in all our models ($p<0.0001$).

2.3.5 *Isocline analysis*

We calculated various isoclines from the brood manipulation results in our raw clutch size meta-analysis. Here, an isocline is a trendline representing the change in fitness returns given an increase in individual clutch size. We present this for a range of life-history characteristics (survival rate and average clutch size), a varying severity of manipulation and at a range of effect sizes observed in our meta-analysis. The aim of this was to investigate why an individual may be constrained to their own optimal clutch size.

An estimated lifetime reproductive fitness was calculated for hypothetical control populations, where all individuals consistently reproduce at the level of a species mean and have a consistent survival rate. We used species average clutch sizes to be 2, 4, 6, 8 and 10 and survival rates of 0.2, 0.3, 0.4, 0.5, 0.6 and 0.7, which represent the range of clutch sizes and survival rates seen in the species in our meta-analysis.

This was then repeated to give a lifetime reproductive success for a hypothetical population that reproduces at an increased level compared to control, replicating brood manipulations. To do this we added a range of 1-5 offspring to the clutch sizes of the control populations. Using a range of increased clutch sizes allowed us to investigate how the severity of the manipulation affects lifetime fitness. We also adjusted the survival rates for these hypothetical populations using the effect size found for brood manipulation studies in our raw clutch size meta-analysis model, which reports the effect on survival of increasing clutch size by one. For this, we used an effect size of -0.25, -0.15 and -0.05, which represent the lower confidence interval, the estimate of the overall effect size and the upper confidence interval

respectively (all were rounded to the closest 0.05 for simplicity). To understand this in a biologically relevant context, for each clutch size, a survival rate was taken from predictions based on the species that reproduce at these levels used in our meta-analysis. We used one effect size for adjusting the survival rates for each of the hypothetical “brood manipulation” populations, which was extracted from predictions of species who reproduce at each clutch size.

We then calculated the selection differential ($LRS_{brood\ manipulation}$ - $LRS_{control}$) between the hypothetical control and “brood manipulation” populations for each variation of survival rate, clutch size and effect size across a range of brood manipulation severity.

2.4 Results

2.4.1 *Survival costs of parental effort*

The relationship between clutch size and survival was significantly different and opposite between observational and brood manipulation studies (p = 0.0007, Figure 2.1A, Table 2.1). Within observed natural variation, parents with larger clutches had increased survival. In contrast, when broods were experimentally manipulated, the opposite relationship was found: parents with increased brood sizes exhibited decreased survival. However, across the species included in this meta-analysis, the average clutch size ranges from 2 to 11. The parental effort required to raise two instead of one chick is potentially doubled, whereas one additional chick in a brood of 11 is likely to require only a marginal increase in effort. The overall effect sizes for the brood manipulation studies were non-significant. Only when risk was expressed as a proportional increase did the effect become significant (Figure 2.1c, Table 2.1).

Irrespective, however, of how clutch size was expressed, the relationship between clutch size and parental survival was significantly opposite for observational and brood manipulation studies (Table 2.1, $p < 0.01$). Males and females did not differ in their survival response to changing clutch size (Supplementary Information S2.3 and S2.4, contrary to Santos & Nakagawa 2012).

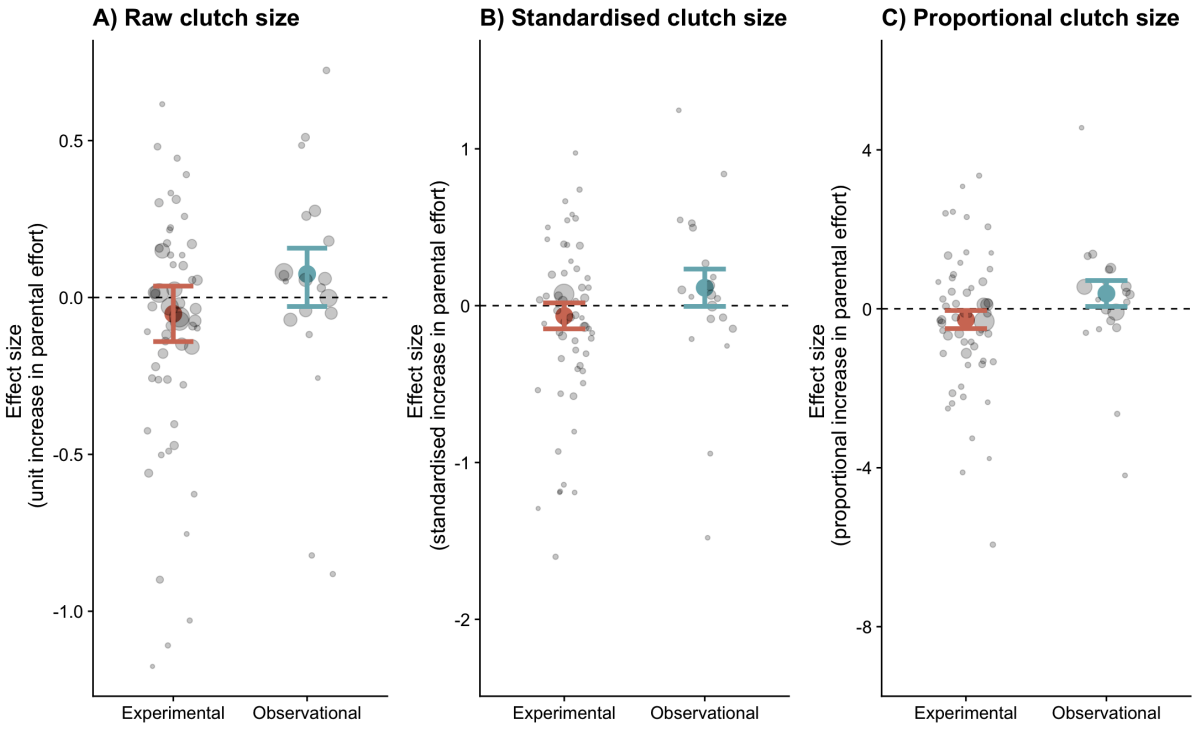


Figure 2.1: The effects size (odds of survival) for three different measures of clutch size: (A) raw, (B) standardised, and (C) proportional clutch size. Coloured points are the combined effect size of the odds ratio for all the studies. Error bars are the 95% confidence intervals. Points are coloured by whether they represent brood manipulation experiments or observational (natural variation in clutch size). Individual effect sizes are considered significant if the 95% confidence intervals do not overlap zero. Grey underlying points are the odds ratio of each study with their size weighted by the variance.

Table 2.1: Effect size estimates from meta-analyses for the odds of survival with increasing clutch size (raw, standardised and proportional clutch size as separate models). Each model included 78 individual effect sizes (20 observational and 58 brood manipulation studies). The p-values indicate the difference between brood manipulations and observational data, with the individual effect p-values in parentheses.

	Parameter	Effect size	95% l.CI	95% u.CI	*p*	(individual)
Raw	Brood manipulation	-0.05	-0.14	0.04	**0.0007**	(0.25)
	Observational	0.07	0.16	-0.03		(0.16)
Standardised	Brood manipulation	-0.07	-0.15	0.02	**0.0065**	(0.12)
	Observational	0.11	0.00	0.23		(0.06)
Proportional	Brood manipulation	-0.27	-0.50	-0.04	**0.0005**	**(0.02)**
	Observational	0.39	0.06	0.71		**(0.02)**

The variance assigned to the random effects in the model was largely accounted for by the reference of the study (Table 2.2). Species accounted for more variation than the phylogenetic signal, indicating that species vary in their survival for their brood size raised but potentially, the phylogeny we present has few closely related species (Supplementary Information S2.6). For this reason (i.e., few closely related species), we also observed variation being assigned to either phylogeny or species changing with the clutch size transformation.

Table 2.2: I^2 values for each model showing the proportion of variation accounted for by the random effects of the model. The phylogenetic signal was also a correlation matrix within the model.

Model	I^2				
	Total	Species	Phylogenetic	Reference	Total species effect (Species + Phylogenetic)
Raw	0.49	0.000000003	0.29	0.21	0.29
Standardised	0.54	0.08	0.00000002	0.46	0.08
Proportional	0.43	0.14	0.00000001	0.29	0.14

2.4.2 *Species differences*

A per-egg increase in clutch size is a proportionally greater test of increased quality in species that lay fewer eggs (Figure 2.2 and 2.3, Table 2.3). When clutch size is experimentally manipulated, there is little variation between species in how their survival is affected by an increase in clutch size (Figure 2.2 and 2.3, Table 2.3).

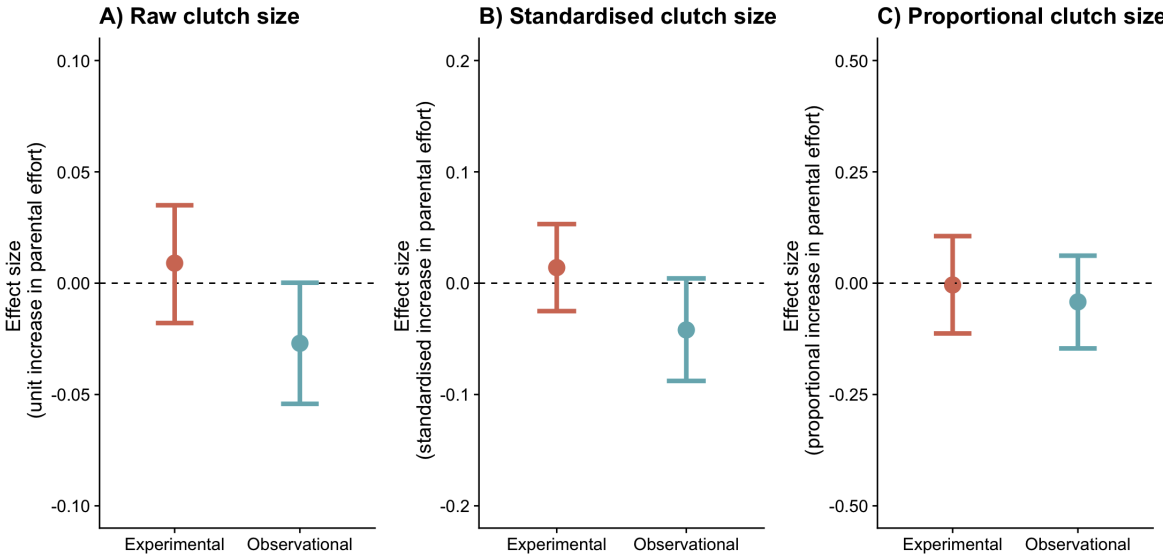


Figure 2.2: The odds of survival for an increasing clutch size (i.e., increase in parental effort) given the mean clutch size of the species. The model used the A) raw (+1 egg), (B) standardised and (C) proportional clutch size measure for the odds of survival. Points are the combined effect size with 95% confidence interval error bars. If the 95% confidence intervals overlap zero, the trend is considered non-significant.

Table 2.3: Model outputs for meta-analyses estimating the effect size of the odds of survival for increasing clutch size (each clutch size estimate is an individual model) given the species average clutch size. 20 observational studies and 58 brood manipulation studies were used in the meta-analyses. The species average clutch size was centred to the average clutch size of all species used in the meta-analysis.

Model	Parameter	Effect size	95% CI lower	95% CI upper	p-value
Raw	Intercept	-0.05	-0.15	0.05	0.36
	Treatment: Observational	0.15	0.08	0.22	**<0.0001**
	Centred species clutch size	0.01	-0.01	0.03	0.34
	Treatment: Observational x Species clutch size	-0.04	-0.07	-0.01	**0.02**
Standardised	Intercept	-0.07	-0.22	0.09	0.42
	Treatment: Observational	0.20	0.07	0.33	**0.002**
	Species clutch size	0.02	-0.03	0.06	0.48
	Treatment: Observational x Species clutch size	-0.06	-0.12	0.00	0.06
Proportional	Intercept	-0.23	-0.72	0.25	0.34
	Treatment: Observational	0.62	0.24	1.01	**0.002**
	Species clutch size	0.03	-0.09	0.14	0.68
	Treatment: Observational x Species clutch size	-0.10	-0.25	0.06	0.21

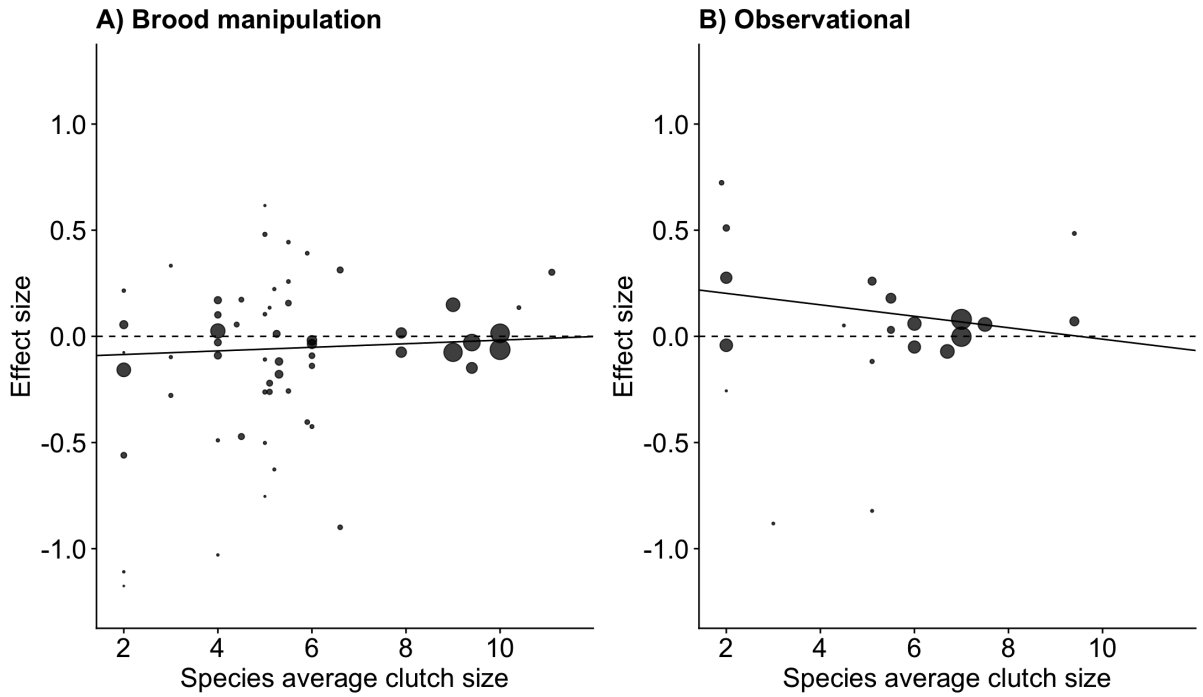


Figure 2.3: The linear regression of the effect of increasing clutch size on parental survival given the average clutch size of the species. The points are the effect size of increased clutch size on parental survival, with the point size reflecting the meta-analytic weights of that study.

