Chapter 3: Benefits and costs of parental care (Ed NJR)

Carlos Alonso-Alvarez and Alberto Velando

3.1. Introduction

In order to explain the huge variation in parental behaviour evolutionary biologists have traditionally used a cost-benefit approach, which enables them to analyse behavioural traits in terms of the positive and negative effects on the transmission of parental genes to the next generation. Empirical evidence supports the presence of a number of different trade-offs between the costs and benefits associated with parental care (Stearns 1992; Harshman and Zera 2007), although the mechanisms they are governed by are still the object of debate. In fact, Clutton-Brock's (1991) seminal book did not address the mechanistic bases of parental care and most work in this field has been conducted over the last 20 years. Research on mechanisms has revealed that to understand parental care behaviour we need to move away from traditional models based exclusively on currencies of energy/time. Nevertheless, despite repeated claims, the integration of proximate mechanisms into ultimate explanations is currently far from successful (e.g. Barnes and Partridge 2003; McNamara and Houston 2009). In this chapter, nonetheless, we aim to describe the most relevant advances in this field.

In this chapter, we employ Clutton-Brock's (1991) definitions of the costs and benefits of parental care. Costs imply a reduction in the number of offspring other than those that are currently receiving care (i.e. parental investment, Trivers 1972), whereas benefits are increased fitness in the offspring currently being cared for. Benefits may be derived directly from resources allocated to the offspring (e.g. food, temperature), indirectly from protection against predators, or from the modification of the environment in which the offspring are developing. We begin this chapter by reviewing the traditional idea of resource allocation trade-off, and also explore how trade-offs need not be based on resources and the relevance of cost-free resources. We then analyze in more detail studies of the benefits and costs of parental behaviour and, above all, work that combines mechanistic and functional explanations. Finally, we address the regulatory mechanisms that allow individuals to take decisions on the basis of a cost/benefit balance.

3.2. Trade-offs and the nature of the parental resources

The idea of evolutionary trade-offs in the expression of different traits is intrinsically associated with the cost-benefit approach. The more a parent spends on caring for an individual offspring, the less it will spend in caring for other offspring in current or future reproduction attempts. The fitness cost-benefit can thus be measured in terms of the number of offspring and thus allows for comparisons between individuals in the same currency. Selection pressures on parental care may act on both resource acquisition and allocation (Figure 1). We describe below the traditional views of resource-based allocation trade-offs in parental care and also provide some alternative/complementary perspectives.

3.2.1. Limiting resources

The allocation of resources required for parental duties may be constrained directly or indirectly by the negative effects they have on other fitness-related traits (Stearns 1992; Roff 2002). Many resources allocated to parental care are subject to the principle of allocation and are considered `limited resources´ that can only be spent once (Figure 1; van Noordwijk and de Jong 1986). To maximize their fitness, parents should distribute these resources optimally, with two major trade-offs: between current and future offspring, and between the quantity and quality of descendants (Stearns 1992).

Figure 1 here

Energy and time are the resources most commonly used in theoretical models to exemplify the currency to be traded off. Energy and time can also be easily combined into `energy per unit of time´ (Parker and Maynard-Smith 1990; Clutton-Brock 1991). Allocation of more energy/time to parental duties is thought to reduce the energy/time available for self-maintenance and hence for future offspring (section 3.4). Energy acquisition and allocation are, however, complex traits affected by different factors and are thus difficult to measure. Animals may provision current offspring using previously accumulated energy stores (capital breeders) or using energy gained contemporaneously (income breeders); nevertheless, weighing up the contribution of both processes in the same individual is difficult (Stearns 1992).

Recent evidence suggests that in addition to macronutrients (i.e. carbohydrates, lipids and proteins), small amounts of certain non-energetic substances such as essential aminoacids, carotenoids, flavonoids, vitamins and minerals (i.e. micronutrients) that are not synthesized by the organism may also need to be traded-off between competing functions. Many of these micronutrients benefit offspring growth and development, as well as parental survival. In fish and bird species, carotenoids increase fecundity and parental care (e.g. Pike *et al.* 2007; Tyndale *et al.* 2008), but are also required for parental immune or antioxidant defences (e.g. Pérez-Rodríguez 2009).

3.2.2 Non-linear relationships between resource allocation and fitness

Optimal parental care is dependent on the shape of the function described by fitness plotted against the resources invested in parental effort (Stearns 1992; Roff 2002). The shape of this relationship is commonly taken for granted, despite the fact that empirical evidence is often weaker than is acknowledged. Although simple monotonic relationships between resources allocated to parental care and fitness have been reported, the most common cases probably involve sigmoid-saturating relationships (Clutton-Brock 1991). For instance, in the diet of the Argentine ant (*Linepithema humile*) queens the size of the pupae (a fitness proxy) positively correlate with the availability of macronutrients (in this case protein), although this effect reaches a plateau when their availability is experimentally increased (Aron *et al.* 2001). Similarly, the benefits accrued from non-energetic micronutrients show concave trends, with diminishing fitness returns as allocation to care increases. In the Chinook salmon (*Oncorhynchus tshawytscha*), hatching success is positively correlated with the amount of carotenoids deposited by the female in the egg yolk, although survival benefits decrease asymptotically (Tyndale *et al.* 2008).

Research on physiological mechanisms, however, has also revealed the presence of thresholds that, when exceeded, lead to a switch in physiological pathways and, ultimately, a control of allocation

strategies (see section 3.5). For example, minimum food availability is needed in income breeders (e.g. Schradin *et al.* 2009), while a critical level of fat stores is necessary to initiate egg-laying in capital breeders (e.g. Alisauskas and Ankney 1994).

3.2.3. Limitations of the resource allocation trade-off perspective.

Current reproduction may divert resources away from maintenance (resource allocation, Figure 1), but increasing evidence suggest that reproduction directly alters physiological homeostasis, which in turn causes somatic damages, and links between resource acquisition (diet) and metabolism may also explain the trade-off between current and future reproduction (Figure 2). A first problem of resource allocation models is that the resources required for offspring may differ from the resources needed for the somatic maintenance of parents. Indeed, parents may provide offspring with a different kind of food to that they use for their own maintenance (e.g. Cherel *et al.* 2005). Differences in currency occur in many trade-offs that animals have to confront during parental duties; this is known as the `common currency problem´ (Houston and McNamara 1999). Theoretical biologists approached this problem by modelling the effect of predators or parasites on foraging: animals should weigh up the benefits of these simultaneous goals, that is, energy collected vs. foraging time and mortality risk (e.g. McNamara and Houston 1986). This problem can be tackled by introducing state variables that characterize the current physiological state (e.g. hunger, size, damage, territory size, etc; Clark and Mangel 2000). The state variable may be, for example, the level of damage, which must not exceed a certain threshold (see above), while the variable to be maximized is fitness.

Figure 2 here

Aside from limiting resources, a number of mechanisms underlying parental care have been discovered when exploring the physiological complexities of organisms. This is the case of oxidative stress, which is an imbalance between the production of reactive oxygen species (ROS) by cell metabolism and the state of antioxidant and repair machineries, and leads to oxidative damage (Kirkwood and Austad 2000). Parental duties may increase cell metabolism and hence ROS production and oxidative damage over time, thereby accelerating senescence (Kirkwood and Austad 2000; Metcalfe & Alonso-Alvarez 2010). Limiting substances such as antioxidants or energy for repair mechanisms may be subject to the principle of allocation constraining parental care (Edward and Chapman 2011). Nevertheless, ROS are very reactive (Kirkwood and Austad 2000) and in some parental activities above a certain threshold antioxidant and repair systems are inefficient and soma damage may be unavoidable (Figure 2). Oxidative damage (as an internal state leading somatic deterioration) may explain the link between uncoupled life-history traits, that is, between activities separated in time and therefore not subject to a direct trade-off. Costs and constraints of nutrient-sensing signalling systems may also be independent of resources, although current evidence is inconclusive (see section 3.5).

Environmental challenges may also imply trade-offs that are independent of limiting resources (Figure 2) as, for example, those derived from risky, damaging or stressful conditions during care (Clutton-Brock 1991; Harshman and Zera 2007), and some are obviously 'all-or-nothing' trade-offs (e.g. predation risk). Mechanistic approaches have also revealed that resource acquisition has

intrinsic trade-offs in diet components. For instance, a recent study by Lee *et al.* (2008) on fruit flies shows that the protein:carbohydrate ratio that maximizes egg production differs from the ratio that maximizes lifespan. Authors suggested that high ratios favour reproduction but impair survival since the organism suffers damage caused by sub-products of protein metabolism such as reactive oxygen species and nitrogenous breakdown substances (Lee *et al.* 2008). Since no diet maximizes both functions, a trade-off between reproduction and maintenance may be the inevitable outcome of resource acquisition, rather than the effect of energy allocation as is proposed by traditional models.

3.2.4 Cost-free resources and resources not involved in care

The distinction between costly and cost-free resources is critical to the understanding of the evolution of parental care since costly resources reduce parents' ability to produce other offspring (Trivers 1972). The production of a particular form of cost-free care probably depends on its `context-dependent´ benefits for offspring development. For example, female birds deposit hormones in the egg yolk, but the cost for mothers is unknown and perhaps even non-existent (Gil 2008). Nevertheless, hormone deposition may have environmental or sex-specific effects on offspring fitness, which may explain differences in hormone levels among the eggs in a single clutch (Groothuis *et al.* 2005; Gil 2008). However, direct female control in testosterone deposition into eggs is currently under debate (e.g. Groothuis et al. 2005).

It should be noted that the allocation of substances to offspring may influence offspring fitness, although in some cases this act should not be regarded as parental care as it is a by-product of the parental environment (Chapter 1). For example, the mothers of many species passively transfer pollutants into eggs, which may in fact be beneficial since they prepare the offspring phenotype for a polluted environment (Ho and Burggren 2010). In this case, selection has probably acted on the offspring's developmental pathways rather than on parents' behaviour.