2.4.3 *Isoclines of selection differentials*

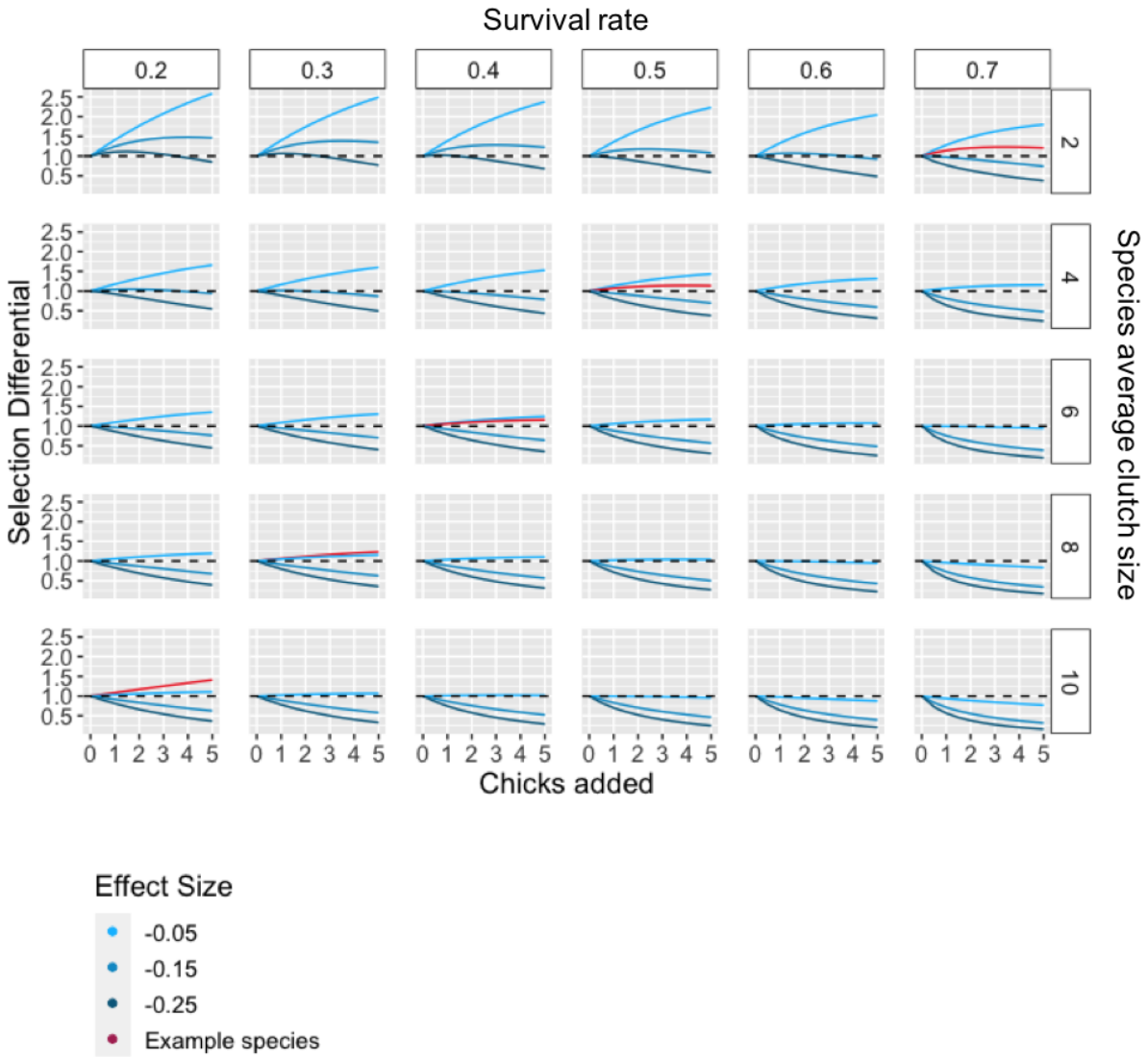
For species that lay fewer eggs and that generally have higher survival rates, the benefit of laying larger clutch sizes is largely offset by the cost of survival, even when the clutch size is increased well beyond what the species is capable of raising (Figure 2.4). The benefits of laying larger clutch sizes would only become apparent if these species had much lower survival rates.

Species with larger clutch sizes similarly have the benefit of laying larger clutches mostly offset by the increased cost of lower survival (Figure 2.4). A higher survival rate increases

the severity of the cost in survival, meaning that larger clutch sizes combined with increased survival rates produces overall reduced fitness.

For all these “example species”, which represent biologically relevant combinations of the variables, the selection differential was observed to lie slightly above one, indicating that, across a bird's life, if they were to increase their clutch size they would gain a slight fitness benefit. The benefit was generally consistent over the range of added chicks.

Figure 2.4: Isoclines of selection differentials among hypothetical control populations (in which individuals reproduce at the species average) and hypothetical brood-manipulated populations (where individuals reproduce at an increased rate compared to control). Survival rates, clutch sizes, the magnitude of the manipulation (chicks added) and effect sizes represent the range of these variables present in the studies used in our meta-analysis. For each clutch size, we used a predicted survival rate and effect size to give isoclines that are biologically meaningful (example birds shown in red).



2.5 Discussion

Our results provide quantitative evidence that it is differences in individual quality that drive variation in clutch size, as demonstrated by individuals that lay larger clutch sizes naturally having higher survival. Here, we are defining quality as a combination of traits that give an individual higher fitness and so the underlying mechanisms for determining quality are yet to be fully investigated. Furthermore, we found the same effect when taking into account the fact that a per-egg increase in clutch size is not an equal investment increase across species (individuals who naturally lay larger clutch sizes have higher survival). It should be noted that it is possible that the quality effect could be representative of a terminal effect, where individuals have lower reproductive output in the year preceding their death and thereby driving the trend for naturally lower laying birds to have lower survival (e.g., Coulson and Fairweather 2001; Rattiste 2004; also see Hammers et al. 2012 for age related changes in reproductive output).

The effect of naturally larger clutch laying birds was significantly opposite to the result of increasing clutch size through brood manipulation, demonstrating that brood manipulation experiments do not perform their intended function to test whether an individual trades off reproduction with self maintenance. Instead, brood manipulations are more likely to be a test of the effects of a shift away from an individual's optimal clutch size. We have, therefore, provided evidence of the van Noordwijk & de Jong (1986) framework - that the classic trade-off between adult survival and the clutch size cared for is only apparent when an individual is forced to raise a clutch outside of its individual optimum.

For the species that, on average, naturally laid fewer eggs, a per-egg increase more severely influenced survival than in species that laid more eggs. This may reflect the constraint that small clutch-laying species face, where a severe increase in parental effort is

required in order to raise an increased clutch size and, consequently, those individuals that produce larger clutches than their counterparts need to be of notably higher quality. We found little evidence for variation among species in the severity of the effect of increasing clutch size by manipulation on survival.

It is possible that although individuals pay a survival cost when their clutch size is manipulated to be larger, the benefit of having larger clutch sizes means their overall lifetime fitness is increased despite a shortened life. However, we found that individuals with larger clutch sizes only experience a marginal increase in fitness and, furthermore, no additional benefits were found when increasing the clutch size more severely. Nevertheless, this still raises the question of why larger clutch sizes are not selected for. It is possible that the identified fitness gains represent a trade-off that we have not explicitly tested here, such as in offspring quality (e.g., Smith et al. 1989; Conrad and Robertson 1992), parental condition other than survival (e.g., Reid 1987; Kalmbach et al. 2004) or future reproductive effort (e.g., Järvistö et al. 2016). Interestingly, the studies that have measured these different domains that contribute to fitness in brood-size manipulation studies concluded that only in combination do these costs result in balancing selection for the current most common brood size in the population (Daan et al. 1990; Verhulst and Tinbergen 1991). It could, therefore, be that the costs of reproduction remain unexplained because the costs are distributed over multiple traits. It is also possible that the traits that make up an individual's quality are not constant. It may be that spatially and temporally heterogeneous environments cause oscillating selection (i.e., variation in phenotypic quality). However, despite being largely accepted as the reason within-population variation is maintained (discussed in Cohen et al. 2020), we have found no evidence that individuals trade-off self-maintenance against parental effort.

2.6 Supplementary Information

S2.1: Excluded studies and the rationale for exclusion.

Reference	Reason for exclusion
Ashcroft, 1979	No clutch or brood size given
Erikstad et al, 2009	No clutch/brood size variation
Wernham & Bryant, 1998	No clutch/brood size variation
Wiebe, 2005	Mate removal, not clutch/brood manipulation
Askenmo, 1979	Doesn't state manipulation size
Tinbergen & Both, 1999	Manipulation is to equalise brood size throughout population
Annett & Pierotti, 1999	Breeding lifespan not survival
Murphy 2007	No survival values given
Lessells, 1986	No clutch or brood size given
Schaub & Hirschheydt, 2009	Clutch size groups too large
Milonoff & Paananen, 1993	Clutch size before manipulation varies significantly
Blondel et al, 1998	No standard deviation reported for survival
Knowles, Wood & Sheldon, 2010	No standard deviation reported for survival
Kluyver, 1970	Combined first and second broods

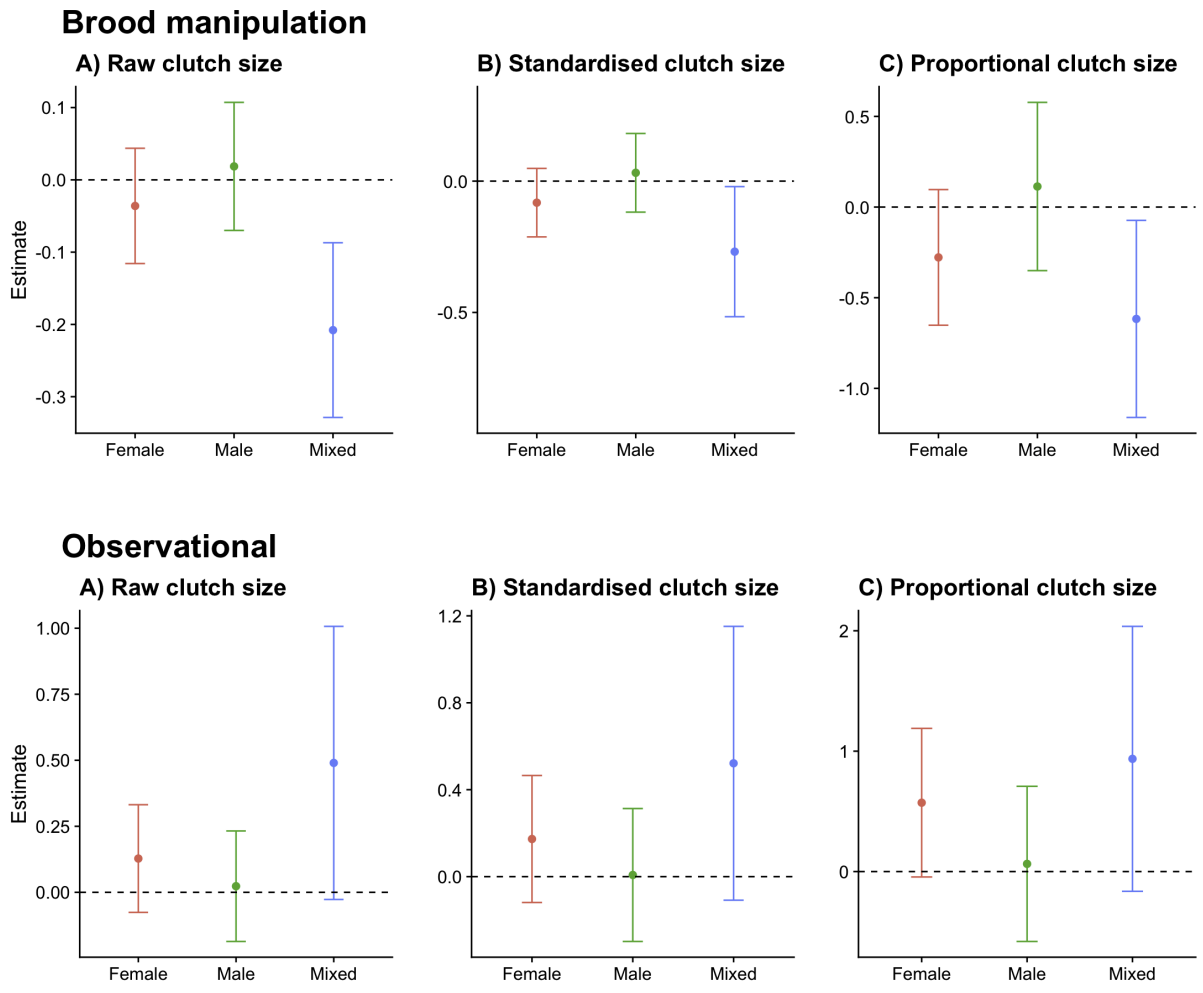
S2.2: Method details of brood manipulation data extraction

Firstly, we re-extracted the raw parental survival values for each given clutch size from the studies. This gave a continuous scale of clutch size, as opposed to Santos & Nakagawa (2012) who compared both brood increases and brood reductions, irrespective of the size of these manipulations, as combined categories to the control category of a study. We then expanded the number of studies by also including mixed-sex studies, where survival returns were combined for both parents, and included studies published in the years following publication of the Santos & Nakagawa (2012) paper upto the 3rd May 2019. To do this we used a key word search on Web of Science and Google Scholar using the following terms: “longevity” OR “lifespan” OR “survival” AND "breeding success" OR "brood size" OR "clutch size" OR "number of chicks" OR "number of eggs" AND "trade-off" OR "trade offs" AND fitness AND life-history AND avian OR bird OR birds OR ornithology.

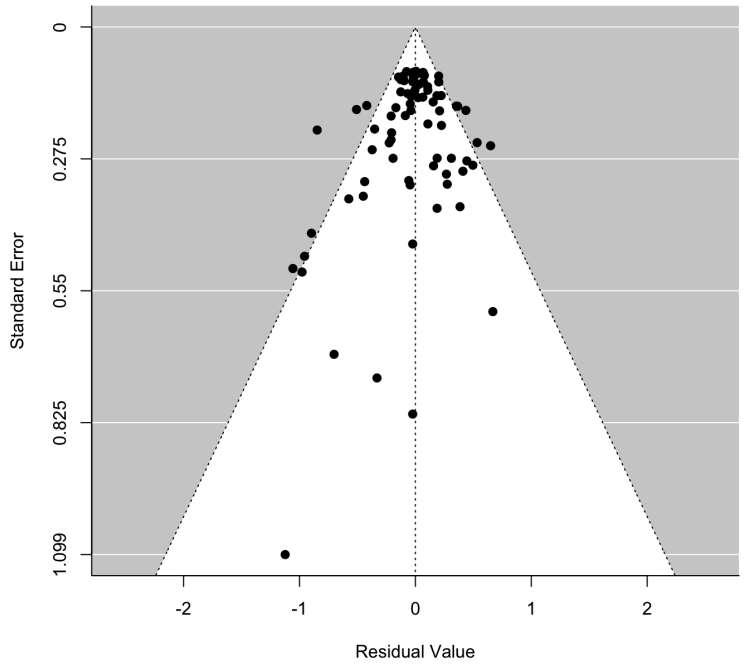
S2.3: Model outputs for survival given increasing clutch size for brood manipulation and observational studies for the different sexes (n_{female}= 41, n_{male}= 27, n_{mix}= 10). Mixed sex studies were found to be at the extremes of the trend, a reflection of species who lay smaller clutch sizes rather than an effect of the mixed sex itself.