3.3 Benefits of parental care: mechanistic basis

Parents obtain benefits from their reproductive expenditure by increasing offspring survivorship during development (short-term benefits) or by improving offspring survival and fecundity in the long term (delayed benefits). Here we address both cases and also examine how parents seem to be able to actively prepare offspring phenotypes for future environmental challenges.

3.3.1 Short-term benefits of parental effects

In a variety of species, parents improve offspring short-term survival by actively protecting descendants from harsh environments (predators, conspecifics, infections) or by allocating (or regulating) limiting resources that favour their development (Clutton-Brock 1991). This type of behaviour is described in Chapter 1. Many may also obtain long-term benefits, although the short-term effects are the most intuitive. Here we briefly describe some representative examples.

In terms of protection, parents prepare and maintain suitable nesting sites or directly defend offspring from predators, brood parasites or conspecifics. Orange-crowned warblers (*Vermivora celata*) elevate the nest site when the perceived risk of predation is high (Peluc *et al.* 2009). In fish, offspring are often guarded and protected in one of the parent's mouths (i.e. `mouth brooding´; e.g. Balshine-Earn and Earn 1998). Aggressive offspring protection is found in many taxa (Chapter 1), while examples of birds being able to discriminate parasitic eggs by visual cues and reject them, thereby preventing offspring mortality, are also well documented (Chapter 13).

In terms of limiting resources, parents may improve offspring viability by regulating the availability of thermal energy, water, oxygen and energetic and non-energetic nutrients. Social insects regulate nest temperature by metabolic heat production, fanning and water evaporation (reviewed in Jones and Oldroyd 2007); likewise, clutch thermoregulation by parents has been commonly reported in vertebrates such as reptiles and birds (e.g. Deeming 2004). In tree frogs, the location of enough large water pools for egg deposition favours tadpole development (e.g. Brown *et al.* 2010), whereas the parents of some crab and fish species enhance survival by oxygenating eggs via fanning (e.g. Baeza and Fernandez 2002; Green and McCormick 2005). Nutrients are supplied in multiple forms (see Chapter 1) and, for example, mothers of social insects such as spiders, frogs and fish produce non-developing eggs or egg-like structures that are used to feed offspring (i.e. `trophic eggs´; reviewed in Perry and Roitberg 2006). The nourishment of offspring by the maternal body (`matriphagy´) has been described in arachnids and some insects (e.g. Suzuki *et al.* 2005; Salomon *et al.* 2011), while foetuses of viviparous caecilian amphibians are known to scrape lipid-rich secretions and cellular materials from their hypertrophied maternal oviducts (e.g. Wake and Dickie 1998). Finally, parents also provide offspring with non-energetic compounds. A good example is the transfer of carotenoids and vitamins to eggs in many vertebrates, which protect embryos from oxidative stress induced by their high anabolic activity (e.g. Surai 2002; Tyndale *et al.* 2008). Males may also transfer substances via their sperm. The males of the Australian field cricket (*Teleogryllus oceanicus*) produce sperm with certain proteins that can be absorbed by eggs and ultimately improve the embryo's chances of survival (Simmons 2011).

3.3.2 Long-term benefits of parental care

Parental care may have a strong influence throughout an offspring's lifespan. Benefits may be delayed and become evident only after care has ceased. There are many examples whereby individuals born in good condition accrue fitness advantages later in life (`silver-spoon´ effect; Grafen 1988). Malnutrition may permanently alter morphology, physiology and/or metabolism during adulthood and cause long-term effects on fitness (reviewed in Monaghan 2008). For example, in zebra finches (*Taeniopygia guttata*) maternal micronutrients in the egg (carotenoids) influence sexual ornamentation displayed by offspring during adulthood (McGraw *et al.* 2005), whereas a lack of macro- and micronutrients (proteins and antioxidants, respectively) as a nestling reduces reproductive capacities in adulthood (Blount *et al.* 2006). In many passerine species, parents provide spiders to chicks despite their relatively low energy content. Spiders contain high amounts of taurine, a free sulphur amino acid that is required for brain development (Arnold *et al.* 2007); blue tit (*Cyanistes caeruleus*) nestlings that were experimentally supplied with taurine later exhibited greater abilities in spatial learning than control birds (Arnold *et al.* 2007).

Parents may also influence offspring fitness by affecting their brain development, thereby positively helping perceptual, cognitive, and learning capabilities in adulthood (e.g. Law *et al.* 2009). In species with prolonged parental care, offspring may devote more time to learning how to forage and practicing social skills, and to being taught by their parents (Hoppitt *et al.* 2008). Early learning can lead to more effective foraging, anti-predator behaviour, defence against brood parasites and mate choice during adulthood (Curio 1993; Brown and Laland 2001; Davies and Welbergen 2009) and therefore increases fitness (Mateo and Holmes 1997).

3.3.3 Parental care and offspring phenotypic adjustment

Genotypes can produce different phenotypes (i.e. reaction norm) in response to distinct environmental conditions (i.e. `phenotypic plasticity´; Pigliucci 2001). A growing literature exists on the effects of the parental phenotype on the phenotype of the offspring (known as parental effects; see Mousseau and Fox 1998; Chapter 14). Here, we concentrate on those parental effects (usually maternal) that have a positive causal influence on the offspring via phenotypic adjustment to the environment they are likely to encounter.

In fluctuating environments with short-term predictability parents can program offspring development to cope with particular situations (Uller 2008). Parents may produce different offspring phenotypes by affecting developmental pathways or by providing morph-specific resources (reviewed in Badyaev 2009). Parental influence can have long-lasting consequences due to phenotypic organization or epigenetic changes resulting from gene expression (West-Eberhard 2003; Ho and Burggren 2010). Early programming is the consequence of both parental behaviour and plasticity in development pathways. Development pathways, especially in adverse environments, may explain how early conditions can affect offspring phenotypes without active parental effects (Monaghan 2008). Here, however, we only address the effects – although as yet not fully demonstrated – that may be subject to selection on parents.

The phenotypic adjustment of progeny by parents is based on two important assumptions: (i) that environmental cues experienced by parents predict the environmental conditions that their offspring will encounter and (ii) that phenotypic plasticity in offspring development is sensitive to signals produced by parents (Mousseau and Fox 1998). Exposure to signals during embryonic development may be particularly likely to cause accommodation effects, since a disproportionately large part of phenotypic organization occurs during this relatively brief stage in the offspring's life history (West-Eberhard 2003). Below, we summarize some relevant studies on how parents may enable offspring phenotypes to deal with pathogens, predators and adverse conditions.

3.3.3.1. Pathogens

Mothers can transfer information about the pathogens that offspring will encounter (`transgenerational immune priming'; Grindstaff *et al.* 2003): for example, mammals transfer antibodies to descendants via placenta, colostrum or breast milk, whereas birds use the egg yolk (Boulinier and Staszewski 2008). Recently it has been shown that parents of some invertebrates (mostly insects) may also transfer some specific immune factors to their offspring (Freitak *et al.* 2009 and references therein). A novel study also challenges the long-held idea that fathers do not

transmit immune information to their offspring: in the red flour beetle (*Tribolium castaneum*) offspring sired by males exposed to heat-killed bacteria were more resistant to a pathogen infection than offspring from non-exposed males (Roth *et al.* 2010). Seminal substances, genomic imprinting and/or micro RNAs in the sperm could explain these findings (Roth *et al.* 2010).

3.3.3.2 Predators

Many animals learn anti-predatory behaviour from conspecifics (e.g. Curio 1993; Mateo and Holmes 1997), although it is still a subject of controversy whether or not parents actually teach their offspring how to cope with predators (see Hoppitt et al. 2008). Parents may also transfer such information via their eggs. In three-spined sticklebacks (*Gasterosteus aculeatus*), maternal exposition to a dummy or a natural predator prior to egg-laying has an important influence on offspring anti-predator behaviour such that the offspring of predator-exposed mothers exhibit closer shoaling behaviour (Giesing *et al.* 2011). These effects would seem to be mediated by the maternal transfer of high levels of hormones with organizational effects (i.e. glucocorticoids; Giesing *et al.* 2011).

3.3.3.3 Other adverse environmental conditions

In many insects, females favour diapause in their offspring as a response to a short photoperiod, low temperatures or a scarcity of potential hosts, thereby increasing their possibilities of survival (Mousseau and Fox 1998). In the bryozoan *Bugula neritina*, females living in crowded or polluted environments produce larvae with higher dispersal potential (Marshall 2008). In crowded environments parents of some avian species may produce competitive and/or aggressive offspring by depositing testosterone in their eggs (Groothuis *et al.* 2005; Gil 2008). Parents may also prepare offspring for future harsh environmental conditions by acting on their epigenome. In rats (*Rattus norvegicus*) maternal care (pup licking and grooming) influences the stress tolerance of their pups by increasing gene expression in the promoter region of the glucocortocoid-receptor gene (Weaver *et al.* 2004; also section 3.5.2). These epigenomic changes persist into adulthood. Offspring unattended by mothers are more likely to keep a low profile and respond quickly to stress, which may be advantageous when food is scarce and danger is high, but is less beneficial when food is abundant.

Parents may also prepare offspring to the level of care they will receive. In a cross-fostering experiment, Hinde *et al.* (2010) found that foster canary (*Serinus canaria*) chicks grow better if they beg at a level similar to that of the original chicks. These results suggest that mothers increase offspring fitness by matching offspring demands to parental capacity.

3.4. The costs of parental care

Explanations for the evolution of parental care are usually based on variations in the cost of behaviour (Clutton-Brock 1991). There is some confusion in the literature regarding the use of the term `cost´. For example, it is commonly stated that parents transfer the cost of parental care to current offspring; yet cost can be only measured in terms of the offspring sacrificed due to the current care. When parents desert, cannibalize or decrease provisioning to the current brood, the current offspring pay a cost, while parents only lose the potential future benefits (section 3.3). In some cases parental care imposes a cost in terms of reduced numbers of brood-mates; this cost rises as clutch size increases and is known as ‘depreciable care’ (Clutton-Brock 1991). An example is

young/egg provisioning, which constrains clutch size in a variety of species (Stearns 1992). On the other hand, other forms of parental care - for example, anti-predator behaviour - can benefit all offspring in a brood ('non-depreciable care', Clutton-Brock 1991) and the costs only depreciate future reproduction ('residual reproductive value').

From a mechanistic point of view four approaches can be used to assess the cost of parental care: phenotypic correlations between traits (Figure 1), phenotypic manipulations, genetic correlations (by using quantitative genetics) and selection experiments (Clutton-Brock 1991; Reznick 1992). Over last two decades a huge body of literature has been produced on the first two methods; by contrast, work on the latter two has been restricted to analyses of life-history traits such as the negative genetic correlation between growth and fecundity (e.g. Roff 2000), although very few studies have been conducted on the mechanisms underpinning these traits (but see Kim *et al.* 2010). Consequently, in the following sections we review the costs of parental care addressed using phenotypic correlations and manipulations as approaches.

Due to their diversity, it is difficult to classify the costs of parental care. Here we broadly divide these costs into non-physiological and physiological costs. The former are mostly related to resource acquisition in the environment (ecological costs), which arise from exposure to predators, rivals, conspecific or interspecific parasites, and from a reduced amount of time for future mating or reproduction. On the other hand, physiological costs are mostly linked to resource allocation but also arise from trade-offs between parental care and homeostasis, whether or not they are based on limiting resources (Figure 2). In terms of fitness components, parental care ultimately entails reduced survival, fewer mating opportunities and poorer capacity to invest in future offspring. All the above-mentioned mechanistic costs are closely interrelated. Reductions in body energy stores or key micronutrients impair immune-capacity, favour stress and may lead to a greater propensity for infection (e.g. Nordling *et al.* 1998), which in turn may reduce the capacity to escape from predators or to reject reproductive parasites, thereby increasing the risk of body injuries. This implies that selection may act simultaneously, whether directly or indirectly, across a variety of different mechanistic costs (Moore and Hopkins 2009).

3.4.1 Non-physiological costs

Experimental evidence supports the positive correlation between infection risk and parental effort (e.g. Knowles *et al.* 2009), although the causal relationship between infection intensity due to parental effort and future reproduction or mortality has still to be conclusively demonstrated. The best evidence probably comes from birds: wild female collared flycatchers (*Ficedula albicollis*) rearing enlarged broods had higher levels of blood parasites (parasitaemia) than control birds; these levels were in turn correlated to overwinter survival (Nordling *et al.* 1998). Nevertheless, the fitness of the experimental females was not studied. In wild great tits (*Parus major*), females with enlarged broods had increased parasitaemia and poorer overwinter survival rates, although the parasitaemia and survival were not correlated (Stjernman *et al.* 2004). However, these correlations do not necessarily imply causation. Examples of infective agents and parental care other than blood parasites are rare. In a study of common eiders (*Somateria mollisima*) female survival was negatively associated with clutch size, but only during an avian cholera epizootic outbreak, thereby suggesting that parental effort reduced resistance to infection and consequently negatively affected fitness (Descamps *et al.* 2009).

Parental care may increase the risk of predation, and predation obviously reduces fitness. Examples of an increase of predation risk due to parental activities are particularly common among invertebrates, where animals carrying eggs suffer higher predation than non-carrying individuals (e.g. Reguera and Gomendio 1999; Li and Jackson 2003), probably due to their conspicuousness, lower escape capacity (e.g. Shaffer and Formanowicz 1996) and/or higher energetic value for predators. In the pipefish (*Nerophis ophidion*), males carrying their brood in a pouch suffer higher predation rates than females, a finding that seems to be related to their greater conspicuousness (Svensson 1988). Clutch or litter burdens also impair escape capacity, which has been well demonstrated in vertebrates (e.g. fish: Ghalambor *et al.* 2004; reptiles: Cox and Calsbeek 2010; birds: Veasey *et al.* 2001; mammals: Schradin and Anzenberger 2001). In lizards and birds this effect seems to be mediated by an impairment of muscle condition (e.g. Veasey *et al.* 2001; Olsson *et al.* 2001). In birds, fat reserves required for egg production may impair take-off and flight capacity, increasing predation risk (Witter and Cuthill 1993), although to the best of our knowledge the link between this loss of escape capacity and mortality has only been demonstrated to date in reptiles (Miles *et al.* 2000; Cox and Calsbeek 2010).

Parents may also suffer injuries while defending their reproductive investment from conspecifics or reproductive parasites. For instance, burying beetles (*Nicrophorus pustulatus*) suffered more injuries when protecting their young without help from their mate (Trumbo 2007). Parental care activity may wear and tear integuments as well: for example, collared flycatchers rearing experimentally enlarged broods suffered greater wear on their primary feathers and the intensity of this feather damage was positively correlated to post-breeding mortality (Mërila and Hemborg 2000).

Reproductive conspecific or interspecific parasites may also impair parents' survival or future reproduction. In the former case, examples can again be found in birds (reviewed in Lyon and Eadie 2008). However, experiments have so far found little evidence of any long-term cost of conspecific parasitism, a finding that is not particularly surprising since all of these studies used precocial species in which the cost of rearing additional offspring tends to be lower (Lyon and Eadie 2008). In the latter case (interspecific brood parasitism) Hoover and Reetz (2006) reported reduced returning rates in prothonotary warblers (*Protonotaria citrea*) parasitized by brown-headed cowbirds (*Molothrus ater*). However, in certain species - including many insects and fishes that do not expend energy feeding their offspring - hosts may not necessarily suffer a cost when receiving eggs from conspecifics or inter-specifics (reviewed in Tallamy 2005; see also Chapter 13).

Finally, parental care consumes time that could be devoted to remating, conducting new reproductive events and/or self-maintenance. The trade-off between parental care and new mating opportunities has generated a prolific literature focused on the evolution of sexual conflict and biparental care (see Chapters, 6, 9 and 11). In the case of the time dedicated to produce more offspring, it has been experimentally demonstrated in captive lace bugs (*Gargaphia solani*) that the time invested in protecting eggs is traded against fecundity in subsequent clutches (Tallamy and Denno 1982). In the case of time invested in self-maintenance, water striders (*Aquarius remigis*) that bred only once a year (univoltine life cycle) had time to recover lipid stores and survived the winter better than breeders that had two reproductive attempts per year (bivoltine cycle); the latter even had lower lifetime fecundity and longevity (Blanckenhorn 1994). Blue tits that produced a second clutch when the first was experimentally removed delayed their moult and produced a plumage with poor insulation capacity, and subsequently had lower overwinter mortality and less reproductive

success the next season (Nilsson and Svensson 1996). We should however note that in the last two cases the evidence is merely correlational and could be confounded by energetic constraints.

3.4.2 Physiological costs of parental care

3.4.2.1. Energetic cost

Physiological costs have been primarily studied in terms of a loss of limiting resources such as energy or nutrients. Using a variety of different techniques the allocation of resources can be estimated by measuring energy expenditure (oxygen consumption, metabolic rates, doubly labelled water, etc; Speakman 2001). The increase of energy expenditure during parental care is particularly relevant in income breeders. However, most organisms also stockpile energy in their bodies and changes in total body mass or growth rates (in indeterminate growers) may also be used as a way of estimating energy loss (Speakman 2001). A third option is to assess the state of body energy stores that accumulate macronutrients (usually fat and muscles).

To our knowledge, in vertebrates an increase in energy expenditure associated with an increased intensity of a particular parental care behaviour has only ever been experimentally demonstrated in birds and mammals. In mammals, studies have been conducted above all on small female rodents in captivity or in semi-captive conditions during gestation and lactation (reviewed in Gittleman and Thompson 1988 and Speakman 2008). However, despite the variety of studies, a link between the energy expenditure in current care and parents' survival and/or future reproductive success is only supported by two avian experiments (Table 1). By contrast, experiments reporting body mass loss or growth delay as a cost of parental care have been performed for fish, reptiles and birds, and have succeeded in linking such costs to fitness (particularly in birds; Table 1). Finally, some reptile and bird studies also have experimentally demonstrated changes in specific body energy stores, although only two have ever reported a link with fitness (Table 1), probably due to technical limitations in the assessment of body composition, which usually requires sacrifice (Speakman 2001).

3.4.2.2 Non-energetic micronutrients

Here we only describe those cases - calcium, carotenoids and methionine - in which a link with parental behaviour has been established. In the case of calcium, allocation to the egg-shell in oviparous species or milk and foetal bones in mammals has been particularly well studied. Calcium levels drop during gestation and lactation in mammals (Speakman 2008 and references therein), although we have found no report of a decline in calcium levels due to parental care in other taxa. Carotenoids are used in physiological functions (e.g. as detoxificants and immunoenhancers), as well and as pigments of integuments (e.g. Perez-Rodriguez 2009). The egg yolk of fish, reptiles and birds contains large amounts of carotenoids that protect the embryo from the effects of oxidative stress as a consequence of growth (e.g. Surai 2002). Nevertheless, evidence that increased parental effort depletes maternal carotenoid levels has only been reported by means of correlations in laying birds (Bortolotti *et al.* 2003). Finally, methionine stimulates fecundity (egg production) in female fruit flies, but only in a specific ratio with other essential aminoacids (Grandison *et al.* 2009). When such a proportion is not met, methionine can become pro-oxidant, reducing parental survival and reproductive success (Grandison *et al.* 2009). This exemplifies the concept of nutritional geometry,

whereby certain nutrients must be present in particular proportions to favour reproduction (see also section 3.2.4). The lack of such adjustment therefore implies a fitness cost to females (Table 1).