Clutch size measure		Sex	Estimate	SE	*p*	CI.lb	CI.ub
Raw	Brood manipulation	Female	-0.04	0.04	0.38	-0.12	0.04
		Male	0.02	0.05	0.68	-0.07	0.11
		Mixed	-0.21	0.06	**0.0007**	-0.33	-0.09
	Observational	Female	0.13	0.10	0.22	-0.08	0.33
		Male	0.02	0.11	0.83	-0.19	0.23
		Mixed	0.49	0.26	0.06	-0.03	1.01
Standardised	Brood manipulation	Female	-0.08	0.07	0.22	-0.21	0.05
		Male	0.03	0.08	0.68	-0.12	0.18
		Mixed	-0.27	0.13	**0.03**	-0.52	-0.02
	Observational	Female	0.17	0.15	0.25	-0.12	0.47
		Male	0.01	0.16	0.96	-0.30	0.31
		Mixed	0.52	0.32	0.10	-0.11	1.15
Mean adjusted	Brood manipulation	Female	-0.28	0.19	0.15	-0.65	0.10
		Male	0.11	0.24	0.63	-0.35	0.58
		Mixed	-0.62	0.28	**0.03**	-1.16	-0.07
	Observational	Female	0.57	0.31	0.07	-0.04	1.19
		Male	0.06	0.33	0.85	-0.58	0.71
		Mixed	0.94	0.56	0.10	-0.16	2.04

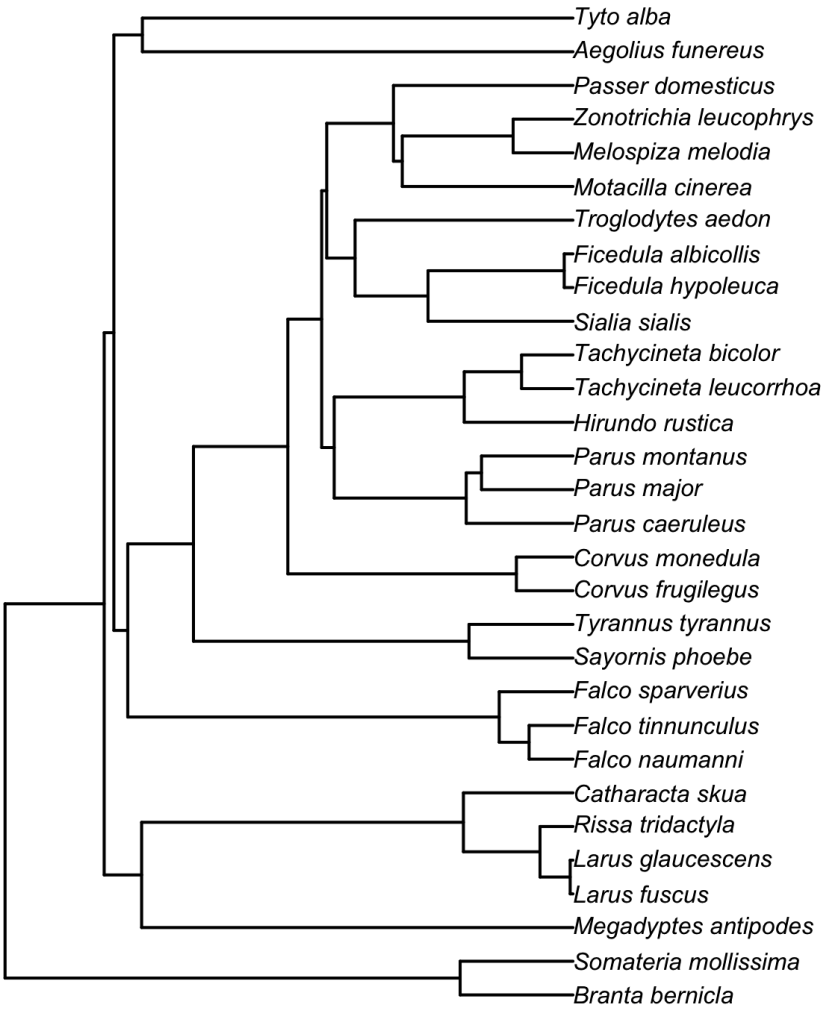
S2.4: Survival effects for increasing clutch size for female (n = 41), male (n = 27) and mixed sex (n = 10) studies. The clutch size was measured in three ways; raw clutch size, standardised and mean adjusted. Separate meta-analyses were run for observational and brood manipulation studies. Points are the combined effect size and whiskers are the 95% confidence intervals.



S2.5: Funnel plot of meta-analysis residuals against standard error. Brood manipulation and observational data are combined.



S2.6: Phylogenetic tree of species included in our meta-analysis.



Chapter 3: The relationship between fecundity and survival in separating the costs of reproduction from quality effects in a wild bird

3.1 Summary

There is mounting evidence to suggest that intraspecific variation in reproductive success is affected by differences in quality between individuals whereas life-history trade-offs are still seen as fundamental to understanding biology. Indeed, if quality is heritable, it is surprising that individuals who are of higher quality are not selected, which would lead to all individuals within a population reproducing at the population mean, removing between individual variance in reproductive success. We used a long-term dataset of breeding records for a closed population of house sparrows (*Passer domesticus*) and its associated pedigree to distinguish between genetic and environmental effects on the relationship between fecundity and survival. We measured age-specific changes in reproductive outputs and estimated the heritability of both offspring production and survival in multivariate animal models. Our results show no evidence for survival costs to reproductive output. Nor did we find any evidence that individuals' fecundity and survival are correlated either in genetic or permanent environmental space. We only found negligible heritability of fecundity and a small between-individual effect on fecundity. We found no heritability or between-individual effect on survival. Our findings show no evidence that variation in fecundity is driven by a trade-off between offspring production and survival. Instead, our results suggest that individuals are inconsistent in their quality relative to the population, preventing selection acting on higher quality individuals.

3.2 Introduction

Theory suggests that, as resources are limited, trade-offs in resource allocation will cause variation between individuals in their life histories, such as in fecundity, offspring quality and individual survival (Kirkwood & Rose, 1991; Lemaître & Gaillard, 2017; Stearns, 1976; van Noordwijk & de Jong, 1986). However, there is little evidence for the reduction in performance of traits that is expected as a result of increased allocation towards reproduction (Reznick 1985; Zera and Harshman 2001b; Santos and Nakagawa 2012; Cohen et al. 2020). There is now growing evidence to suggest that the reason why individuals reproduce at different rates is largely due to differences in individual quality (Chapter 2; Pettifor et al. 1988; McLean et al. 2019; Cohen et al. 2020). It is currently unknown to what extent these quality differences are genetic or a result of the environment (McCleery et al. 2004). As environmental effects can obscure genetic variation, by separating these directly in a quantitative genetics framework it is possible to identify the otherwise elusive costs of reproduction.

In genetic space, high quality individuals are those who are genetically predisposed to have a higher fitness in relation to the population. It would be expected that these individuals are selected. Estimating the genetic component of fitness associated traits requires lifetime observations of individual fecundity and survival, data for which are challenging to collect in wild populations. Furthermore, molecular methods have only relatively recently become widely available for measuring genetic variation. It has been shown that individuals who produce more offspring also have higher survival (Chapter 2). However, should genetic quality predict both reproductive fitness and survival, then selection should favour individuals who reproduce at a higher rate and/or survive for longer. In this scenario, we would expect little or no variation between the genetically-determined component of reproductive performance of individuals within a population, as all individuals should be selected towards

reproducing at the population mean (Fisher 1958). Heritability, however, has been well documented for fecundity-related traits, showing that additive genetic variation persists (Kruuk et al. 2000; Teplitsky et al. 2009b; Brommer et al. 2010; Schroeder et al. 2012). By contrast, there has been mixed evidence of heritability of survival or longevity (heritability of lifespan in males but not females, Merilä and Sheldon 2000; no heritability of longevity, Kruuk et al. 2000; no heritability of longevity, Schroeder et al. 2012; heritability of lifespan, Vedder et al. 2021). Whether individual quality, through both increased fecundity and survival, is heritable therefore warrants investigation.

Phenotypic quality can be considered as a collection of traits that make an individual well suited to particular situations and/or environmental conditions but that are detrimental in others. In this scenario, variation in reproductive output between individuals would be maintained as selection pressures are inconsistent over time or similarly, if an individual has benefitted from favourable early-life conditions, they may perform better without necessarily having a genetic predisposition to higher fitness and survival. Laboratory studies of genetically identical *Caenorhabditis elegans* demonstrate variable lifespans (Kirkwood and Finch 2002; Sánchez-Blanco and Kim 2011). Phenotypic variation is also driven by more than current environmental variability; mechanisms such as mutation, early-life conditions, oxidative damage, injury and stochasticity also cause between-individual variation in survival (Kirkwood et al. 2005; Hamel et al. 2009b). There are several studies that provide evidence that an individual's environment shapes its life-history trajectory and, thereby, leads to different phenotypic quality between individuals, for example: manipulation of early-life conditions (Alonso‐Alvarez et al. 2006; Spagopoulou et al. 2020), lifetime manipulation of environmental conditions (Marasco et al. 2018b), and observational evidence of varying environmental conditions (Pettorelli et al. 2001).

The complexities of fitness related traits require state-of-the-art modelling approaches to separate genetic and permanent environment effects while controlling for other factors that are known to affect an individual's fitness, such as its age. Fitness outputs are confounded with the effects of ageing. It was previously thought that wild animals will not experience senescence as external factors, such as infection or predation, will prevent an individual living to its maximum potential age (Kirkwood and Austad 2000). However, there are now many well documented cases of senescence in the wild (Monaghan et al. 2008; Nussey et al. 2008; Ricklefs 2008; Schroeder et al. 2012). Iteroparous species, such as passerines that begin breeding in the first year after birth and then throughout their lives, typically show a pattern of ageing where reproductive output increases in the first few years of life then declines later (Bouwhuis et al. 2010; Hammers et al. 2012). It is argued that an individual's investment priorities will change over a lifetime. For example, the disposable soma theory suggests that an individual increases its reproductive output to the detriment of somatic repair, resulting in late-life senescence (Kirkwood 1977). The pattern of ageing is also influenced by the environment throughout an individual's lifespan through age-specific changes in resource allocation (Schroeder et al. 2012; Marasco et al. 2018b).

To our knowledge, no study has disentangled the genetic and environmental effects on individual quality. In this study, we explore evidence for correlations between fecundity and survival in genetic and permanent environmental space. By doing so we explore whether there is a cost to reproduction in terms of survival or whether individuals are predisposed to producing more offspring and surviving for longer. This work sheds light on why between-individual variation in fitness is maintained.

3.3 Materials and Methods

3.3.1 *Data collection*

Data were collected on a population of house sparrows *Passer domesticus* on Lundy Island in the Bristol Channel (51º10'N, 4º40'W). The population has been closely monitored since 1996 but for the purpose of this analysis we excluded data from before 2001 to ensure that all records are from birds of known age and that the majority of the population is known (>99%). Individuals are ringed with a BTO metal ring, a unique combination of colour rings and are fitted with a passive-integrated transponder as either nestlings or fledglings, meaning that precise ages are known for all individuals. Nests were monitored throughout each breeding season, so that reproductive outputs for the majority of breeding attempts are known. As sparrows do not undertake long-distance flight, the population is considered to be closed to immigration and emigration (Schroeder et al. 2015). Therefore, we also have an accurate estimation of when an individual dies. This information means we have complete life histories at an individual level. All individuals had a blood sample taken from either nail clipping or from the brachial vein (all procedures were performed under UK Home Office licence). The genetic pedigree was assembled using 13 microsatellite loci to identify genetic parents (Dawson et al. 2012).

3.3.2 *Fitness measures*

We exclusively used female breeding information in this analysis as it has previously been shown that male parent survival is largely unaffected by the clutch size raised, probably because clutch size is determined by the female (Chapter 2). The fitness components used in this analysis were the annual number of broods produced and the total annual number of eggs produced (see Results and Supplementary Information for other fitness proxies). If a bird was known to be alive in a year but had no breeding record, its fecundity was recorded

as zero unless it was the last year in which a bird was known to be alive, in which case fecundity was recorded as a missing value.

3.3.3 *Data analysis*

All analyses were performed using R version 3.6.2 (R Development Core Team 2009).

Mortality risks relative to offspring production were calculated using Cox proportional hazards models using the coxme package (Therneau 2020). Survival was a binary response variable indicating whether the bird survived to the following calendar year or not. The total number of eggs produced was a fixed effect and the focal year was a random effect in the model. We repeated the same model analysis but this time with death ascribed to the year prior to the last breeding attempt, thereby removing the influence of any terminal effect (i.e., where reproductive output is reduced in the final year in which an individual is alive) on mortality risk.

We ran separate univariate models of (i) annual fecundity and (ii) annual survival in order to test for evidence of genetic variance of the traits using MCMCglmm version 2.29 (Hadfield 2010). (i) We included age represented as the change in age (Δ age = age - individual average age), its squared term and individual average age (i.e., the mean of the ages an individual reaches over its lifetime) as covariates in a logit link MCMC mode (van de Pol and Wright 2009). The permanent environmental effect and genetic effect were also included as random effects in the model. The genetic effect is a matrix for the additive genetic relationship between individuals, determined by the pedigree. The focal year was also included as a random effect. (ii) Survival probability was analysed using MCMCglmm, where survival was estimated using an approximation of the Cox Proportional Hazards model (see Chenyang Zhong et al. 2019). Age was expressed as a categorical fixed effect. The

between-individual effect and genetic effect were included as random effects. The focal year was also included as a random effect.

We ran bivariate models of fecundity and survival using MCMCglmm. Fecundity was measured as (a) the total annual number of broods produced and (b) the total annual number of eggs produced and was normally distributed. Survival was again estimated using an approximation of the Cox Proportional Hazards model, using a Poisson distribution. Age and its quadratic term were fixed effects for fecundity. We also ran models both with and without the terminal effect as a fixed effect. We removed the categorical age effect for survival, as survival did not vary significantly between age groups and removing the effect did not change the conclusions drawn from our results. We modelled the permanent environmental effect, genetic effect (estimated by the pedigree) and focal year effect as random effects in the model. We repeated this analysis but for the fecundity in the final year we removed any fecundity values and instead included these as missing information if the bird was known to have a complete breeding season in the final year, thereby determining if the terminal effect was the result of selective disappearance. Survival through a complete breeding season was determined by whether a bird had an active nest in August or was seen in that or a later month in its last breeding year. Sightings of individuals outside of the nest are largely derived from mist netting of adults and juveniles in early winter.

For analyses in MCMCglmm, the burn-in period was 60,000, the chain length was 460,000 iterations and the thinning interval was 200. For the random effects, parameter expanded priors were used. For residual variances, inverse wishart priors were used (V =1 and nu =0.002 for the univariate models, and V= diag(2) and nu = 1.002 for the bivariate models).

3.4 Results

There was no clear response to selection acting on fecundity within the population as fecundity showed no trend of increasing or decreasing over the course of the study of the population (Figure 3.1).

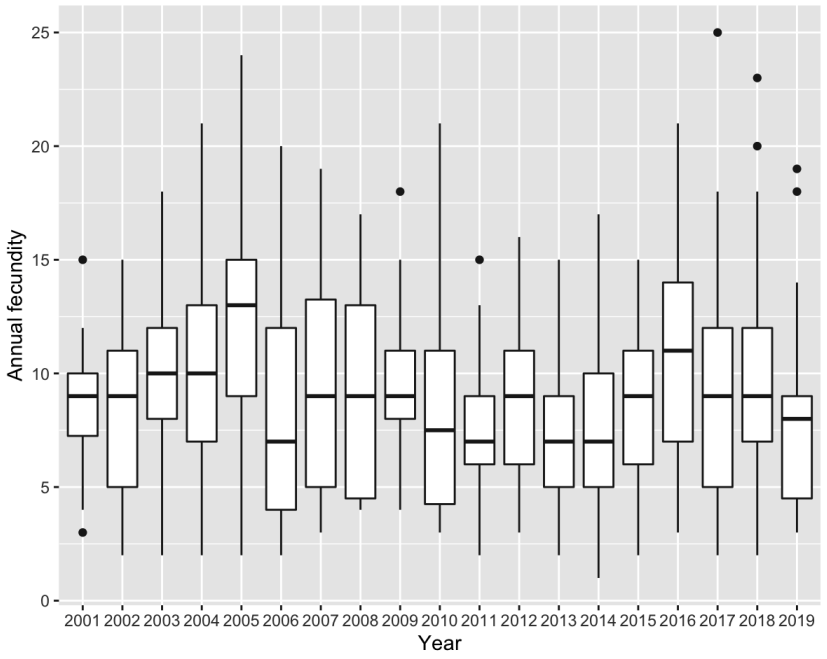


Figure 3.1: Total annual fecundity (eggs produced) for individual females in each year of the study. Boxes are first and third quartiles with medians as the horizontal lines, and whiskers represent the data to the lowest and highest observation within 1.5 times the interquartile range.