3.4.2.3 Physiological stress

Parental care may lead to an exhaustion of energy stores, which in turn leads to physiological stress (section 3.5.2). Physiological stress may also be triggered by other environmental stressors (Wingfield and Sapolsky 2003). Such a state ultimately provokes damage in the parents. This has been estimated by assessing levels of `heat shock proteins´ (HSPs), molecules that repair protein damage induced by a variety of stressors (Sorensen *et al.* 2003). High HSP values have been related to decreased fecundity in fruit flies (Sorensen *et al.* 2003). As far as we know, only one study has ever related parental care and HSPs: in blue tits, parents whose brood was experimentally enlarged had increased blood HSP levels. In vertebrates, glucocorticoids levels in the blood (acute or baseline; section 3.5.2) are the most analysed proxy of physiological stress, high values revealing high stress levels. Experiments in birds and fish support the idea that an increase in glucocorticoid levels is a consequence of parental effort (e.g. Magee *et al.* 2006; Golet *et al.* 2004). Nonetheless, recent reviews have questioned the link between this effect and fitness (e.g. Breuner et al. 2008; Bonier *et al.* 2009) and in fact we have only found one study that supports this assertion (Table 1).

3.4.2.4 Oxidative stress

The cost of parental care in terms of oxidative stress is supported by correlative evidence, mostly in mammals (e.g. Upreti and Misro 2002; Myatt and Cui 2004), but also by some experiments on birds. In the latter case, zebra finches whose parental effort was increased by brood enlargement had less resistance to ROS at the end of reproduction than controls (Alonso-Alvarez *et al.* 2004; Wiersma *et al.* 2004). However, although medical studies have suggested that oxidative stress generated during gestation in mammals compromises the life of the mother during birth (e.g. Myatt and Cui 2004), a link between reproductive oxidative stress and fitness is only supported by a limited number of experiments and correlations (Table 1). When exposed to a pro-oxidant agent (paraquat), the female fruit flies that were experimentally stimulated to produce eggs died faster than non-breeders (Table 1). However, the authors of this experiment did not test whether oxidative stress was experimentally increased, despite the fact that antioxidants inhibit paraquat-induced mortality in *Drosophila* (Bonilla *et al.* 2006). In zebra finches a negative correlation between the number of breeding events and resistance to oxidative stress has been reported by a study (Table 1) that also found that resistance to oxidative stress was positively related to short-term mortality. However, here parental effort could have included mating effort. Lastly, it has been found that males of two reef-fish species that protect their broods in their mouths suffer from hypoxia (Östlund-Nilsson and Nilsson 2004). Hypoxia could be an alternative cost of parental care but is probably also associated with oxidative stress (Metcalfe and Alonso-Alvarez 2010).

3.4.2.5 Immunosuppression

Parental care may also lead to immunosuppression, mostly as an indirect consequence of other physiological costs. In vertebrates, immunity is reduced by high energy expenditure, loss of body energy stores, carotenoid or micronutrient depletion, glucocorticoid stress response and oxidative stress (e.g. French *et al.* 2007; Bourgeon *et al.* 2009; Perez-Rodriguez 2009). Examples from other taxa are scarce (e.g. insects: Fedorka et al 2004). The impact of parental effort on

immunocompetence is well supported by experiments on birds, in which incubation and brood-rearing efforts were manipulated and the capacity to establish innate or acquired immune responses were accordingly impaired (Table 1). Immunosuppression is also well known as a process associated with implantation and gestation in mammals (e.g. Medina *et al.* 1993). Immunosuppression protects the embryo from maternal immune defences, although the consequences for maternal fitness are still unclear (Speakman 2008). In fact, the link between this immunosuppression and fitness is only supported by a handful of studies (Table 1).

3.4.2.6 The cost of regulatory systems

Endogenous (e.g. neuroendocrine) control systems involved in parental decisions (section 3.5) may *per se* create constraints and costs (Lessells 2008). However, selection may favour simple costless parental rules that, albeit not optimal in all situations, perform well on average (McNamara and Houston 2009). Some molecular signals involved in reproductive activities may have a negative effect on soma maintenance (Leroi 2001; Edward and Chapman 2011). A number of studies on fruit flies and the nematode *Caenorhabitis elegans* suggest that the negative effect of reproduction on longevity arises from a signalling pathway (involving a steroid or insulin-like hormone) rather than from direct resource competition (reviewed in Edward and Chapman 2011). Nevertheless, molecular signals may also activate the physiological mechanisms needed for reproduction that in turn generate damage (Barnes and Partridge 2003). Hence, it is still to be established whether the neuroendocrine control system mediates or creates costs in parental care (Lessells 2008).

3.5. Costs and benefits in the balance

A given level of care reflects the balance between its costs and benefits in a given environment. To reach this balance organisms have evolved control systems that translate the environmental cues perceived by the sense organs into molecular (neuroendocrine) signals that influence physiology, gene expression and behaviour (Lessells 2008).

Parents must balance costs and benefits by taking decisions that maximize fitness, although control mechanisms that integrate environmental cues may produce sub-optimal reaction norms in some circumstances (McNamara and Houston 2009). Indeed, reaction norms are subject to constraints and costs of control regulating systems (previous section). This may explain the variability in evolutionary pathways of parental care between taxa. Here, we review how parental decisions are regulated by physiological pathways and promote either parental effort or favour self-maintenance.

3.5.1 Molecular signals promoting parental effort

Vitellogenin is the precursor of most of the protein content of yolk in nearly all oviparous species. In insects, vitellogenin is produced from food and accumulated in the body, thereby directly linking resource availability to egg production (Page and Amdam 2007). In honeybees, when food resources are scarce vitellogenin levels fall, which in turn triggers foraging behaviour outside the colony as opposed to nursing behaviour (reviewed in Page and Amdam 2007). In insects, the vitellogenin signalling pathway is also linked to the juvenile hormone (JH) pathway (Page and Amdam 2007). JH signalling seems to link resource availability to vitellogenin secretion. For instance, in lubber

grasshoppers (*Romalea microptera*) a threshold of food availability activates JH synthesis, which then stimulates vitellogenin production and oogenesis (Fronstin and Hatle 2008).

Leptin (or leptin-like proteins) controls food intake and immune response in vertebrates (reviewed in Henson and Castracane 2003). It is mostly produced by adipocytes or lipogenic organs and it has been suggested that high circulating levels of leptin – i.e. when they exceed a certain threshold (Henson and Castracane 2003) - permit reproduction to begin (sexual maturity or egg-laying). For instance, in great tits an artificial increase of leptin levels stimulated females to lay a second clutch (Lõhmus and Bjorklund 2009). Female Siberian hamsters (*Phodopus sungorus*) whose circulating leptin levels were experimentally increased had lower rates of infanticide and produced more pups than controls (French *et al.* 2009). Thus, the availability of resources might even stimulate some parental behaviour by means of leptin signalling.

Prolactin also promotes and maintains incubation, gestation and offspring care in vertebrates (Freeman *et al.* 2000). In birds, an experimental reduction of circulating prolactin inhibits incubation and also leads to brood desertion, whereas an experimental increase favours incubatory and protective behaviour (reviewed in Angelier and Chastel 2009). Prolactin levels are negatively controlled by glucocorticoids (next section). Treatment with glucocorticoids decreases prolactin levels in the blood of birds and rodents (Angelier and Chastel 2009). High fat reserves maintain glucocorticod secretion at low rates and hence prolactin-controlled behaviour may continue (Wingfield and Sapolsky 2003; Angelier and Chastel 2009; Spée *et al.* 2010). As in the case of vitellogenin and JH, these studies suggest that several hormones are simultaneously engaged in linking resource availability and parental care.

3.5.2. Pathways inhibiting parental effort

Some physiological changes as a result of short- or long-term unpredictable or uncontrollable stimuli (stressors) cause a redirection of resources to short-term vital processes and impair or threaten homeostasis, but can also inhibit parental investment. Such changes are usually known as the `stress response´ (see McEwen and Wingfield 2003). In invertebrates, stress responses are modulated by HSPs. In fruit flies, for example, high levels of HSP70 (one of the major HSP proteins) increase longevity but reduce egg quality (i.e. hatching success; reviewed in Sorensen *et al.* 2003).

In vertebrates, the hypothalamic-pituitary-adrenal (HPA) axis induces a release of glucocorticoids into the blood a few minutes after exposure to a stressor (acute stress response; e.g. Wingfield and Sapolsky 2003). Baseline (low) glucocorticoid levels are nonetheless required for normal metabolism (Wingfield and Sapolsky 2003). The HPA response promotes the reallocation of resources from energy consuming systems (immunity, reproduction, etc.) to short-term survival (Wingfield and Sapolsky 2003). In addition to environmental (including social) stimuli, glucocorticoid secretion is triggered when lipid stores are exhausted and proteins from muscles and other tissues are catabolised to produce energy (e.g. Spée *et al.* 2010). In those circumstances, glucocorticoids stimulate glucogenesis and accelerate protein breakdown, thus optimizing energy production (Challet *et al.* 1995) but also leading to clutch or brood desertion (Spée *et al.* 2010; Wingfield and Sapolsky 2003). Stress hormones may also favour reproductive effort under some circumstances (e.g. Bonier *et al.* 2009 and references therein). For instance, high glucocorticoid levels intensify

behaviour such as nest defence or foraging during reproduction (Bonier *et al.* 2009), while in mammals glucocorticoid-mediated immunosuppression prevents immune-induced damage to the foetus (e.g. Medina *et al.* 1993).