3.4.1 *Mortality risk given offspring production*

First, we explored whether there was a detectable relationship between fecundity and survival. We found that individuals that laid larger clutch sizes had a reduced mortality risk (Table 3.1). When the last year of reproduction was discounted, the trend remained negative

and of similar magnitude to when it was included, but became non-significant, indicating that reproduction is particularly reduced in the last year a bird is alive.

Table 3.1: Results of Cox hazards models for mortality given offspring production (as total eggs produced in a year). 960 observations from 495 individuals were used in this analysis. Mortality is defined separately in the reported models as the last reproductive year and the year prior to the last reproductive year. The latter removes any terminal effect present in the last breeding year.

		coef	exp(coef)	SE	*z*	*p*
Mortality is last reproductive year	Offspring production	-0.032	0.968	0.013	-2.46	**0.014**
Mortality is year prior to last reproductive year	Offspring production	-0.021	0.980	0.022	-0.94	0.35

3.4.2 *Age-related changes in offspring production*

After an initial increase, as an individual grows older, fewer offspring are produced per annum; however, it is birds that live longer that produce more offspring annually that drive this pattern (Figure 3.2, Table 3.2). Within-individual changes with age follow the commonly found bell-shaped pattern with age, with offspring production starting and accelerating from the first year of breeding but levelling off in mid-late life. We found similar age-related fecundity patterns when using different fitness proxies (number of chicks or number of fledglings), but the number of recruits produced did not vary over the course of an individual's lifetime (Supplementary Information S3.1 and S3.2).

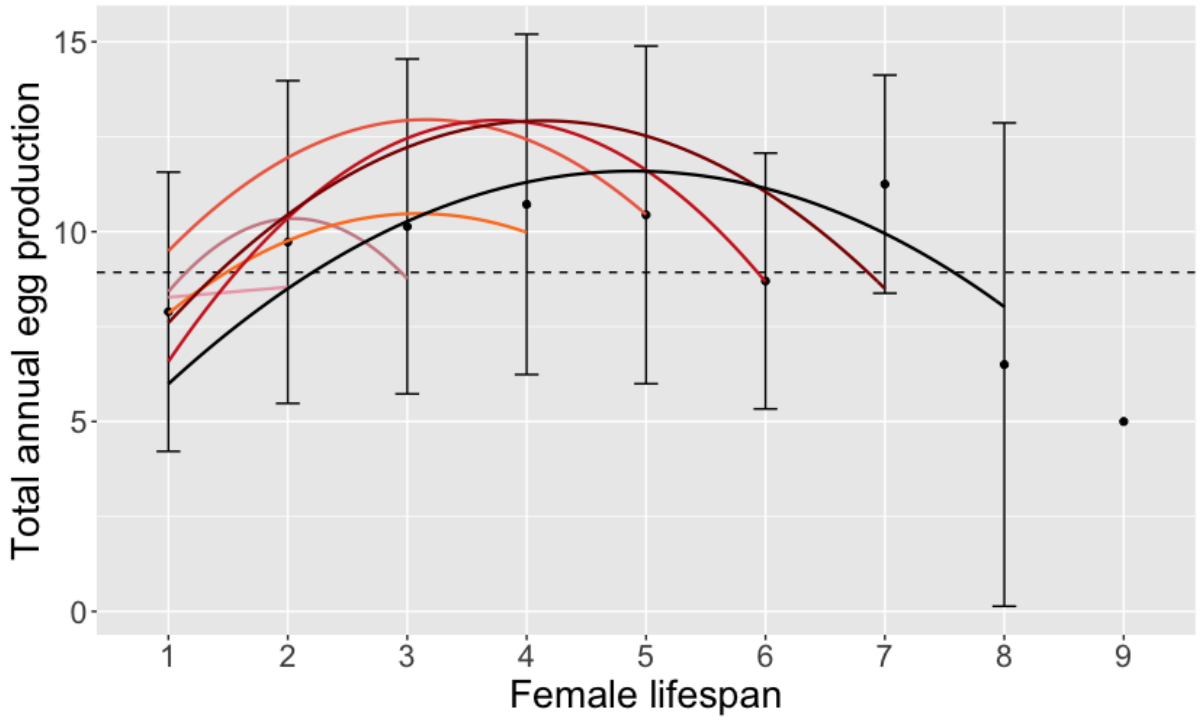


Figure 3.2: Total annual offspring production (number of eggs produced) given the age of the female parent. Points are the population mean offspring production for the given lifespan. Whiskers are the standard deviation from the mean. The curves represent the within-individual change in offspring production as females age for different lifespans observed.

3.4.3 *Heritability of fecundity and survival*

We found the heritability of annual egg production to be 0.06 (95% CI: 0.00-0.11) (Table 3.2). We also found a permanent environment effect on annual fecundity (Individual ID; posterior mean = 3.06). The repeatability of the permanent environmental effect was 0.18 (95% CI: 0.09-0.21).

Table 3.2: Model outputs for a univariate animal model of annual fecundity (n = 996 observations from 506 individual females). Annual fecundity is total annual eggs produced by a female.

	Posterior mean	95% CI - lower	95% CI - upper	pMCMC	Variance components
Fixed:					
(Intercept)	6.20	5.16	7.18	<0.001	
Δ age	0.31	0.07	0.56	0.022	
Average age	1.41	1.04	1.76	<0.001	
Δ age^2	-0.50	-0.68	-0.30	<0.001	
Random:					
Between-individual	3.06	1.02	4.73		0.18
Genetic	0.99	0.00	2.48		0.06
Focal year	1.63	0.52	3.03		0.10
Residual	11.02	9.73	12.32		0.66

We found no evidence of heritability of survival (h^2 = 0.008 (95% CI: 0.00-0.01), Table 3.3). Survival did not differ significantly between age groups. Focal year explained the most variance in survival probability.

Table 3.3: Model outputs for a univariate model of survival probability. Survival was estimated for each age using an approximation of the Cox proportional hazards model. 1144 observations were used from 507 individuals for this analysis.

	Posterior mean	95% CI - lower	95% CI - upper	pMCMC	Proportion of variance
Fixed:					
Age 1	-0.74	-1.09	-0.46	<0.001	
Age 2	-0.63	-0.96	-0.29	<0.001	
Age 3	-0.74	-1.12	-0.39	<0.001	
Age 4	-0.78	-1.21	-0.39	<0.001	
Age 5	-1.00	-1.55	-0.38	<0.001	
Age 6	-1.11	-2.16	-0.05	0.02	
Age 7	-0.61	-1.86	0.70	0.33	
Age 8	-1.43	-4.00	1.00	0.31	
Age 9	-15.00	-32.92	1.67	0.04	
Random:					
Between-individual	0.003	0.00	0.01		0.008
Genetic	0.003	0.00	0.01		0.009
Focal year	0.39	0.09	0.78		0.97
Residual	0.004	0.00	0.01		0.01

3.4.4 *Annual brood production*

We found a positive among-individual correlation between the number of broods produced in a year and an individual's survival, where individuals that produced more broods had a higher survival probability (Figure 3.3a, Supplementary Information S3.4). The correlation

became negative when the terminal effect was included in the model (Figure 3.3b, Supplementary Information S3.4). However, we found that when selective disappearance was accounted for (i.e., removing individual fecundity records from the data for individuals who do not survive through the entire breeding season), no between-individual correlation was found (Figure 3.3c, Supplementary Information S3.4). We found no correlation between annual brood production and survival in the genetic space in any of the models, though the mode was slightly positive (Figure 3.3, Supplementary Information S3.4).

As also observed in the univariate analysis, we found strongly positive between-individual variance in fecundity but only a small positive genetic effect of fecundity in all the models (Figure 3.3, Supplementary Information S3.4). There was no between-individual effect or heritability of survival. Variance in survival was largely explained by the focal year.

Similarly to the univariate model of fecundity, we found that as age increased, the annual number of broods produced also increased, but the strength of this trend decreased with age (Supplementary Information S3.3). We also found a negative terminal effect; birds had lower brood production in their final year (Supplementary Information S3.3B). However, when selective disappearance was accounted for, the terminal effect was positive (Supplementary Information S3.3C).

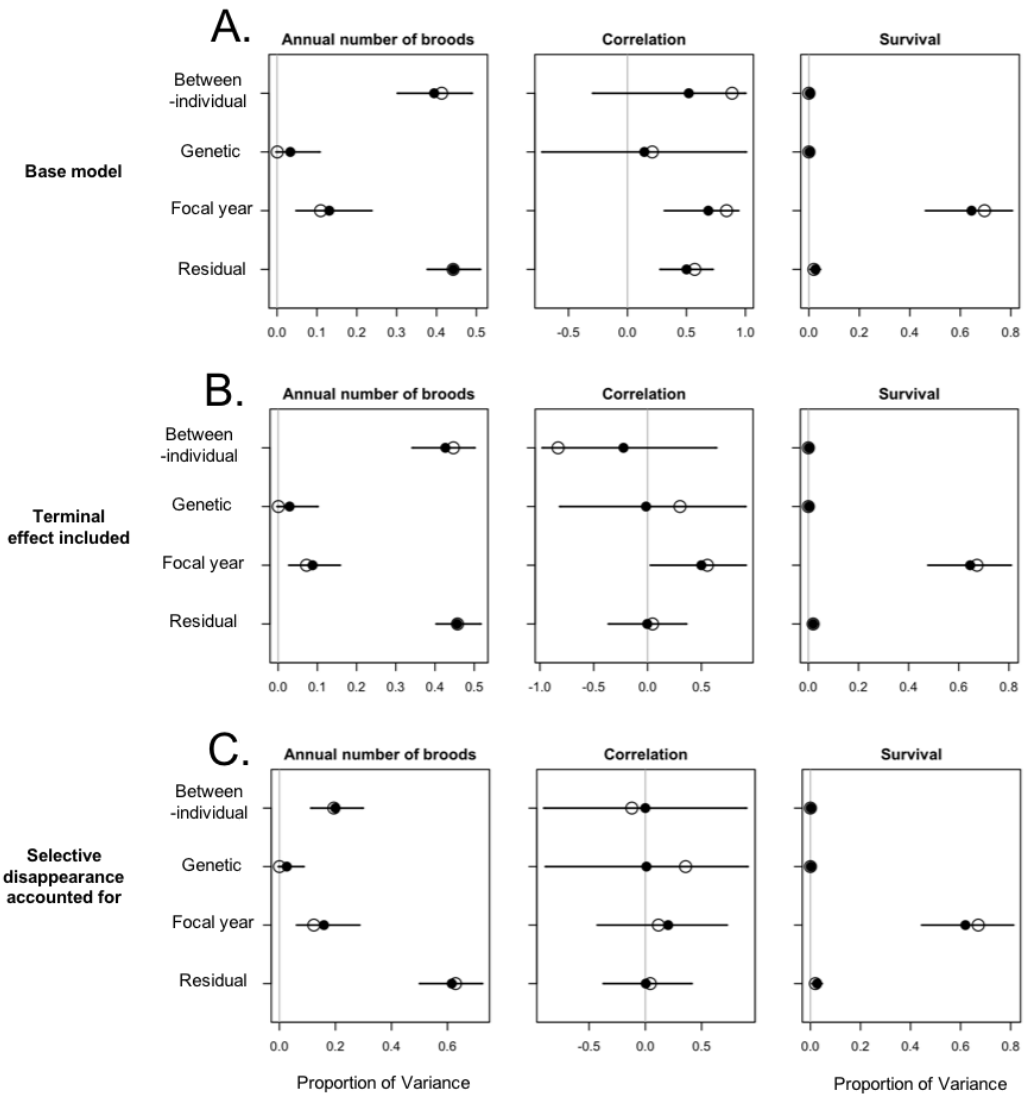


Figure 3.3: The proportion of variance explained by annual brood production and survival and the between trait correlations for these traits for each random effect in the model. A. outputs from the base model, where age and its quadratic effect were included as fixed effects for fecundity; B. outputs of the base mode with the terminal effect included for fecundity and C. outputs for the model described in B, but with fecundity data for individuals that were not present at the end of their final breeding season removed. Filled circles are the mean and open circles are the mode variance of the posterior MCMC samples. Whiskers are the 95% confidence intervals. Note that a negative correlation indicates that lower brood production had a lower survival probability.

3.4.5 *Total annual egg production*

We found no between-individual correlation between total annual egg production and survival (Figure 3.4, Supplementary Information S3.6; fixed effects are presented in Supplementary Information S3.5). However, the posterior mean and mode did consistently lie in negative space for the between-individual effect, with 57% of iterations lying below zero for the most conservative estimate (data accounting for selective disappearance and terminal effect included, Figure 3.3C, Supplementary Information S3.6). There was no genetic correlation between total annual egg production and survival (Figure 3.4, Supplementary Information S3.6). Similarly to the number of broods produced in a year, there was positive between-individual variance and small heritability of egg production (Figure 3.4, Supplementary Information S3.6). Variance in survival was largely accounted for by the focal year. The number of chicks produced in a year yielded similar results to the total annual egg production (Supplementary Information S3.7 and S3.8).