In birds, the glucocorticoid threshold that promotes desertion seems to depend on the reproductive value of current and future reproduction. House sparrows (*Passer domesticus*) raising experimentally enlarged broods reduced their acute (glucocorticoid) stress response in comparison with parents with reduced broods (Lendvai *et al.* 2007). A comparative analysis of 64 bird species showed that species with a higher value current brood compared to future breeding had weaker corticosterone responses during acute stress; interestingly, females in species with more female-biased parental care also had weaker corticosterone responses (Bokony *et al.* 2009). A decrease in stress response during reproduction may not only be produced by inhibiting glucocorticoid secretion, but also by altering levels of glucocorticoid protein carriers in the blood or by blocking the glucocorticoid action on target tissues (central nervous or reproductive systems; Wingfield and Sapolsky 2003).

Finally, the hypothalamic-pituitary gonadal (HPG) axis induces a release of sexual steroids that trigger mating behaviour at the expense of parental care, thereby playing a role in the trade-off between time devoted to care vs. time devoted to alternative mating opportunities (section 3.3). It is well known from bird and mammalian studies that circulating testosterone levels decline when males start the parental care period and experiments have shown that an increase in testosterone levels in males dramatically inhibits parental duties (e.g. Adkins-Regan 2005; McGlothlin *et al.* 2007). In dark-eyed juncos (*Junco hyemalis*) the males that respond to aggression by increasing testosterone levels are also those that contribute less to care, suggesting that the testosterone release pathway may even constitute a constraint for parental care evolution (McGlothlin *et al.* 2007).

3.6. Final remarks

In this chapter we have summarized important advances in the understanding of the mechanisms underlying the costs and benefits of parental care. Research in this area has increased enormously since the publication of Clutton-Brook's (1991) seminal book. Recurrent problems however remain unsolved. Research into costly mechanisms has provided new insights into the role of specific metabolites or oxidative stress (section 3.4) and into control systems (section 3.5), although empirical support for the relationship between mechanisms and fitness is still weak in many cases. This issue is well illustrated in Table 1. Although all the analysed physiological costs can be intuitively associated with fitness costs, few studies have ever examined just how parental care induces physiological costs and simultaneously produces fitness costs (PC$\rightarrow$FC, 29 cases in 24 studies). Rarer still are studies that at the same time assess the positive correlation between physiological and fitness costs in the same dataset (PHC$\rightarrow$FC, 16 cases in 15 studies). Moreover, most work only assesses fitness proxies over a short period of time. In conclusion, evidence is still quite weak and the strongest support perhaps comes from experiments on body mass loss in birds (Table 1). Nonetheless, oscillations in body mass may not necessarily imply long-term costs, but rather be the result of tissue remodelling (i.e. in capital breeders; Speakman 2008).

Despite these difficulties, advances resulting from the study of phenotypic correlations and, above all, from manipulative experiments have opened up new perspectives such as the role of specific

macro- and micronutrients in parental care. Another question is the inevitable (obligate) costs of parental effort, some derived from resource acquisition rather than from the allocation of limited resources in competitive trade-offs. Obligate costs would include damage such as feather deterioration in hole-nesting birds, injuries caused when defending offspring, specific diet choice or physiological damage (for example, damage revealed by increased levels of the repairing HSPs) induced by stress. Nevertheless, perhaps the best example is that of oxidative damage, since it may depend not only on limited resources such as antioxidants but also on metabolism and cell respiration; in other words, damage is inherent to a simple increase in metabolic activity (Metcalfe and Alonso-Alvarez 2010). Thus, in relation to mechanisms, the debate has so far been centred on resource allocation models (Leroi *et al.* 2001; Barnes and Partridge 2003), but, as already mentioned, many cost and benefits have different currencies (section 3.2). State models may help us to understand optimal parental decisions, i.e. the internal milieu of the individual determines its decisions when facing external challenges (Clark and Mangel 2000). More empirical demonstrations manipulating the state of individuals and assessing their consequences on parental care are still needed.

Another often neglected aspect is the fact that some parental actions should not necessarily be considered as parental investment just because they are seemingly cost-free and their benefits are context-dependent. For instance, the allocation of maternal hormones and immunoglobulins to the egg or embryo seems to be cost-free for the parents and their effects on offspring depend on when they will act and on their interaction with the particular environment at that time (Groothuis et al. 2005; Boulinier and Staszewski 2007). The study of individual states and context-dependence are promising areas for future empirical approaches since they may also serve to detect subtle hidden costs for parents.

One question meriting further attention is the possibility that parents inflict a certain level of stress on their offspring to favour their fitness. For instance, it has been recently shown that a moderate reduction in the food intake of yellow-legged gull (*Larus michaellis*) nestlings reduces oxidative damage during development (Noguera *et al.* 2011). This could be explained as a `hormetic effect´. The `hormetic model´ proposes that fitness returns may describe a positive quadratic relationship with levels of a stressor, intermediate levels producing the highest fitness returns (reviewed in Costantini *et al.* 2010). The best known examples of a hormetic response are those induced by heat stress in insects (see HSPs) and dietary restriction in vertebrates. Few studies have ever addressed the hormetic response in the context of parental care (Noguera *et al.* 2011) and we ultimately still need to know whether or not parents can actively inflict stress on their offspring and what the impact of such potential stress-inducing strategies on offspring fitness is.

The study of the control of signalling systems has opened new avenues of research, but more studies are required that link environmental cues with specific control systems. The opportunities to manipulate care by offspring, by partners or by reproductive parasites will also depend on the control systems of parental decisions, including transduction and molecular signalling pathways. Constraints in these regulatory mechanisms need to be explored since they may explain parental decisions and exactly why decisions may be not optimal in some circumstances (McNamara & Houston 2009). Moreover, we need to know whether the physiological signalling pathways that activate parental behaviour - thereby controlling decisions and resource allocation - are modulating costs or whether they are *per se* costly (Lessells 2008).

To conclude, studies of both phenotypic correlations and manipulative experiments have led to many relevant advances. However, approaches based on quantitative genetics or selection experiments aimed at disentangling genetic architecture (Reznick 1992) are still restricted to analyses of life history traits (e.g. Roff 2002) and little work has been conducted on the mechanisms underpinning these traits to date (see Kim et al. 2010). The challenge for the future is to solve this deficiency.

References

Adkins-Regan, E. (2005). Hormones and animal social behavior. Princeton University Press, Princeton, NJ.

Alisauskas, R.T. and Ankney, C.D. (1994). Nutrition of breeding female ruddy ducks: the role of nutrient reserves. *Condor,* 96, 878-97.

Alonso-Alvarez, C., Bertrand, S., Devevey, G., Prost, J., Faivre, B. and Sorci, G. (2004). Increased susceptibility to oxidative stress as a proximate cost of reproduction. *Ecology Letters*, 7, 363-68.

Alonso-Alvarez, C., Bertrand, S., Devevey, G., *et al.* (2006). An experimental manipulation of life-history trajectories and resistance to oxidative stress. *Evolution*, 60, 1913–24.

Angelier, F. and Chastel, O. (2009). General and Comparative Endocrinology Stress, prolactin and parental investment in birds: A review. *General and Comparative Endocrinology*, 163, 142-8.

Ardia, D.R., Schat, K. and Winkler, D.W. (2003). Reproductive effort reduces long-term immune function in breeding tree swallows (*Tachycineta bicolor*). *Proceedings of the Royal Society B*, 270, 1679-83.

Arnold, K. E., Ramsay, S L., Donaldson, C. and Adam, A. (2007). Parental prey selection affects risk-taking behaviour and spatial learning in avian offspring. *Proceedings of the Royal Society B*, 274, 2563–9.

Aron, S., Keller, L. and Passera, L. (2001). Role of resource availability on sex, caste and reproductive allocation ratios in the Argentine ant *Linepithema humile*. *Journal of Animal Ecology*, 70, 831-9.

Badyaev, A.V. (2009) Evolutionary significance of phenotypic accommodation in novel environments: an empirical test of the Baldwin effect. *Philosophical Transactions of the Royal Society B*, 364, 1125–41.

Baeza, J.A. and Fernandez, M. (2002). Active brood care in *Cancer setosus* (Crustacea: Decapoda): the relationship between female behaviour, embryo oxygen consumption and the cost of brooding. *Functional Ecology*, 16, 241-51.

Balshine-Earn, S. and Earn, D.J.D. (1998). On the evolutionary pathway of parental care in mouth-brooding cichlid fishes. *Proceedings of the Royal Society B*, 265, 2217-22.

Barnes, A. I. and Partridge, L. (2003). Costing reproduction. *Animal Behaviour,* 66, 199–204.

Blanckenhorn, W. (1994). Fitness consequences of alternative life histories in water striders, *Aquarius remigis* (Heteroptera: Gerridae). *Oecologia*, 97, 354–65.

Blount, J.D., Metcalfe, N.B., Arnold, K. E., Surai, P.F. and Monaghan, P. (2006). Effects of neonatal nutrition on adult reproduction in a passerine bird. *Ibis*, 148, 509–14..

Bókony, V., Lendvai, A.Z., Liker, A., Angelier, F., Wingfield, J.C. and Chastel, O. (2009). Stress response and the value of reproduction: are birds prudent parents? *American Naturalist*, 173, 589-98.

Bonier, F., Martin, P.R., Moore, I.T. and Wingfield, J.C. (2009) Do baseline glucocorticoids predict fitness? *Trends in Ecology and Evolution*, 24, 634–42.

Bortolotti, G.R., Negro, J.J., Surai, P.F. and Prieto, P. (2003). Carotenoids in eggs and plasma of red-legged partridges: effects of diet and reproductive output. *Physiological and Biochemical Zoology*, 76, 367-74.

Boulinier, T. and Staszewski, V. (2008). Maternal transfer of anti-bodies: raising immuno-ecology issues. *Trends in Ecology and Evolution*, 23, 282–8.