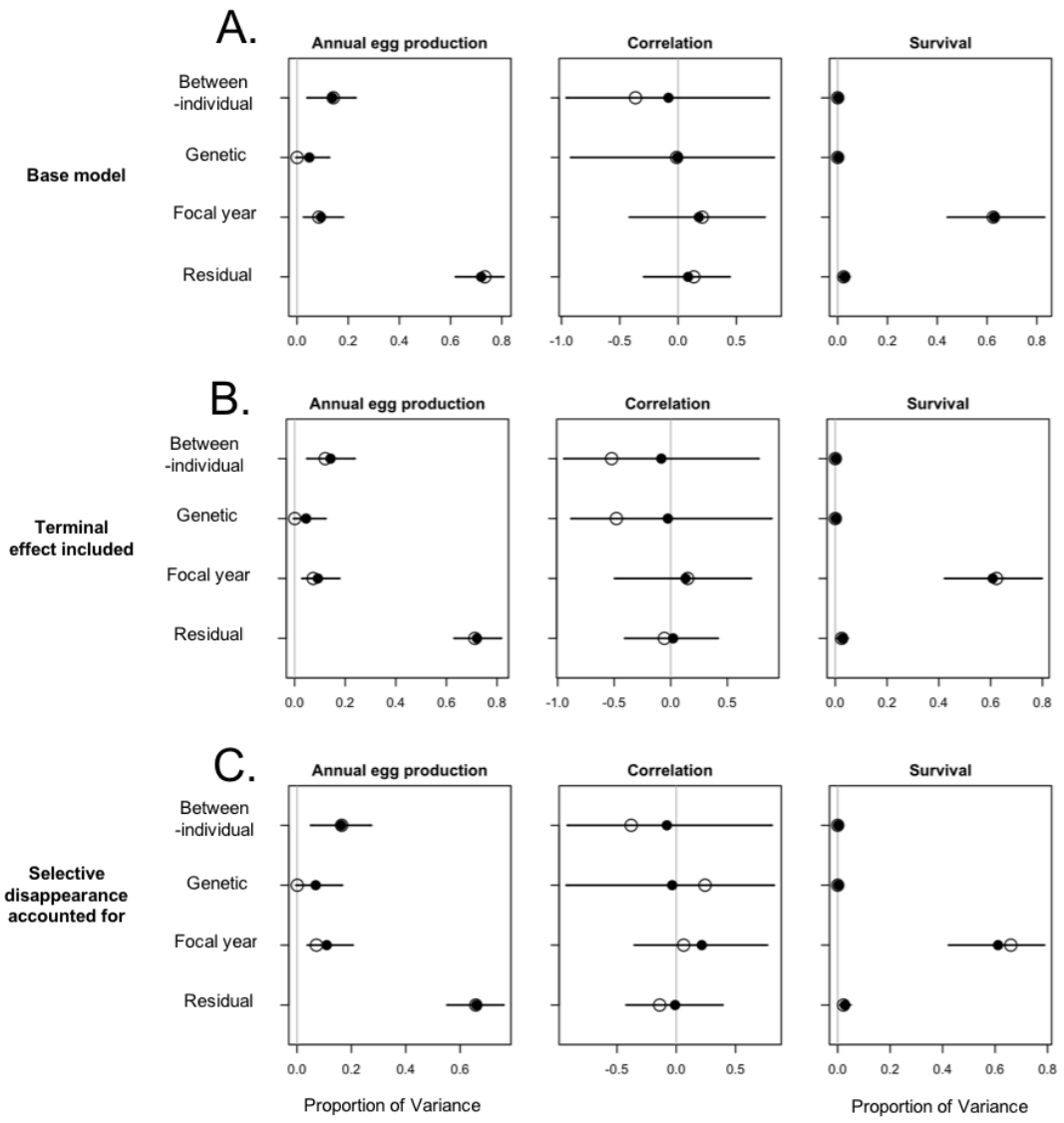


Figure 3.4: The proportion of variance explained by the total annual number of eggs produced and survival, and the between trait correlations for these traits for each random effect in the model. A. outputs from the base model, where age and its quadratic effect were included as fixed effects for fecundity; B. outputs of the base mode with the terminal effect included for fecundity; and C. outputs for the model described in B, but with individuals not present through the whole of their final breeding season removed. Filled circles are the mean and open circles are the mode variance of the posterior MCMC samples. Whiskers are the 95% confidence intervals. Note that a negative correlation indicates that lower egg production is associated with a lower survival probability.

3.5 Discussion

Our results demonstrate the importance of accounting for age-specific changes in fecundity. We detected a negative trend between fecundity and mortality risk but found no correlation between fecundity and survival in the bivariate analysis. This difference is probably due to the modelling of age-associated changes in fecundity. As birds aged, the number of offspring they produced increased, whilst at the oldest ages there is evidence of late-life senescence, partly explained by terminal fitness declines. However, we found that when between- and within- individual age-related changes in reproductive output are considered, it becomes apparent that it is individuals that live longer that drive this trend. As individuals age they produce more offspring, the rate of which decreases at later ages, but individuals with longer lifespans produce more offspring each year. Lundy sparrows, therefore, at the population level, show a commonly found pattern of senescence, with increases in reproductive output in early life followed by a steady decline in late life. However, Schroeder et al. (2012), using the same population, found that fecundity decreases as an individual ages and fecundity does not vary with lifespan. Investigation into this showed our results differ due to differences in modelling approaches, specifically where we have defined age as the change in age from the individual's mean, whereas Schroeder et al. used raw age.

Annual fecundity was shown to have a small heritability of 0.06 (95% CI: 0.00-0.11) (or 0.03–0.05 in the bivariate analyses). However, the between-individual effect explained more variation in fecundity than the genetic effect in both univariate (0.18, 95% CI: 0.09-0.21) and bivariate analyses (0.20, 95% CI: 0.12-0.30 for brood production and 0.16, 95% CI: 0.05-0.27 for egg production). However, as the residual variation in fecundity explained the most variation (0.62, 95% CI: 0.50-0.72 for number of broods and 0.66, 95% CI: 0.56-0.76 for the number of eggs), quality was not always consistent between individuals. This supports the theory that variation in reproductive fitness is in part a result of variation in

phenotypic quality. That is, in certain conditions, some individuals will outperform others but if the conditions change, these individuals will not remain “high quality”. For example, silver spoon effects have shown individuals who had better early life conditions have increased early-life reproduction but lower late-life reproduction (Spagopoulou et al. 2020).

A key finding of this work is that the negative terminal effect of fecundity is driven by individuals with an incomplete final breeding season (i.e., selective disappearance). Individuals that do have a complete breeding season in their final year produce more broods than birds of the same age that are still alive (Supplementary Information S3.9). The reason for this is possibly one of resource allocation. An individual at the end of its life will not benefit from allocating resources to self maintenance, when this will not ensure the individual will survive to the following breeding season (Bonneaud et al. 2004; Hanssen 2006; Froy et al. 2013). Instead, a better strategy would be to invest heavily in reproduction to maximise the genetic contribution to the future population. Without accounting for selective disappearance, we would have concluded that individuals have a terminal decline in reproductive effort in their final year for which we were able to determine through sighting efforts both within and outside of the breeding season. We therefore highlight that it is important to determine if terminal declines are true declines in fecundity in the final year or whether breeding information is incomplete. By contrast we have shown that the apparent negative terminal effect is a result of individuals having perished and therefore not completing a full breeding season.

We have found that the costs of reproduction are minimal, counter to the currently favoured hypothesis that life-history trade-offs drive variation in fecundity (Stearns 1976). We found a small negative effect of the annual number of eggs produced, however, this was not reflected in the number of broods produced and the effect was negligible for the number of chicks produced. A negative correlation between brood production and survival was found to

be driven by selective disappearance in the final year of breeding. We found no detectable genetic correlation between fecundity (either as the annual number of broods, eggs or chicks produced) and survival. Similarly, we found no correlation between fecundity and survival in the permanent environmental effect. It is possible that our data did not have the power to detect this effect. However, even if this were the case, it is unlikely that the selection pressure would be large enough to produce any phenotypic change in the population. It is also unlikely that our study lacked the power to detect a genetic correlation, as other studies have shown genetic effects of survival with lower sample sizes (387 individuals compared to 507 in our dataset) than used here (Vedder et al. 2021a).

Though it might be expected that survival costs will be closely correlated in relation to the number of broods, eggs and chicks produced, the effects of incubation and chick rearing should not be overlooked or underestimated. Each stage of offspring rearing requires unique characteristics that incur specific costs. The cost of laying an egg is often overlooked but can have substantial impacts on a parent's condition before any parental care is given, which in turn impacts on subsequent parental ability (Monaghan et al. 1998). Incubation is energetically challenging for birds (mainly females in passerine species) that are constrained to being at the nest for a considerable period of time. As such, the costs can be incurred through a reduction in self-care or through carry-over effects into the nestling period (Heaney and Monaghan 1996; Voss et al. 2006; Nord and Williams 2015). During chick rearing, the costs of increased clutch sizes have been most well studied in terms of increased provisioning effort. It has been well documented that parents are able to care for clutch sizes larger than those they normally lay (Monaghan and Nager 1997). However, costs are incurred via the effort required to acquire the quality and quantity of food delivered (Wright et al. 1998). In this study, we found no or negligible costs, measured as parental survival, associated with fecundity measured as either the number of broods, eggs or chicks

produced. This does not conflict with the costs found in previous studies, but indicates that these costs alone are not enough to drive the variation in brood size.

One possible explanation for the lack of an apparent cost of reproduction in our study is that there are highly complex interactions between individual condition and the environment. Our results show no (or negligible) among-individual correlation between fecundity and survival. This shows that an individual is not consistent in its quality. We define quality as a collection of traits that make an individual predisposed to perform above the population average at a given time, be that in fecundity, survival and/or access to desired resources. For example, older individuals may be advantaged with experience when environmental conditions are unfavourable (Lunn et al. 1994), whereas in other years senescence would cause them to have lower reproductive output than younger birds in the population (Hammers et al. 2012).

Another (but not mutually exclusive) explanation for our results showing no costs of reproduction is offspring variability. As our results showed no genetic correlation between fecundity and survival, no heritability of survival and small heritability but a large between-individual effect of fecundity, we can conclude that offspring are variable in their fitness. Why this is, remains unclear. One possibility is that trade-offs occur within the nest through limits on the amount of parental care that can be given (e.g., provisioning). Another possibility is that offspring vary in their life-history strategy. Creating variable offspring within a brood has the potential to increase the likelihood of the parent having a successful breeding attempt (i.e., having at least one offspring recruit) if environmental conditions are unfavourable. There is growing evidence that offspring within the same brood follow alternative life-history trajectories dictated by their specific developmental conditions (Slagsvold et al. 1984; Groothuis et al. 2005; Muller and Groothuis 2013; Drummond and Rodríguez 2013; Vedder et al. 2021b).

3.6 Supplementary Information

S3.1: Univariate animal models of alternative fecundity proxies: total annual eggs produced, number of chicks in the nest at 5 days old, total number of fledglings produced and total number of recruits. 928 observations were used in this analysis from 510 females.

Fecundity trait		Posterior mean	95% CI - lb	95% CI - ub	pMCMC
Annual egg production					
Fixed	(Intercept)	6.07	5.09	7.17	<0.001
	Δ age	0.30	0.01	0.54	0.02
	Average age	1.53	1.16	1.90	<0.001
	Δ age^2	-0.56	-0.75	-0.36	<0.001
Random	Between-individual	2.67	0.75	4.50	
	Genetic	1.41	0.00	3.10	
	Focal Year	1.65	0.45	3.02	
	Residual	10.98	9.77	12.37	
Chicks at age 5					
Fixed	(Intercept)	3.03	2.27	3.85	<0.001
	Δ age	0.39	0.19	0.59	<0.001
	Average age	1.06	0.76	1.32	<0.001
	Δ age^2	-0.37	-0.51	-0.23	<0.001
Random	Between-individual	1.51	0.58	2.42	
	Genetic	0.51	0.00	1.26	
	Focal Year	0.60	0.13	1.24	
	Residual	6.44	5.67	7.19	
Fledglings	(Intercept)	3.04	2.26	3.84	<0.001
Fixed	Δ age	0.39	0.20	0.57	<0.001
	Average age	1.05	0.79	1.32	<0.001
	Δ age^2	-0.37	-0.51	-0.22	<0.001
Random	Between-individual	1.54	0.57	2.50	
	Genetic	0.51	0.00	1.28	
	Focal Year	0.63	0.12	1.32	

	Residual	6.43	5.64	7.19	
Recruits	(Intercept)	0.34	0.07	0.57	0.01
Fixed	Δ age	-0.01	-0.06	0.04	0.86
	Average age	0.09	0.03	0.15	0.002
	Δ age^2	-0.04	-0.08	-0.01	0.02
Random	Between-individual	0.01	0.00	0.02	
	Genetic	0.01	0.00	0.02	
	Focal Year	0.21	0.07	0.39	
	Residual	0.41	0.38	0.46	

S3.2: The proportion of variance explained by the random effects for alternative fecundity proxies: total annual eggs produced, number of chicks in the nest at 5 days old, total number of fledglings produced and total number of recruits (n = 928 observations, 510 females).

Fecundity measure	Between-individual	Genetic	Focal year	Residual
Eggs	0.16	0.08	0.09	0.66
Chicks at age 5	0.17	0.06	0.07	0.71
Fledglings	0.17	0.06	0.07	0.71
Recruits	0.01	0.01	0.31	0.67

S3.3: Model output for the fixed effects in bivariate models of fecundity, measured as annual number of broods produced, and survival. Survival was measured as an approximation of the Cox proportional hazards model. 1118 observations from 507 females were used for these models. The associated random effects for these models can be found in S3.4.

Model		Posterior mean	95% CI - lower	95% CI - upper	pMCMC
A. Base model	Intercept	1.93	1.70	2.14	<0.001
	Age	0.32	0.23	0.41	<0.001
	Age^2	-0.03	-0.05	-0.02	<0.001
B. Base model + terminal effect	Intercept	1.96	1.75	2.16	<0.001
	Age	0.41	0.31	0.51	<0.001
	Age^2	-0.04	-0.05	-0.02	<0.001
	Terminal effect	-0.40	-0.49	-0.29	<0.001
C. Selective disappearance data: Base model + terminal effect	Intercept	1.43	1.04	1.76	<0.001
	Age	0.47	0.30	0.65	<0.001
	Age^2	-0.05	-0.08	-0.02	0.002
	Terminal effect	0.25	0.05	0.45	0.008

S3.4: The variance components predicting fecundity (annual number of broods produced) and survival (n = 1118 observations, 507 females) and the correlation between the two traits estimated in a bivariate model. Values are the posterior means with credible intervals in parentheses. The results for the main effects of these models can be found in S3.3.

		Proportion of Variance		
Model		Total annual egg production	Survival	Correlation
A. Base model	Between-individual	0.39 (0.30-0.48)	0.00 (0.00-0.01)	0.54 (-0.27-0.99)
	Genetic	0.03 (0.00-0.10)	0.00 (0.00-0.01)	0.15 (-0.73-0.98)
	Year	0.13 (0.05-0.23)	0.65 (0.48-0.82)	0.68 (0.33-0.93)
	Residual	0.44 (0.37-0.50)	0.02 (0.01-0.04)	0.49 (0.26-0.69)
B. Base model + terminal effect	Between-individual	0.43 (0.34-0.50)	0.00 (0.00-0.01)	-0.22 (-0.97-0.63)
	Genetic	0.03 (0.00-0.10)	0.00 (0.00-0.01)	-0.01 (-0.81-0.90)
	Year	0.09 (0.03-0.16)	0.65 (0.48-0.80)	0.50 (0.04-0.91)
	Residual	0.46 (0.41-0.51)	0.02 (0.01-0.03)	0.00 (-0.35-0.35)
C. Selective disappearance data: Base model + terminal effect	Between-individual	0.20 (0.12-0.30)	0.00 (0.00-0.00)	0.00 (-0.89-0.89)
	Genetic	0.03 (0.00-0.08)	0.00 (0.00-0.00)	0.01 (-0.88-0.90)
	Year	0.16 (0.07-0.28)	0.62 (0.45-0.81)	0.20 (-0.42-0.72)
	Residual	0.62 (0.50-0.72)	0.03 (0.01-0.04)	0.00 (-0.36-0.40)

S3.5: Model output for the fixed effects in bivariate models of fecundity, measured as total annual egg production, and survival (n = 1118 observations, 507 females). Survival was measured as an approximation of the Cox proportional hazards model. The results of the associated random effects can be found in S3.6.

Model		Posterior mean	95% CI - lower	95% CI - upper	pMCMC
A. Base model	Intercept	5.02	3.75	6.24	<0.001
	Age	2.38	1.77	3.11	<0.001
	Age^2	-0.27	-0.38	-0.17	<0.001
B. Base model + terminal effect	Intercept	5.04	3.72	6.31	<0.001
	Age	2.39	1.75	3.09	<0.001
	Age^2	-0.26	-0.37	-0.15	<0.001
	Terminal effect	-0.24	-0.94	0.40	0.49
C. Selective disappearance data: Base model + terminal effect	Intercept	4.66	3.09	5.99	<0.001
	Age	2.68	1.99	3.44	<0.001
	Age^2	-0.29	-0.41	-0.17	<0.001
	Terminal effect	0.70	-0.14	1.44	0.09

S3.6: Variance components from bivariate models predicting fecundity (total annual egg production) and survival, and the correlation between the two traits (n = 1118 observations, 507 females). Values are the posterior means with credible intervals in parentheses. The main effects from these models can be found in S3.5.

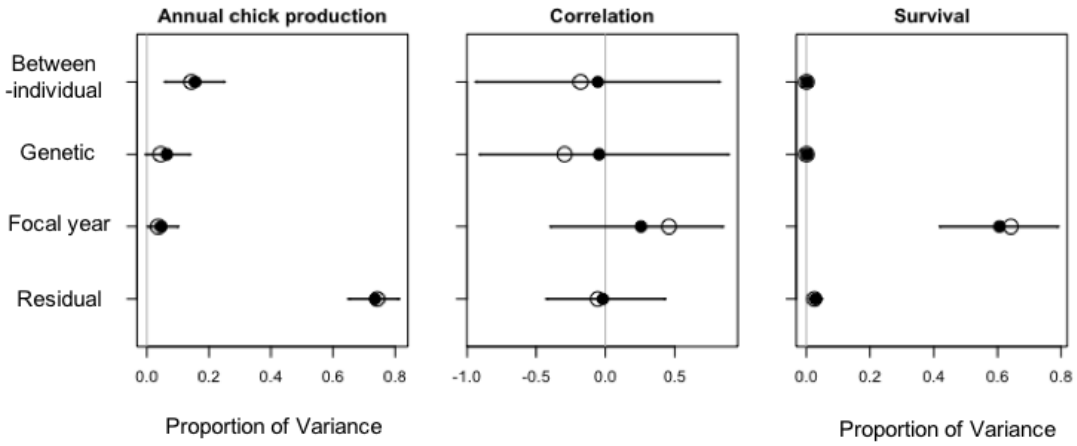
Model		Proportion of Variance		
		Total annual egg production	Survival	Correlation
A. Base model	Between-individual	0.14 (0.05-0.23)	0.00 (0.00-0.01)	-0.08 (-0.94-0.77)
	Genetic	0.05 (0.00-0.12)	0.00 (0.00-0.00)	-0.03 (-0.87-0.88)
	Year	0.10 (0.03-0.18)	0.60 (0.40-0.77)	0.13 (-0.49-0.70)
	Residual	0.72 (0.63-0.80)	0.03 (0.01-0.05)	0.02 (-0.40-0.41)
B. Base model + terminal effect	Between-individual	0.14 (0.05-0.23)	0.00 (0.00-0.01)	-0.08 (-0.94-0.77)
	Genetic	0.05 (0.00-0.12)	0.00 (0.00-0.01)	-0.03 (-0.87-0.88)
	Year	0.09 (0.03-0.17)	0.61 (0.43-0.79)	0.13 (-0.49-0.70)
	Residual	0.72 (0.63-0.81)	0.03 (0.01-0.05)	0.02 (-0.40-0.41)
C. Selective disappearance data: Base model + terminal effect	Between-individual	0.16 (0.05-0.27)	0.00 (0.00-0.01)	-0.08 (-0.91-0.80)
	Genetic	0.07 (0.00-0.16)	0.00 (0.00-0.00)	-0.03 (-0.92-0.82)
	Year	0.11 (0.04-0.20)	0.61 (0.43-0.78)	0.22 (-0.34-0.76)
	Residual	0.66 (0.56-0.76)	0.03 (0.01-0.05)	-0.01 (-0.41-0.38)

S3.7: Model outputs for a bivariate model of fecundity and survival. Fecundity is measured as the total annual number of age 5 nestlings (n = 1118 observations, 507 females). Survival was modelled as an approximation of the Cox proportional hazards model. Random effects values are the posterior means with credible intervals in parentheses.

Fixed effects	Posterior mean	95% CI - lower	95% CI - upper	pMCMC
Intercept	2.39	1.61	3.23	<0.001
Age	1.58	1.11	2.03	<0.001
Age^2	-0.16	-0.23	-0.09	<0.001
Terminal effect	-0.22	-0.72	0.24	0.36

Random effects	Proportion of Variance		
	Total annual egg production	Survival	Correlation
Between-individual	0.16 (0.06-0.25)	0.00 (0.00-0.01)	-0.06 (-0.93-0.82)
Genetic	0.06 (0.00-0.14)	0.00 (0.00-0.01)	-0.05 (-0.90-0.88)
Year	0.05 (0.01-0.10)	0.61 (0.42-0.79)	0.26 (-0.39-0.84)
Residual	0.74 (0.65-0.81)	0.03 (0.01-0.05)	-0.02 (-0.42-0.42)

S3.8: Variances of fecundity (age 5 nestlings) and survival for the random effects of the model, and their correlation.



S3.9: Model outputs for a univariate animal model for fecundity (annual brood production) to confirm that the negative terminal effect is driven by individuals that died part way through the breeding season (n = 996 observations from 506 females). Results are presented for the fixed effects age and the quadratic effect of age, as well as an interaction between whether it was an individual's last breeding attempt and whether the individual had a complete breeding record for that year. A complete breeding record was defined as whether an individual was sighted in or after August (the end of the breeding season) of the focal year. The between-individual effect, genetic effect and focal year were included as random effects in the model. The random effects are reported as the repeatability (lower 95% confidence interval - upper 95% confidence interval).

Fixed effects	Posterior mean	95% CI - lower	95% CI - upper	pMCMC
Intercept	2.22	1.15	3.12	<0.001
Age	0.41	0.33	0.50	<0.001
Age^2	-0.04	-0.06	-0.03	<0.001
Terminal effect	-1.23	-2.17	-0.19	0.02
Complete breeding record	-0.36	-1.31	0.64	0.50
Terminal effect x complete breeding record	1.14	0.16	2.11	0.02
Random effects	Between-individual	Genetic	Year	Residual
	0.44 (0.33-0.50)	0.0007 (0.00-0.11)	0.07 (0.03-0.15)	0.46 (0.39-0.51)

Chapter 4: Separating the genetic and environmental drivers of body temperature during the development of endothermy in an altricial bird

4.1 Summary

When altricial birds hatch, they are unable to regulate their own temperature, but by the time they fledge they are thermally independent of their parents. Nestling body temperature has been shown to affect future survival and thermoregulation. However, it is currently unknown to what extent body temperature during endothermy development is driven by genetically derived variation or by the nest conditions. We use thermal images of cross-fostered house sparrows throughout the nestling period to separate genetic and environmental drivers of body temperature. Our results show small heritability of body temperature, which reduces over the nestling period. However, we did find that there are effects from the natal environment which carry over into the late nestling stage. Our results also provide evidence that this is independent of physiological growth. We also provide evidence that higher body temperatures are selected for independently of body mass. We therefore, demonstrate the natal environment influences future offspring phenotype in a novel measure; body temperature. We suggest that body temperature is therefore likely a good predictor of individual quality, though quality is inconsistent across natal and rearing conditions.

4.2 Introduction

Conditions during early life have been shown to affect future traits including physiological and fitness traits (Lindström 1999; Metcalfe and Monaghan 2001; Lummaa and Clutton-Brock 2002). Passerine birds are heterothermic endotherms, where body temperatures are influenced by both intrinsic and extrinsic factors, such as body condition (Nord et al. 2013), time of day (Binkley et al. 1971; Barrett and Takahashi 1995) and exposure to stressors (Jerem et al. 2019). Optimisation of body temperature has fundamental benefits for the individual, such as energy conservation (Tattersall et al. 2016) and improved ability to remain active (Torre-Bueno 1976).

There is evidence to suggest that conditions within the nest affect individuals later in life (Metcalfe and Monaghan 2001; Saino et al. 2018; Spagopoulou et al. 2020). To date, the literature on body temperature in birds has focussed on the adaptive response to environmental conditions and mostly reports experimental studies. Early temperature acclimation has been shown to improve thermoregulation and survival when nestlings were subsequently thermally stressed (Arjona et al., 1988, 1990; Yahav and Hurwitz, 1996; Shinder et al., 2002). A recent study by Andreasson et al. (2016) demonstrated variation in the rate of endothermy development in nestling blue tits (*Cyanistes caeruleus*), depending on brood size. Therefore, it is known that the nest environment affects the thermal characteristics of nestlings and this has future fitness implications. However, little is known about whether there is a genetic component of body temperature or whether thermal characteristics of individuals are determined by the environment during development. The rate of growth can vary between young with individuals able to compensate for slow development with faster growth when conditions become more favourable (Metcalfe and

Monaghan 2001). It is therefore important to determine if between-individual differences in body temperature are a result of differences in physiological development or whether body temperature development is independent of growth.

There has been some suggestive evidence in captive chickens that surface region temperatures are heritable (Loyau et al. 2016). However, this needs to be explored in a natural setting, under natural selection pressures. Metabolic rate is considered the key physiological trait for determining an individual's energy expenditure. As body temperature is the result of metabolic heat production, this can be considered as an indicator of an individual's metabolic state. There is evidence that metabolic rate has a heritable component (Rønning et al. 2007) – a trait also shown in wild blue tits, *Cyanistes caeruleus* (Nilsson et al. 2009). However, a recent study by McFarlane et al. (2021) demonstrated that it is prenatal effects, rather than genetic effects, that drive metabolic rate variation in nestlings, which the previous studies were unable to disentangle. Therefore, the next step is to determine how much body temperature is genetically derived or is a result of the conditions during development, and how the drivers change over the nestling period. As a nest can be considered to be a reasonably stable environment and nestlings are easily cross-fostered between broods, nestlings provide the ideal opportunity to monitor between-individual body temperatures while largely being able to account for environmental variability, including variation in parental care. Cross-fostering whole broods also minimises the effect of between-sibling competition conflating the results.

Surface temperature has previously been shown to correlate linearly with core temperature in birds (Hill et al. 1980; Giloh et al. 2012). The use of non-invasive temperature measurements via thermal imaging can be used to determine an individual's thermal condition (McCafferty 2013; Nord et al. 2016). There has also been recent suggestive

evidence that eye-region temperature remains relatively stable when birds are faced with a perceived risk of an energetic shortfall, suggesting the eye region offers prime target for monitoring body temperature indicative of core temperature (Winder et al. 2020). Here, we use eye-region temperature measurements throughout the nestling period to determine to what extent body temperature is driven by natal and rearing conditions. Pre-hatching and post-hatching conditions have been shown to affect nestling phenotype (Mousseau 1998). Maternal effects such as egg yolk composition and incubation behaviour affect nestlings before they have even hatched (Saino et al. 2003; Nord and Nilsson 2011). As cross-fostering is often assumed to remove non-genetic parental effects, it is important to determine how much the natal environment affects the nestling condition in future. We first investigated heritability of body temperature at specific ages throughout the nestling period. We then determined if, as nestlings age, their body temperature becomes more or less driven by the natal conditions, or if body temperature is determined by rearing conditions.

4.3 Materials and methods

4.3.1 *Study population*

Breeding data for a closed population of house sparrows *Passer domesticus* were collected on Lundy Island in the Bristol Channel, (51˚100 N, 4˚ 400 W). The thermal data for this study were collected during the breeding season in two consecutive years, 2018 and 2019. The breeding efforts of each individual have been monitored since ~1996, allowing for the collection of longitudinal life-history data, which forms the basis of our pedigree. Nearly all individuals (>99%) are fitted with a unique colour ring combination, a British Trust for Ornithology metal ring and a passive-integrated transponder. House sparrows have up to four broods a year (mean = 2.3), which gives multiple repeats for each breeding pair. A

genetic pedigree was assembled using 13 microsatellite loci from blood samples taken from the brachial vein or nail clips (Dawson et al. 2012). All blood sampling procedures were performed under UK Home Office licence.

Nestlings were cross-fostered at two days of age post-hatching. Where possible we used a triad approach, where up to three broods had nestlings rotated between the three broods. As many nestlings were cross fostered as possible – i.e., equal brood sizes had all nestlings cross fostered and unequal brood sizes had the number of chicks in the smallest clutch cross fostered. If only one brood was at age two on a day, then no cross fostering occurred.

4.3.2 *Thermal image collection*

Each hatched brood was visited on four occasions, when the nestlings were 2, 5, 10 and 12 days old. In this population, nestlings within a brood usually hatch within ~24 hours of each other, meaning hatching asynchrony does not cause multiple age categories within a nest. House sparrow nestlings have been shown to develop endothermy by 9.5 days post hatching (Dunn 1975). Therefore, this age range captures a near-poikilothermic stage through to that of thermal independence. Visits were limited to the morning to minimise the effect of time of day. Social parents were identified using videos of the nestboxes to determine the parents' unique colour-combination rings. A blood sample was taken from each nestling on days 2 and 12 post-hatching, which allowed genetic parentage to be assigned (described in Schroeder et al. 2015). On each visit, we obtained a thermal image of each nestling, as follows. All the nestlings were temporarily removed from the nest to allow measurement. A thermal image of the right side of each bird's head was then obtained using a C3 FLIR camera (FLIR® Systems, Inc.). Birds within a nest were thermally photographed in a randomised order to prevent smaller/runt chicks, which will cool more rapidly than their larger siblings, from any tendency to being photographed last. The air temperature and

relative humidity were also recorded at the time of image capture. Individual birds were identified from a toenail clipped on day 2.

4.3.3 *Temperature extraction*

The nestling maximum head temperature (hereafter referred to as body temperature, for simplicity) was extracted from the image using the Thermimage package (Tattersall 2017) in R version 3.6.2 (R Development Core Team 2009) and by selecting the hottest pixel in the image of the bird's head. In some cases, the hottest pixel was not on the bird (e.g., when birds were in poor health) and, therefore, we extracted the maximum head temperature identified in FLIR Tools by drawing a temperature selection box exclusively around the bird's head. Eye-region temperature measured in this way has been shown to have similar variation to surface temperature measures and subcutaneous temperature measures with surface body temperature shown to have high repeatability (Nord et al. 2016; Jerem et al. 2019). The atmospheric temperature and relative humidity (from recordings taken at the time of measurement) were corrected for during image extraction in either Thermimage or FLIR Tools.

4.3.4 *Data analysis*

We determined the heritability of body temperature and body mass in separate univariate models for each age (2, 5, 10 and 12 days) using animal models, which use the relatedness structure of a pedigree to estimate additive genetic effects (Henderson 1988; Kruuk 2004). We were unable to run one multivariate model to assess heritability due to insufficient power. Air temperature (for body temperature only) and year were included as fixed effects in the models. In addition to additive genetic effects, the natal brood and the brood in which the nestling was raised (rearing brood) were modelled as random effects. Rearing brood was not

included as a random effect in the model for day 2 as temperature measures were taken at the point of cross-fostering.

We then modelled body temperature using a multivariate mixed model, with body temperature at each age being treated as different traits. This model was used to determine the correlation in body temperature between ages. We were unable to add the additive genetic effect into this model due to insufficient power. Age, air temperature and year were fixed effects. We modelled natal brood and rearing brood as age-specific random effects with a 4x4 unstructured covariance matrix, and the same covariance structure for the residual effects. We repeated this analysis but included body mass as a fixed effect and as an interaction term with age to determine to what extent body temperature is separate from body mass.

The analyses described above were run in MCMCglmm version 2.29 (Hadfield 2010) using R version 3.6.2 (R Development Core Team 2009). The burn-in period was 60,000 for all analyses and a chain length of 460,000 iterations and a thinning interval of 200. Parameter expanded priors were used for the random effect variances and inverse wishart priors were used for the residual variances (V = 1 and nu = 0.002 for the univariate models, and V= 1e-6 and nu=5 for the multivariate models).

65% of nestlings survived to fledging in 2018 and 80% in 2019. We therefore explored whether body temperature predicts survival to the next age using linear mixed effect models in lme4 (Bates et al. 2015). All ages were analysed in separate models. Body mass and year were fixed effects in the models. Natal brood (all ages) and rearing brood (Age 5, 10 and 12 only) were random effects in the models.