Bourgeon, S., Le Maho, Y. and Raclot, T. (2009). Proximate and ultimate mechanisms underlying immunosuppression during the incubation fast in female eiders: roles of triiodothyronine and corticosterone. *General and Comparative Endocrinology*, 163, 77-82.

Breuner, C.W., Patterson, S.H. and Hahn, T.P. (2008). In search of relationships between the acute adrenocortical response and fitness. *General and Comparative Endocrinology*, 157, 288–95.

Brown, C. and Laland, K.N. (2001). Social learning and life skills training for hatchery reared fish. *Journal of Fish*

Biology, 59, 471-93.

Brown, J.L., Morales, V. and Summers, K. (2010). A key ecological trait drove the evolution of biparental care and monogamy in an amphibian. *American naturalist*, 175, 436-46.

Challet, E., Le Maho, Y., Robin, J.-P., Malan, A. and Cherel, Y. (1995). Involvement of corticosterone in the fasting-induced rise in protein utilization and locomotor activity. *Pharmacology, Biochemistry and Behaviour*, 50, 405–12.

Cherel Y., Hobson K.A. and Weimerskirch H. (2005). Using stable isotopes to study resource acquisition and allocation in procellariiform seabirds. *Oecologia*, 145, 533-40.

Clark, C.W. and Mangel, M. (2000). *Dynamic state variable models in Ecology.* Oxford University Press, Oxford, UK.

Clutton-Brock, T.H. (1991). *The Evolution of Parental Care*. Princeton University Press, Princeton.

Costantini, D., Metcalfe, N.B. and Monaghan, P. (2010). Ecological processes in a hormetic framework. *Ecology letters*, 13, 1435-47.

Cox, R.M. and Calsbeek, R. (2010). Severe costs of reproduction persist in Anolis lizards despite the evolution of a single-egg clutch. *Evolution*, 64, 1321–30.

Cox, R.M., Parker, E.U., Cheney, D.M., Liebl, A.L., Martin, L.B. and Calsbeek, R. (2010). Experimental evidence for physiological costs underlying the trade-off between reproduction and survival. *Functional Ecology*, 24, 1262-69.

Curio, E. (1993). Proximate and developmental aspects of antipredator behavior. *Advances in the Study of Behavior*, 22, 135-238.

Davies, N.B. and Welbergen, J.A., (2009). Social transmission of a host defense against cuckoo parasitism. Science, 324, 1318-20

Deeming, D.C. (2004). Reptilian incubation: environment, evolution and behaviour. Nottingham University Press, Nottingham.

Deerenberg, C., Pen, I., Dijkstra, C., Arkies, B.J., Visser, G.H. and Daan, S. (1995). Parental energy expenditure in relation to manipulated brood size in the European kestrel. Zoology, Analysis of Complex Systems, 99, 38-47.

Descamps, S., Gilchrist, H.G., Joel, B., Buttler, E.I. and Forbes, M.R. (2009). Costs of reproduction in a long-lived bird: large clutch size is associated with low survival in the presence of a highly virulent disease. Biology Letters, 5, 278-81.

Dijkstra, C., Bult, A., Bijlsma, S. and Daan, S. (1990). Brood size manipulations in the kestrel (*Falco tinnunculus*): effects on offspring and parent survival. *Journal of Animal*, 59, 269-85.

Doughty, P. and Shine, R. (1998). Reproductive energy allocation and long-term energy stores in a viviparous lizard (*Eulamprus tympanum*). *Ecology*, 79, 1073-83.

Edward, D.A. and Chapman, T. (2011). Mechanisms underlaying reproductive trade-offs: costs of reproduction. In T. Flatt and A. Heyland eds. *Mechanisms of life history evolution*. Oxford University Press, Oxford.

Fedorka, K.M., Zuk, M. and Mousseau, T.A. (2004). Immune suppression and the cost of reproduction in the ground cricket, *Allonemobius socius*. *Evolution*, 58, 2478-85.

Finkel, T. and Holbrook, N.J. (2000) Oxidants, oxidative stress and the biology of aging. *Nature*, 408, 239–47.

Freeman, M.E., Kanyicska, B., Lerant, A. and Nagy, G. (2000). Prolactin: structure, function, and regulation of secretion. *Physiological Reviews*, 80, 1523-1631.

Freitak, D., Heckel D.G. and Vogel, H. (2009). Dietary-dependent trans-generational immune priming in an insect herbivore. *Proceedings of the Royal Society B,* 276, 2617–24.

French, S.S., DeNardo, D.F. and Moore, M.C. (2007). Trade-offs between the reproductive and immune systems: facultative responses to resources or obligate responses to reproduction? *American Naturalist*, 170, 79-89.

French, S.S., Greives, T.J., Zysling, D.A., Chester, E.M. and Demas, G.E. (2009). Leptin increases maternal investment. *Proceedings of the Royals Society B,* 276, 4003-11.

Fronstin, R.B. and Hatle, J.D. (2008). A cumulative feeding threshold required for vitellogenesis can be obviated with juvenile hormone treatment in lubber grasshoppers. *Journal of Experimental Biology*, 211, 79-85.

Ghalambor, C. K., Reznick, D.N. and Walker, J.A. (2004). Constraints on adaptive evolution: the functional trade-off between reproduction and fast-start swimming performance in the Trinidadian guppy (*Poecilia reticulada*). American Naturalist, 164, 38–50.

Giesing, E., Suski, C.D. Warner, R.E. and Bell, A.M. (2011). Female sticklebacks transfer information via eggs: Effects of maternal experience with predators on offspring. *Proceedings of the Royal Society B*, 278, 1753-9.

Gil, D. (2008). Hormones in avian eggs: Physiology, ecology and behavior. *Advances in the Study of Behavior,* 38, 337-98.

Gittleman, J.L. and Thompson, S.D. (1988). Energy Allocation in Mammalian Reproduction. *American Zoologist*, 28, 863-75.

Golet, G.H., Irons, D.B. and Estes, J.A. (1998). Survival costs of chick rearing in black-legged kittiwakes. *Journal of Animal Ecology*, 67, 827-41.

Golet, G.H., Schmutz, J.A, Irons, D.B. and Estes, J.A. (2004). Determinants of reproductive costs in the long-lived black-legged kittiwake: a multiyear experiment. *Ecological Monographs*, 74, 353-72.

Grafen, A. (1988). On the uses of data on lifetime reproductive success. In T.H. Clutton-Brock ed. *Reproductive Success,* pp. 454–71. University of Chicago Press, Chicago.

Grandison, R.C., Piper, M.D.W. and Partridge, L. (2009). Amino-acid imbalance explains extension of lifespan by dietary restriction in *Drosophila*. *Nature*, 462, 1061-4.

Green, B.S. and McCormick, M.I. (2005). O_2 replenishment to fish nests: males adjust brood care to ambient conditions and brood development. *Behavioral Ecology*, 16, 389-97.

Grindstaff, J.L., Brodie, E.D. and Ketterson, E.D. (2003). Immune function across generations: integrating mechanism and evolutionary process in maternal antibody transmission. *Proceedings of the Royal Society B*, 270, 2309-19.

Groothuis, T.G.G., Müller, W., von Engelhardt, N., Carere, C. and Eising, C. (2005). Maternal hormones as a tool to adjust offspring phenotype in avian species. *Neuroscience and Biobehavioral Reviews,* 29, 329-52

Hanssen, S.A., Hasselquist, D., Folstad, I. and Erikstad, K.E. (2005). Cost of reproduction in a long-lived bird: incubation effort reduces immune function and future reproduction. *Proceedings of the Royal Society B*, 272, 1039-46.

Harshman, L. and Zera, A.J. (2007). The cost of reproduction: the devil in the details. *Trends in Ecology and Evolution*, 22, 80–6.

Henson, M.C. and Castracane, V.D. (2003). *Leptin and Reproduction*. M.C. Henson and V.D. Castracane, eds. Kluwer Academic/Plenum Publishers, New York.

Hinde, C.A., Johnstone, R.A and Kilner, R.M. (2010). Parent-offspring conflict and coadaptation. *Science*, 327, 1373-76.

Ho, D.H. and Burggren, W.W. (2010). Epigenetics and transgenerational transfer: a physiological perspective. *The Journal of Experimental Biology,* 213, 3-16.

Hoover, J.P. and Reetz, M.J. (2006). Brood parasitism increases provisioning rate, and reduces offspring recruitment and adult return rates, in a cowbird host. *Oecologia*, 149, 165-73.

Hoppitt, W.J.E. Brown, G.R., Kendal, R., Rendell, L., Thornton, A., Webster, M.M. and Laland, K.N. (2008). Lessons from animal teaching. *Trends in Ecology and Evolution*, 23, 486-93.

Houston, A.I. and McNamara, J.M. (1999) *Models of Adaptive Behaviour.* Cambridge University Press

Jacobsen, K.-O., Erikstad, K.E. and Saether, B.-E. (1995). An experimental study of the costs of reproduction in the kittiwake *Rissa Tridactyla*. *Ecology*, 76, 1636-42.

Jones, J.C. and Oldroyd, B.P. (2007). Nest thermoregulation in social insects. *Advances in Insect Physiology*, 33, 153-91.

Kalmbach, E., Griffiths, R., Crane, J.E. and Furness, R.W. (2004) Effects of experimentally increased egg production on female body condition and laying dates in the great skua *Stercorarius skua*. *Journal of Avian Biology*, 35, 501-14.

Kim S-.Y., Velando, A., Sorci, G. and Alonso-Alvarez, C. (2010). Genetic correlation between resistance to oxidative stress and reproductive life span in a bird species. Evolution, 64, 852-857.

Knowles, S.C.L., Nakagawa, S. and Sheldon, B.C. (2009). Elevated reproductive effort increases blood parasitaemia and decreases immune function in birds: a meta-regression approach. *Functional Ecology*, 23, 405–15.