4.4 Results

Body temperature increased with age but the rate of increase levelled off towards later ages (Age 2: 31.21ºC ± 0.14, Age 5: 34.47ºC ± 0.11, Age 10: 37.56ºC ± 0.11, Age 12: 38.39 ± 0.07). Body mass also followed a similar trend, increasing at the greatest rate at early ages (Age 2: 3.78g ± 0.04, Age 5: 9.51g ± 0.12, Age 10: 19.22g ± 0.20, Age 12: 21.17g ± 0.18). At Age 2, 69% of nestlings survived to the following age (n_{total} = 696 nestlings), compared to 74% (n_{total} = 582) at Age 5, 76% (n_{total} = 519) at Age 10 and 2% (n_{total} = 490) at Age 12 (survival at this age is resighting as an adult).

We found that body temperature had a small heritability at Age 2 and negligible heritability at the other ages (Age 2, h^2 = 0.04 (95% CI: 0.00-0.18); Age 5, h^2 = 0.001 (95% CI: 0.00-0.21); Age 10, h^2 = 0.0007 (95% CI: 0.00-0.18); Age12, h^2 = 0.0003 (95% CI: 0.00-0.06); Figure 4.1, Table 4.1). At early ages, body temperature was driven by the rearing environment. For Age 2 nestlings, the natal brood explained the most variation in body temperature. However, as this temperature measurement occurred before the cross-foster, the natal brood effect is representative of environmental conditions in the nest (i.e., a rearing brood effect). Variation in Age 5 nestlings body temperature was largely explained by the rearing brood effect. At the ages when the nestlings were thermally independent of the parents (Age 10 and Age 12), variation in body temperature was explained more instead by the natal environment than the rearing environment. The amount of residual variation explaining body temperature increased as the nestlings aged.

Air temperature had a significant positive effect on body temperature at all ages. In 2019, birds were warmer at ages 2 and 5 compared to those recorded in 2018. However, birds were cooler at ages 10 (not significant) and 12 compared to 2018.

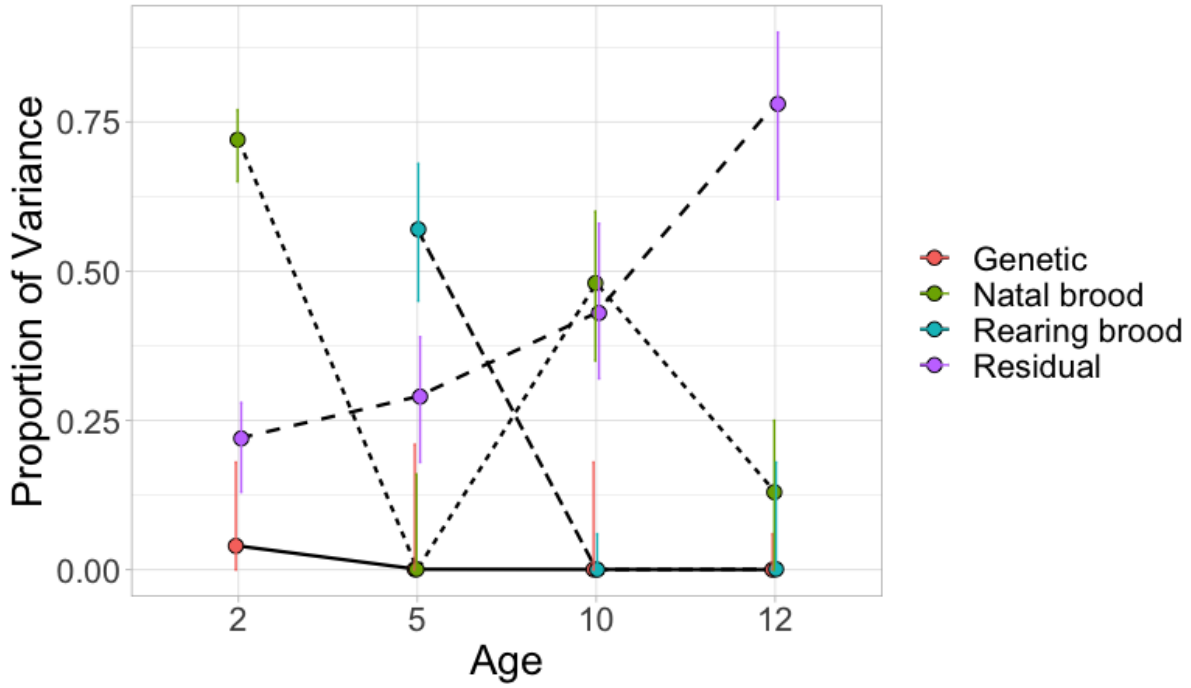


Figure 4.1: The proportion of variance in body temperature at each age (days) over the nestling period for the random effects in the model. Error bars are the credible intervals.

Table 4.1: Variance components from univariate animal models of body temperature at each age (n = 696 Age 2 nestlings, 582 Age 5 nestlings, 519 Age 10 nestlings and 490 Age 12 nestlings). Values for fixed and random effects are the posterior mean (95% credible intervals). Significant values are in bold.

	Age (days)			
	2	5	10	12
Fixed				
Intercept	**20.19 (18.56-21.79)**	**26.21 (24.83-27.56)**	**33.76 (32.31-35.33)**	**34.89 (33.93-36.00)**
Air temperature	**0.58 (0.50-0.66)**	**0.43 (0.36-0.50)**	**0.21 (0.13-0.28)**	**0.18 (0.13-0.23)**
Year (2019)	**0.82 (0.09-1.49)**	**0.59 (0.05-1.13)**	-0.36 (-0.96-0.21)	**-0.51 (-0.84--0.17)**
Random				
Genetic	0.64 (0.00-1.44)	0.41 (0.00-1.10)	0.35 (0.00-1.15)	0.04 (0.00-0.15)
Natal brood	5.57 (4.57-7.10)	0.32 (0.00-0.83)	2.76 (1.62-3.75)	0.32 (0.00-0.61)
Rearing brood		2.95 (2.04-3.80)	0.10 (0.00-0.36)	0.18 (0.00-0.44)
Residual	1.70 (1.14-2.17)	1.49 (0.98-1.90)	2.59 (1.91-3.20)	1.79 (1.44-2.09)
Proportion of variation				
Genetic	0.04 (0.00-0.18)	0.001 (0.00-0.21)	0.0007 (0.00-0.18)	0.0003 (0.00-0.06)
Natal brood	0.72 (0.65-0.77)	0.0008 (0.00-0.16)	0.48 (0.35-0.60)	0.13 (0.00-0.25)
Rearing brood		0.57 (0.45-0.68)	0.0003 (0.00-0.06)	0.001 (0.00-0.18)
Residual	0.22 (0.13-0.28)	0.29 (0.18-0.39)	0.43 (0.32-0.58)	0.78 (0.62-0.90)

We found that chicks born in the same nest who have higher temperatures at earlier ages (2 and 5 days) also have higher temperatures at older ages (5 and 10 days, respectively) (Table 4.2). This relationship weakened slightly when body mass was taken into account, but still remained (Table 4.3). However, by age 12 the correlation with early ages disappears and temperature is instead correlated with that at age 10, though this effect is removed when body mass is included in the model (Table 4.3).

We also found that nestlings raised together who were warmer at earlier ages (2 and 5 days) were more likely to be warmer at later ages (5 and 10 days) (Table 4.2). However, this effect was removed when body mass was accounted for.

Table 4.2: Posterior means (95% credible intervals) for the random effects of the model of body temperature by age (n = 2287 observations from 700 individual nestlings). Variances (95% credible intervals) are on the diagonal. Covariances are below the diagonal and indicate the direction of the temperature relationship between ages. The correlations (95% credible intervals) are above the diagonal and indicate the strength as well as the direction of the relationship between temperature at the given ages. Significant means and correlations are in bold. Non-significant correlations indicate temperature at an earlier age does not predict temperature at an older age.

		2	5	10	12
Natal Brood:					
	2	0.30 (0.23-0.39)	**0.75** **(0.54-0.95)**	**0.43** **(0.26-0.61)**	0.26 (-0.10-0.58)
	5	**0.09** **(0.06-0.12)**	0.05 (0.02-0.08)	**0.63** **(0.29-0.90)**	0.41 (-0.12-0.88)
	10	**0.08** **(0.05-0.10)**	**0.05** **(0.02-0.07)**	0.10 (0.07-0.14)	**0.49** **(0.11-0.94)**
	12	0.02 (0.00-0.04)	0.01 (0.00-0.03)	**0.02** **(0.00-0.04)**	0.02 (0.00-0.03)
Rearing Brood:					
	2	0.11 (0.05-0.19)	**0.41** **(0.09-0.72)**	0.43 (-0.33-1.00)	0.63 (0.03-1.00)
	5	**0.08** **(0.01-0.13)**	0.38 (0.21-0.58)	0.72 (0.08-1.00)	0.42 (-0.01-0.98)
	10	0.02 (-0.01-0.05)	0.06 (-0.01-0.11)	0.02 (0.00-0.05)	0.46 (-0.37-1.00)
	12	0.03 (0.00-0.07)	0.04 (-0.01-0.08)	0.01 (-0.01-0.03)	0.03 (0.00-0.06)
Residual:					
	2	0.20 (-0.16-0.24)	**0.12** **(0.02-0.22)**	0.05 (-0.07-0.15)	-0.07 (-0.17-0.48)
	5	**0.02** **(0.01-0.04)**	0.17 (0.13-0.20)	0.10 (-0.03-0.21)	0.09 (-0.05-0.22)
	10	0.01 (-0.02-0.04)	0.02 (0.00-0.05)	0.29 (0.23-0.34)	0.11 (-0.01-0.25)
	12	-0.01 (-0.03-0.01)	0.02 (-0.01-0.04)	0.26 (0.00-0.06)	0.18 (0.15-0.23)

Table 4.3: Posterior means (95% credible intervals) for the random effects of the model of body temperature by age (n = 2287 observations from 700 individual nestlings). The model included an interaction term between age and body mass. Variances (95% credible intervals) are on the diagonal. The covariances (direction of the effect) are below the diagonal and correlations (magnitude and direction of the effect) (95% credible intervals) are above the diagonal. Significant means and correlations are in bold. Non-significant correlations indicate temperature at an earlier age does not predict temperature at an older age.

	2	5	10	12
Natal Brood:				
2	0.49 (0.34-0.65)	**0.53 (0.16-0.88)**	0.19 (-0.01-0.39)	-0.28 (-0.74-0.04)
5	**0.07 (0.02-0.11)**	0.04 (0.00-0.07)	0.46 (-0.03-0.92)	-0.41 (-0.99-0.19)
10	0.06 (0.00-0.11)	0.04 (-0.01-0.07)	0.20 (0.12-0.28)	0.09 (-0.51-0.58)
12	-0.03 (-0.09-0.00)	-0.01 (-0.04-0.01)	0.01 (-0.03-0.04)	0.03 (0.00-0.07)
Rearing Brood:				
2	0.14 (0.02-0.26)	0.22 (-0.19-0.63)	0.19 (-0.71-1.00)	0.28 (-0.65-1.00)
5	0.04 (-0.06-0.14)	0.51 (0.27-0.80)	0.46 (-0.34-1.00)	0.18 (-0.46-0.98)
10	0.01 (-0.05-0.06)	0.05 (-0.03-0.14)	0.02 (0.00-0.08)	0.37 (-0.72-1.00)
12	0.01 (-0.05-0.08)	0.02 (-0.05-0.09)	0.01 (-0.02-0.05)	0.04 (0.00-0.11)
Residual:				
2	0.23 (0.18-0.28)	0.02 (-0.08-0.12)	0.01 (-0.11-0.12)	-0.10 (-0.21-0.01)
5	0.00 (-0.02-0.03)	0.18 (0.15-0.23)	0.03 (-0.09-0.13)	0.04 (-0.01-0.17)
10	0.00 (-0.03-0.03)	0.01 (-0.02-0.03)	0.32 (0.26-0.38)	0.08 (-0.06-0.20)
12	-0.02 (-0.05-0.00)	0.01 (-0.02-0.03)	0.02 (-0.02-0.05)	0.23 (0.18-0.28)

Nestlings with higher body temperatures had higher survival probabilities to the next age, independently from body mass (Table 4.4). However, body temperature at age 12 did not predict survival to adulthood (estimate = 0.00 ± 0.04, Table 4.4). Body mass also predicted survival but only at earlier ages (Table 4.4).

Table 4.4: Generalised linear mixed effects model outputs for survival to the next age predicted by body temperature. Each age was modelled separately (n = 696 Age 2, 582 Age 5, 519 Age 10 and 490 Age 12). Survival for Age 12 was determined by resighting adults.

			Estimate	SE	*z*	*p*
Age 2						
	Fixed effects	(Intercept)	-12.07	2.28	-5.28	**<0.001**
		Body temperature	0.31	0.07	4.25	**<0.001**
		Body mass	0.91	0.19	4.83	**<0.001**
		Year (2019)	3.49	0.66	5.26	**<0.001**
	Random effects	Natal brood	8.88	3.00		
Age 5						
	Fixed effects	(Intercept)	-9.31	2.99	-3.11	**0.002**
		Body temperature	0.18	0.09	1.89	**0.058**
		Body mass	0.48	0.10	4.95	**<0.001**
		Year (2019)	3.62	0.67	5.41	**<0.001**
	Random effects	Natal brood	0.03	0.16		
		Rearing brood	7.32	2.71		
Age 10						
	Fixed effects	(Intercept)	-6.42	1.84	-3.48	**0.001**
		Body temperature	0.20	0.05	3.62	**<0.001**
		Body mass	0.05	0.03	1.60	0.11
		Year (2019)	3.33	0.53	6.29	**<0.001**
	Random effects	Natal brood	0.00	0.01		
		Rearing brood	1.35	1.16		
Age 12						
	Fixed effects	(Intercept)	0.14	1.53	0.09	0.93
		Body temperature	0.00	0.04	-0.05	0.96
		Body mass	0.00	0.02	0.08	0.94
		Year (2019)	-0.08	0.13	-0.63	0.53
		Natal brood	0.00	0.00		
		Rearing brood	0.00	0.00		

4.5 Discussion

This study quantified the drivers of body temperature in a cross-fostering experiment of nestling house sparrows. We found small heritability of body temperature and the natal environment was a stronger driver of body temperature after development of endothermy than the environment the nestling was raised in.

Heritability of body temperature was small at early ages but decreased as the nestlings aged. At age 2, most variation in body temperature was explained by the natal brood. At this age, nestlings can be considered poikilothermic, where body temperature is almost entirely derived from the incubation efforts of the parents (Dunn 1975). As we have repeated measures for chicks at this age from the same parent, as house sparrows have multiple broods in one year, it is likely that this heritability is somewhat confounded with parental incubation effects. Individual parents differ in how much time they spend incubating eggs and very young chicks (Ricklefs and Smeraski 1983; DuRant et al. 2013). If individual parents are consistent in the incubation efforts throughout their lives, this could lead to inflated estimates of the heritability of nestling body temperature. Cross-fostering after laying (i.e., before egg incubation) would be an interesting study to determine what effect brooding has on the thermal properties of nestlings. Heritability remained at a similar level at age 5 and decreased slightly by age 10. These results are somewhat similar to Hadfield et al. (2013), who found the proportion of variance of body mass explained by natal nest effects are present at early ages but reduce rapidly during ontogeny, though the effects we observe are more persistent. A key finding of this work is that, at older ages, body temperature is driven by the natal environment. At age 5, body temperature was largely driven by the rearing brood, whereas at age 10 and 12, body temperature was more driven by the natal brood than the rearing brood. This is probably indicative of the point at which nestlings become thermally independent (similar age to that reported in Dunn 1975). Our results also

show as nestlings age, residual variance explaining body temperature increases. It is likely that other factors, such as microhabitats (as demonstrated in Andreasson et al. 2018) and seasonal effects drive variation in temperature. It would be interesting for future research to determine how factors outside of the nest environment influence body temperature of nestlings.