Landwer, A.J. (1994). Manipulation of egg production reveals costs of reproduction in the tree lizard (*Urosaurus ornatus*). *Oecologia*, 100, 243-9.

Law, A.J., Pei, Q., Walker, M. *et al.* (2009). Early parental deprivation in the marmoset monkey produces long-term changes in hippocampal expression of genes involved in synaptic plasticity and implicated in mood disorder. Neuropsychopharmacology, 34, 1381-94

Lee, K.P., Simpson, S.J., Clissold, F.J. *et al.* (2008). Lifespan and reproduction in *Drosophila*: new insights from nutritional geometry. *Proceedings of the National Academy of Science USA*, 105, 2498–503.

Lendvai, A.Z., Giraudeau, M. and Chastel, O. (2007) Reproduction and modulation of the stress response: an experimental test in the house sparrow. *Proceedings of the Royal Society B*, 274, 391-7.

Leroi, A.M. (2001). Molecular signals versus the Loi de Balancement. *Trends in Ecology and Evolution,* 16, 24–9.

Lessells, C.M. (1986). Brood size in Canada geese: a manipulation experiment. *Journal of Animal Ecology*, 55, 669-89.

Lessells, C.(K.)M. (2008). Neuroendocrine control of life histories: what do we need to know to understand the evolution of phenotypic plasticity? *Philosophical Transactions of the Royal Society B*, 363, 1589-98.

Li, D. and Jackson, R.R. (2003). A predator's preference for egg-carrying prey: a novel cost of parental care. *Behavioral Ecology and Sociobiology*, 55, 129-36.

Lõhmus, M. and Björklund, M. (2009). Leptin affects life history decisions in a passerine bird: a field experiment. *PloS One*, 4, e4602.

Lyon, B.E. and Eadie, J.M. (2008). Conspecific brood parasitism in birds: a life-history perspective. *Annual Review of Ecology, Evolution, and Systematics*, 39, 343-63.

Madsen, T. and Shine, R. (1993). Costs of reproduction in a population of European adders. *Oecologia*, 94, 488-95.

Magee, S.E., Neff, B.D. and Knapp, R. (2006). Plasma levels of androgens and cortisol in relation to breeding behavior in parental male bluegill sunfish, Lepomis macrochirus. *Hormones and Behavior*, 49, 598-609.

Marshall, D.J. (2008). Transgenerational plasticity in the sea: context-dependent maternal effects across the life history. *Ecology*, 89, 418–27.

Mateo, J.M., and Holmes, W.G. (1997). Development of alarm-call responses in Belding's ground squirrels: The role of dams. *Animal Behaviour*, 54, 509–24.

McEwen, B. and Wingfield, J.C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, 43, 2-15.

McGlothlin, J.W., Jawor, J.M. and Ketterson, E.D. (2007). Natural variation in a testosterone-mediated trade-off between mating effort and parental effort. *American Naturalist*, 170, 864-75.

McGraw, K.J., Adkins-Regan, E. and Parker, R.S. (2005). Maternally derived carotenoid pigments affect offspring survival, sex-ratio, and sexual attractiveness in a colorful songbird. *Naturwissenschaften*, 92, 375–80.

McNamara, J.M, and Houston A.I. (2009). Integrating function and mechanism. *Trends in Ecology and Evolution,* 24, 670-5.

McNamara, J.M. and Houston, A.I. (1986). The common currency for behavioral decisions. *American Naturalist,* 127, 358–78.

Medina, K.L., Smithson, G. and Kincade, P.W. (1993). Suppression of B lymphopoiesis during normal pregnancy. *The Journal of Experimental Medicine*, 178, 1507-15.

Mërila, J. and Hemborg, C. (2000). Fitness and feather wear in the Collared Flycatcher *Ficedula albicollis*. *Journal of Avian Biology*, 31, 504-10.

Merino, S., Moreno, J., Tomás, G., *et al.* (2006) Effects of parental effort on blood stress protein HSP60 and immunoglobulins in female blue tits: a brood size manipulation experiment. *Journal of Animal Ecology*, 75, 1147–53.

Metcalfe, N.B. and Alonso-Alvarez, C. (2010) Oxidative stress as a life-history constraint: the role of reactive oxygen species in shaping phenotypes from conception to death. *Functional Ecology*, 24, 984-96.

Miles, D.B., Sinervo, B. and Frankino, W.A. (2000). Reproductive burden, locomotor performance, and the cost of reproduction in free ranging lizards. *Evolution*, 54, 1386-95.

Monaghan, P. (2008). Early growth conditions, phenotypic development and environmental change. *Philosophical transactions of the Royal Society B,* 363, 1635-45

Moore, I.T. and Hopkins, W.A. (2009). Interactions and trade-offs among physiological determinants of performance and reproductive success. *Integrative and Comparative Biology*, 49, 441-51.

Mousseau, T.A. and Fox, C.W. (1998). Maternal effects as adaptations. Oxford University Press, Oxford, UK.

Myatt, L. and Cui, X.L. (2004). Oxidative stress in the placenta. *Histochemistry and Cell Biology*, 122, 369–82.

Nilsson, J.Å. and Svensson, E. (1996). The cost of reproduction: a new link between current reproductive effort and future reproductive success. *Proceedings Proceedings of the Royal Society B*, 263, 711-4.

Noguera, J.C., Lores, M., Alonso-Álvarez, C. and Velando, A. (2011). Thrifty development: early-life diet restriction reduces oxidative damage during later growth. *Functional Ecology,* In Press.

Nordling, D., Andersson, M., Zohari, S. and Lars, G. (1998). Reproductive effort reduces specific immune response and parasite resistance. *Proceedings of the Royal Society B*, 265, 1291-8.

Nur, N. (1984). The consequences of brood size for breeding blue tits. I. Adult survival, weight change and the cost of reproduction. *Journal of Animal Ecology*, 53, 479-96.

Nur, N. (1988). The consequences of brood size for breeding blue tits. III. Measuring the cost of reproduction: survival, future fecundity, and differential dispersal. *Evolution*, 42, 351-62.

Olsson, M., Shine, R. and Bak-Olsson, E. (2000). Locomotor impairment of gravid lizards: is the burden physical or physiological? *Journal of Evolutionary Biology*, 13, 263-8.

Östlund-Nilsson, S. and Nilsson, G.E. (2004). Breathing with a mouth full of eggs: respiratory consequences of mouthbrooding in cardinalfish. *Proceedings of the Royal Society B*, 271, 1015-22.

Otero, M., Lago, R. and Lago, F., *et al.* (2005). Leptin, from fat to inflammation: old questions and new insights. *FEBS Letters*, 579, 295-301.

Page, R.E. and Amdam, G.V. (2007). The making of a social insect: developmental architectures of social design. *BioEssays*, 29, 334-43.

Parker, G.A. and Maynard Smith, J. (1990). Optimality Theory in Evolutionary Biology. *Nature*, 348, 27-33.

Peluc, S.I., Sillett, T.S., Rotenberry, J.T. and Ghalambor, C.K. (2008). Adaptive phenotypic plasticity in an island songbird exposed to a novel predation risk. *Behavioral Ecology*, 19, 830-5.

Pérez-Rodríguez, L. (2009). Carotenoids in evolutionary ecology: re-evaluating the antioxidant role. Bioessays, 31, 1116–26.

Perry, C. and Roitberg, D. (2006). Trophic egg laying: hypotheses and tests. *Oikos*, 112, 706–14.

Pigilucci, M. (2001.) *Phenotypic plasticity.* John Hopkins University Press, Baltimore, MD.

Pike, T.W., Blount, J.D., Lindström, J., and Metcalfe, N.B. (2007). Dietary carotenoid availability influences a male's ability to provide parental care. *Behavioral Ecology*, 18, 1100-5.

Reguera, P. and Gomendio, M. (1999). Predation costs associated with parental care in the golden egg bug *Phyllomorpha laciniata* (Heteroptera: Coreidae). *Behavioral Ecology*, 10, 541-4.

Reid, W.V. (1987). The cost of reproduction in the glaucous-winged gull. *Oecologia*, 74, 458-67.

Reznick, D. (1992). Measuring the costs of reproduction. *Trends in Ecology and Evolution*, 7, 42-5.

Roff, D.A. (2002). *Life History Evolution*. Sinauer Associates, Sunderland, MA.

Roth, O., Joop, G., Eggert, H. *et al.* (2010). Paternally derived immune priming for offspring in the red flour beetle, *Tribolium castaneum*. *Journal of Animal Ecology*, 79, 403-13.

Sabat, A.M. (1994). Costs and benefits of parental effort in a brood-guarding fish (*Ambloplites rupestris*, Centrarchidae). *Behavioral Ecology*, 5, 195-201.

Salmon, A.B., Marx, D.B. and Harshman, L.G. (2001). A cost of reproduction in *Drosophila melanogaster*: stress susceptibility. *Evolution*, 55, 1600-8.

Salomon, M., Mayntz, D., Toft, S. and Lubin, Y. (2011). Maternal nutrition affects offspring performance via maternal care in a subsocial spider. *Behavioral Ecology and Sociobiology*, in press. DOI:10.1007/s00265-010-1132-8

Schradin, C. and Anzenberger, G. (2001). Costs of infant carrying in common marmosets, *Callithrix jacchus*: an experimental analysis. *Animal Behaviour*, 62, 289-95.

Schradin, C., Schneider, C. and Yuen, C.H. (2009). Age at puberty in male African striped mice: the impact of food, population density and the presence of the father. Functional Ecology, 23, 1004-13.

Shaffer, L. and Formanowicz, D.R. Jr. (1996). A cost of viviparity and parental care in scorpions: reduced sprint speed and behavioural compensation. *Animal Behaviour*, 51, 1017-24.