We also found carry-over effects of natal brood on temperature at age 2: warmer birds at age 2 also tended to be warmer at later ages. The carry over effects to age 12 were confounded with body mass and the trend disappeared when this was accounted for. The correlation between body temperature at age 2 and later ages was more strongly explained by the natal brood than the rearing brood. Though this result should be considered with caution as for age 2, the natal brood and the rearing brood are the same at this age, we found natal brood drives carry over effects from age 5 to age 10. We found carry over effects from age 2 ages 10 and 12, and from age 5 to age 12. However, these correlations disappeared when body mass was accounted for, suggesting the temperature correlations at these ages are indicative of growth - birds who were warmer at earlier ages were warmer at late ages but were also the larger nestlings. Andreasson et al. (2018) showed that thermoregulation can be costly for growth. However, the carry over effects between ages 2 to 5, ages 2 to 10 and ages 5 to 10 remained when body mass was accounted for. This, first, confirms that higher temperatures are not a result of digestive thermogenesis, where excess heat is produced as a result of metabolic increase during digestion. Secondly, if nestlings were trading-off investment in endothermy development over growth, we would not expect to find correlations in body temperature when body mass is accounted for. Thomson et al. (2017) found a small effect of nest of origin and heritability of body mass and found as blue tit (*Cyanistes caeruleus*) nestlings age, body mass becomes more driven by the social parents. Our results of body temperature did not follow this trend and so also provides evidence that the effect of the natal environment on body temperature is independent of

growth. Our findings show the importance of distinguishing between condition indexes, as body mass and body temperature are correlated (Supplementary information S4.1); however, our results show there are fundamentally different drivers in this variation.

Year of hatching affected body temperature, with nestlings at ages 2 and 5 being cooler but nestlings at ages 10 and 12 being warmer in 2018 than in 2019. Recorded air temperatures were higher over the breeding season in 2018 (19.3ºC) compared to 2019 (17.9ºC). It is possible that warmer temperatures enabled parents to leave the nest more frequently and/or for longer periods, meaning that younger nestlings in warmer weather, who were yet to become thermally independent, were allowed to cool as they were less likely to reach a critically low body temperature or perhaps food availability was lower in 2018 despite the higher temperature. Alternatively, it is possible that the warmer weather meant that more poor-condition nestlings survived for longer than when the weather conditions were worse. However, this latter hypothesis is not supported by our data as the proportion of nestlings that survived to fledging was lower (65% in 2018 and 80% in 2019).

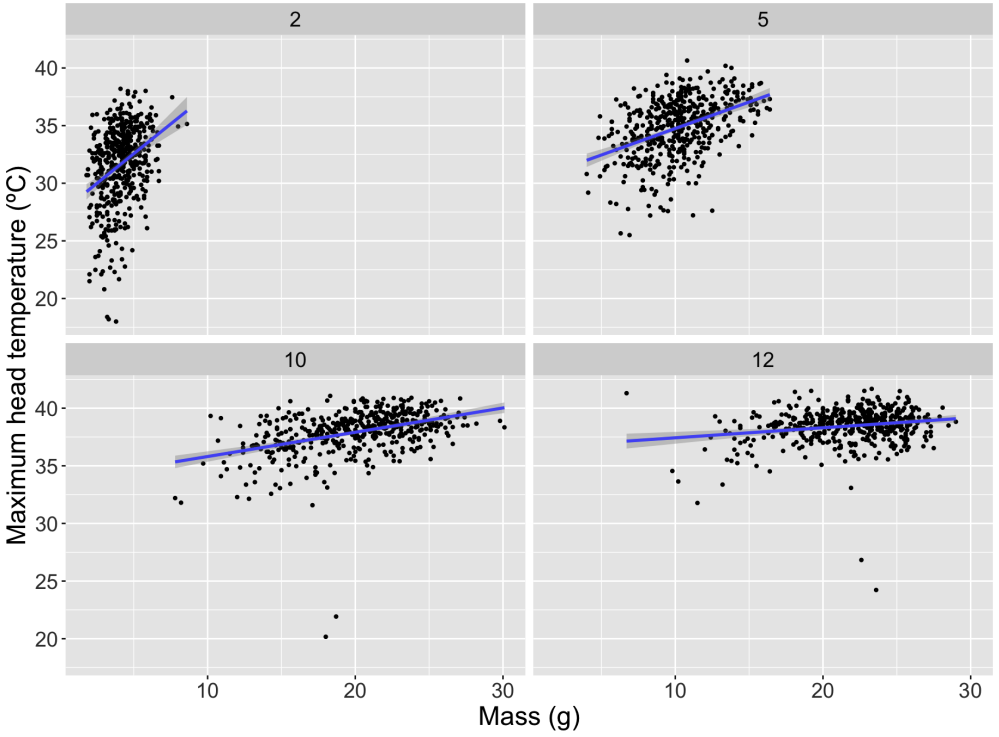
We found that selection is acting on body temperature - body temperature predicts survival to the next age with warmer birds being more likely to survive. The selection on body temperature was independent from body mass, showing the selection effect is not a result of larger nestlings being selected for. Interestingly, at age 10, body mass did not significantly predict survival but body temperature strongly predicted survival. This demonstrates the potential for body temperature to be a key indicator of individual quality during the nestling period. By age 12, this trend disappeared, possibly because by this age the cooler nestlings had died. It is also possible that, after fledging, many other extrinsic factors lead to the death of an individual and therefore, the body temperature is negligible in explaining mortality. At both Age 10 and Age 12, body mass did not predict survival. Though many other studies have found fledgling mass to predict survival (e.g., Naef-Daenzer et al. 2001; Monrós et al.

2002; Morrison et al. 2009), it is likely that the very low survival rates to adulthood (2% survival from Age 12 to adulthood) mean there is not enough power to detect this effect. Though it is not explicitly tested in this analysis, it is likely that the natal environment is a strong driver of survival, as we found carry over body temperature effects between early and mid aged nestlings and also variation in late age nestlings body temperature was explained more by natal brood than rearing brood. Body temperature therefore indicates the quality of a nestling, though this may not translate into adulthood but only briefly explored in this study, it nevertheless indicates quality in nestlings that is independent of growth.

Our results from this study show that the natal environment has lasting effects on body temperature throughout the nestling period. The same trend is not reflected in body mass. We have also shown that body temperature is selected for independently of body mass. Further research is needed to fully understand the mechanisms that cause the natal environment to influence body temperature beyond the initiation of endothermy development. It would also be interesting, though challenging, to follow the effects of this trend throughout adulthood to determine if there are subsequent fitness consequences of the natal environment.

4.6 Supplementary Information

S4.1: The correlation between body mass and body temperature at each age. Regression lines are from locally estimated scatterplot smoothing. Variable temperatures at Age 2 and Age 5 are likely due to the nestlings inability to thermoregulate and so temperatures are likely to be highly influenced by environmental factors, such as ambient temperature and rainfall.



Chapter 5: Discussion

This research provides quantitative evidence that life-history trade-offs need not underlie variation in fitness-related traits. Though I do not dispute previous findings of life-history trade-offs (e.g., Gustafsson and Sutherland 1988; Charnov and Ernest 2006; Lemaître et al. 2015), my results demonstrate that the power of life-history theory to explain variation in traits observed within and between species might be overestimated in the field. It could be that several trade-offs combine to determine the full costs of reproduction, though the trade-offs individually are negligible (such as in Daan et al. 1990; Verhulst and Tinbergen 1991). However, our results clearly show that differences in individual quality cause between-individual differences in reproductive output that appear to be more important than the underlying genetic constraints imposed by trade-offs.

Brood manipulation experiments are the most feasible experiment in the field for determining the presence of a trade-off between parental care and survival. However, our results (Chapter 2) have, importantly, demonstrated that brood manipulations do not result in the predicted trade-off between parental care and self maintenance. Instead, brood size manipulation studies are more probably a test of the effect of imposing a deviation from the individual's optimal level of parental investment. Boonekamp et al. (2014) demonstrated that life-long enlargement of clutch size in jackdaws leads to increased actuarial senescence, compared to reduced clutch sizes. Our results supported this, however we furthered this research by demonstrating that, over the course of an individual's life, increasing parental effort beyond an individual’s optimum through brood manipulation does not lead to fitness gains, as survival costs outweigh the yearly increase in offspring production. It is for this reason that individuals are constrained to reproducing at their individual optimal level. It has been speculated that species that are longer lived should invest in their survival over current

reproduction and shorter lived species should invest in reproduction over survival, meaning low variation in survival for long-lived species and high variation in survival for short-lived species (Gaillard and Yoccoz 2003). However, in this thesis, we have demonstrated a generalisable trend for reproduction being driven by quality across species with different life-history strategies; individuals that naturally produce more offspring also had higher survival probabilities, though the strength of the effect was more severe in longer-lived species.

My results in Chapter 2 show that individuals that naturally lay larger clutch sizes have a higher probability of survival. This supports the individual quality hypothesis, whereby certain individuals have a suite of (not necessarily heritable) traits that collectively provide higher fitness outputs than the population average. The length of time for which an individual survives is a key determinant of fitness, as the longer an individual survives, the more reproductive attempts it has. However, when I studied the genetic and permanent environmental effects of fecundity and survival in a long-term dataset of breeding activity of house sparrows (Chapter 3), I found no heritability or between-individual effect of survival. It could be that, even though genetically similar individuals are predisposed to producing more offspring on an annual basis, variation in lifespan between these individuals would mean they have different lifetime reproductive rates. My results, however, showed no heritability of fecundity and found only a small between-individual effect of fecundity. Nor did I find any evidence that reproduction has a discernible cost in terms of survival. As these traits are large components of individual fitness, it is unlikely that quality is determined by traits we have not measured. Therefore, individuals are likely to vary in quality relative to the population.

I do not believe that my results of Chapter 3, which found no quality effects, dispute the findings of my meta-analysis results in Chapter 2, which found quality effects (individuals

who have high reproduction also have high survival). It is possible that the positive relationship between fecundity and survival in studies of naturally-varying clutch size in Chapter 2 is the result of terminal decline. The results of this chapter showed that individuals that produce more offspring have a higher survival rate, which also means that individuals of lower productivity have a lower survival rate. This could, therefore, be the result of a terminal effect, where individuals in their last year of life are in poor condition and therefore reproduce at a lower rate (Coulson and Fairweather 2001). In Chapter 3, I found that an individual had reduced fecundity in the breeding season before its death. Further investigation showed this to be the result of selective disappearance, it is likely a terminal decline was the result of individuals dying part way through the breeding season and so having an incomplete breeding attempt. By contrast, we found that birds that have a complete final breeding attempt produced more offspring than individuals of the same age who did not die subsequently after the focal breeding season. Most studies are unlikely to be able to determine whether a terminal effect is the result of selective disappearance; however, 65% of the studies used in Chapter 2 studied species which have one breeding attempt, and so an incomplete breeding season cannot result in a terminal effect for these species (assuming number of eggs and chicks is not affected in these studies if the nest does not survive to fledging).

Alternatively, the combined results in Chapters 2 and 3 indicate that individuals vary in quality. It is likely that, because environments are variable spatially and temporally, selection pressures are not constant in size or direction (Nussey et al. 2007; Bell 2010; Sæther and Engen 2015). This would then mean that, within a population, the individuals that are of high quality will differ over time. If individuals are compared within a population in a given year, certain individuals will be of higher quality, however, if the same individuals were monitored throughout the course of their lives, they would not be of consistently higher quality – this distinction likely explains the different conclusions drawn from Chapters 2 and 3. Currently,

life-history studies using long-term datasets are able to analyse fitness patterns associated with ageing and social structure, estimate selection, and link traits between life-history stages and generations (Clutton-Brock and Sheldon 2010). Our understanding of life histories will take a considerable leap when we are able to explore all these factors in unison, as well as accounting for individual phenotypic plasticity. The reason this is yet to be achieved is due to the complexity and the power needed to build these models, which is challenging even with the long-term datasets available currently.

Understanding how variation in life histories arises and is maintained crucially depends on how an individual's phenotype originates. Life-history trade-off theory assumes that variation in fitness is maintained by differences in resource allocation and acquisition between individuals. However, it is well established that past environmental conditions affect future fitness. For example, early life conditions and parental effects are highly important in determining the future fitness of an individual (Metcalfe and Monaghan 2001; Saino et al. 2018; Spagopoulou et al. 2020). Distinguishing between environmental effects pre- and post-hatching is highly important, as it is often assumed that cross-fostering at hatching largely removes the effects of the natal environment (Hadfield et al. 2013). However, the results of Chapter 4 show that there are carry-over effects of the natal environment on body temperature throughout the nestling period, and found that, after development of endothermy, the natal environment accounts for more variance in body temperature than the rearing environment. This effect was independent of body mass and so demonstrates that the effect of body temperature is not a result of growth rate. Further, I showed that selection favours warmer nestlings. The fact that body temperature, independent from growth, determines survival to the next age is a key step forward in our understanding of how an individual's quality is determined in fluctuating environments, where regulation of body temperature is likely to be of high importance.

As we found that a low level of heritability of body temperature and natal brood only partly explained variation in body temperature after development, it can be assumed that parents are producing variable offspring. This hypothesis is supported by species that have hatching asynchrony, such as in common terns, which experience different fitness costs during the nestling period and adulthood determined by hatching order (Vedder et al. 2021b). This study also provides an example of how parents can increase the speed of mortality of the last chick to hatch in order to mitigate resource wastage on occasions when this chick is unlikely to survive. This, importantly, demonstrates that not all offspring within a brood provide the parents with equal fitness opportunities. Though researchers have not necessarily assumed that all offspring are equal, it is challenging to determine the fitness gain that each offspring provides to a parent over the course of its life. It is even more challenging to build this into a complete picture of population-level, generalisable trends. The fact that there are so many sexually reproducing species suggests that mechanisms that create variation are beneficial. By producing variable offspring, parents increase their chances of having some successful offspring in unstable environments; the bet-hedging hypothesis (Kozlowski and Stearns 1989; Laaksonen 2004). Indeed, it has been shown that fluctuating environmental conditions can drive alternative selection pressures on certain traits (for example, residency vs seasonal migration in European shags (*Gulosus aristotelis*) Acker et al. 2021). Future research should determine whether different individuals perform better in certain environmental conditions or whether individuals are indeed consistent in their quality, but are able to adjust their allocation to reproduction to benefit from the current conditions. In this latter scenario, it may be possible that the better strategy is to not reproduce in years where the conditions are poor and offspring are unlikely to survive. This therefore adds a layer of complexity to life-history studies, which generally assume that producing more offspring is beneficial. An exciting future development in life-history studies will be to fully understand how each of an individual's offsprings' fitness contributes to an individual's lifetime fitness.

5.1 Conclusions

Variation in fitness traits within a population has largely been attributed to the trade-off between reproduction and self maintenance. Exploring the costs of increased parental effort demonstrated that the observed costs of reproduction are driven by individuals providing parental care outside their individual optimal level of investment. Instead, it is likely that differences in individual quality lead to variation in fitness traits. However, we found no evidence that quality is heritable or consistent between individuals. We also explore the drivers of body temperature in nestlings and determine that early-life effects have carry-over implications on body temperature into the late nestling period, which are independent from growth. This work is the foundation for future research investigating complex interactions between adult and early-life environmental conditions, genetic predispositions, age specific effects and individual plasticity.

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