Simmons, L.W. (2011). Allocation of maternal- and ejaculate-derived proteins to reproduction in female crickets, *Teleogryllus oceanicus*. *Journal of evolutionary biology*, 24, 132-8.

Sorensen, J.G., Kristensen, T.N. and Loeschcke, V. (2003). The evolutionary and ecological role of heat shock proteins. *Ecology Letters*, 6, 1025-37.

Speakman, J.R. (2001). *Body Composition Analysis of Animals: a handbook of non-destructive methods.* Cambridge University Press, Cambridge.

Speakman, J.R. (2008). The physiological costs of reproduction in small mammals. *Philosophical Transactions of the Royal Society of London B,*363, 375-98.

Spée, M., Beaulieu, M., Dervaux, A., Chastel, O., Le Maho, Y. and Raclot, T. (2010). Should I stay or should I go? Hormonal control of nest abandonment in a long-lived bird, the Adélie penguin. *Hormones and Behavior*, 58, 762-8.

Stearns, S.C. (1992). *The evolution of life histories.* Oxford University Press Oxford, UK.

Stjernman, M., Råberg, L. and Nilsson, J.-A. (2004). Survival costs of reproduction in the blue tit (*Parus caeruleus*): a role for blood parasites? *Proceedings of the Royal Society B*, 271, 2387-94.

Surai, P.F. (2002). *Natural antioxidants in avian nutrition and reproduction*. Nottingham University Press, Nottingham

Suzuki, S., Kitamura, M. and Matsubayashi, K. (2005). Matriphagy in the hump earwig, *Anechura harmandi* (Dermaptera : Forficulidae), increases the survival rates of the offspring. *Journal of Ethology*, 23, 211-13.

Svensson, I. (1988). Reproductive costs in two sex-role reversed pipefish species (Syngnathidae). *Journal of Animal Ecology*, 57, 929-42.

Tallamy, D.W. (2005). Egg dumping in insects. *Annual Review of Entomology*, 50, 347-70.

Tallamy, D.W. and Denno, R.F. (1982). Life-history trade-offs in *Gargaphia solani* (Hemiptera, Tingidae) – the cost of reproduction. *Ecology*, 63, 616-20.

ten Cate, C., Lea, R., Ballintijn, M. and Sharp, P. (1993). Brood size affects behavior, interclutch interval, LH levels, and weight in ring dove (*Streptopelia risoria*) breeding pairs. *Hormones and Behaviorand behavior*, 27, 539-50.

Tinbergen, J.M. and Verhulst, S. (2000). A fixed energetic ceiling to parental effort in the great tit? *Journal of Animal Ecology*, 69, 323-34.

Török, J., Hegyi, G., Tóth, L. and Könczey, R. (2004). Unpredictable food supply modifies costs of reproduction and hampers individual optimization. *Oecologia*, 141, 432-43.

Trivers, R.L. (1972). Parental investment and sexual selection. In B. Campbell (ed.) *Sexual selection and the descent of man*, 1871-1971, pp 136-79. Aldine, Chicago.

Trumbo, S.T. (2007). Defending young biparentally: female risk-taking with and without a male in the burying beetle, *Nicrophorus pustulatus*. *Behavioral Ecology and Sociobiology*, 61, 1717-23.

Tyndale, S., Letcher, R., Heath, J. and Heath, D. (2008). Why are salmon eggs red? Egg carotenoids and early life survival of Chinook salmon (*Oncorhynchus tshawytscha*). *Evolutionary Ecology Research*, 10, 1187–99.

Uller, T. (2008). Developmental plasticity and the evolution of parental effects. *Trends in Ecology and Evolution*, 23, 432–8.

Upreti, K. and Misro, M. (2002). Evaluation of oxidative stress and enzymatic antioxidant activity in brain during pregnancy and lactation in rats. *Health and Population-Perspectives and Issues*, 25, 105-12.

van Noordwijk, A.J. and de Jong, G. (1986). Acquisition and allocation of resources: their influence on variation in life history tactics. *American Naturalist,* 128, 137–42.

Veasey, J.S., Houston, D.C. and Metcalfe, N.B. (2001). A hidden cost of reproduction: the trade-off between clutch size and escape take-off speed in female zebra finches. *Journal of Animal Ecology*, 70, 20-4.

Wake, M.H. and Dickie, R. (1998). Oviduct structure and function and reproducive modes in amphibians. *Journal of Experimental Zoology*, 282, 477–506.

Wang, Y., Salmon, A.B. and Harshman, L.G. (2001). A cost of reproduction: oxidative stress susceptivility is associated with increased egg production in *Drosophila melanogaster. Experimental Gerontology*, 36, 1349-59.

Weaver, I.C.G., Cervoni, N., Champagne, F.A. *et al.* (2004). Epigenetic programming by maternal behaviour. *Nature Neuroscience*, 7, 847–54.

West-Eberhard, M. (2003). Developmental plasticity and evolution. Oxford University Press, Oxford, UK.

Wiersma, P., Selman, C., Speakman, J.R. and Verhulst, S. (2004). Birds sacrifice oxidative protection for reproduction. *Proceedings of the Royal Society of London B*, 271, S360–3.

Wingfield, J.C. and Sapolsky, R.M. (2003). Reproduction and resistance to stress: when and how. *Journal of Neuroendocrinology*, 15, 711–24.

Witter, M.S. and Cuthill, I.C. (1993). The ecological costs of avian fat storage. *Philosophical transactions of the Royal Society of London B,* 340, 73-92.

Figure 1. A schematic illustration of resource allocation between current reproduction and future reproduction in relation to resource acquisition (bottom panels, from van Noordwijk and de Jong 1986). Within individuals, the resources should invested between competing functions (negative correlation). Among individuals, it will depend on variation in resource acquisition (top panels), and despite of negative genetic correlation in resource allocation, the phenotypic correlation between competitive functions may be positive (right bottom panel).

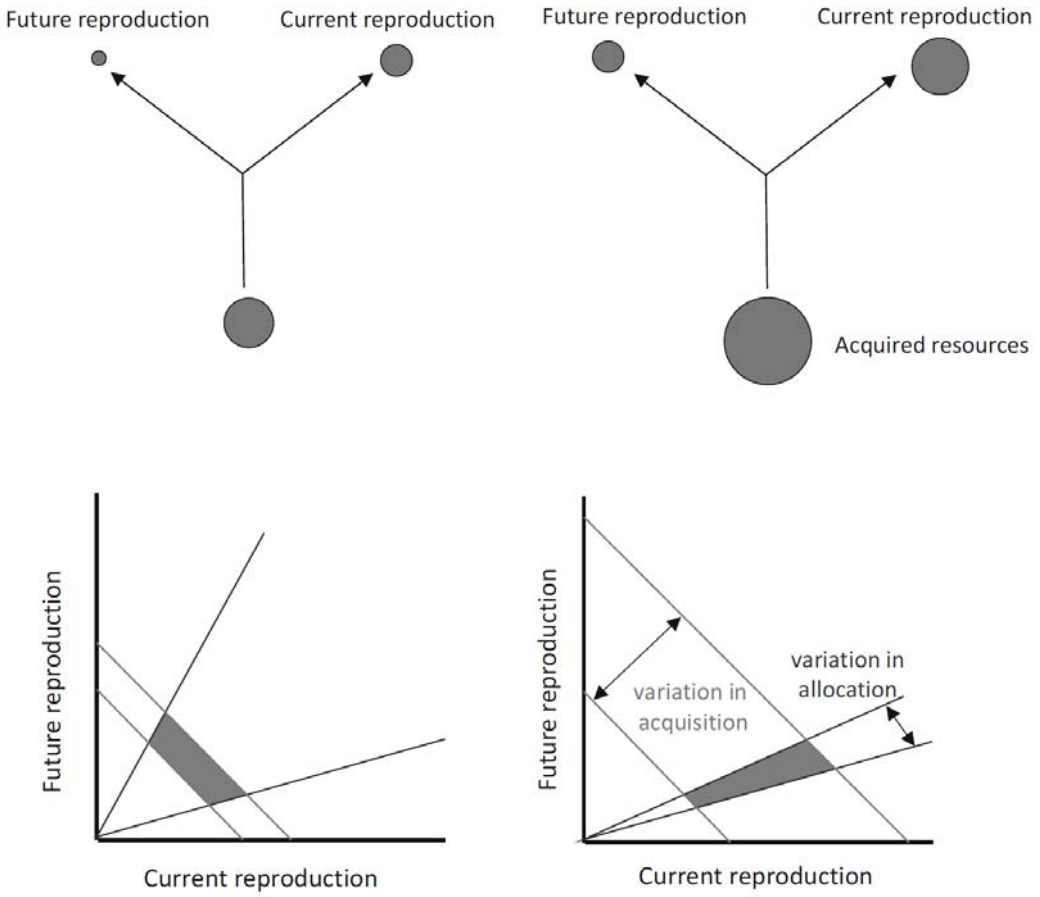

Figure 2. Hypothetical mechanisms linking parental care and somatic maintenance (i.e., survival). The trade-off between these life-history traits may rise from a variety of separate mechanisms. Environmental factors, including food availability, physical conditions, risk of predation or parasitism, and competitor abundance, may impose acquisition costs, limiting macro and micronutrients needed for both functions. Traditionally, the models have been centered on energy limitation, i.e. the allocation of macronutrients between current and future reproduction (Figure 1), but reproduction can also be limited by specific micronutrients. Importantly, parental care may cause direct somatic damage via physiological imbalance (e.g. due to some signalling pathways or stress). Environmental factors governing resource acquisition may also affect physiological homeostasis (e.g. stress). Moreover, the diet composition that maximizes parental care may differ from that maximizing somatic maintenance due to the different damage effects of different subproducts of nutrient metabolism.

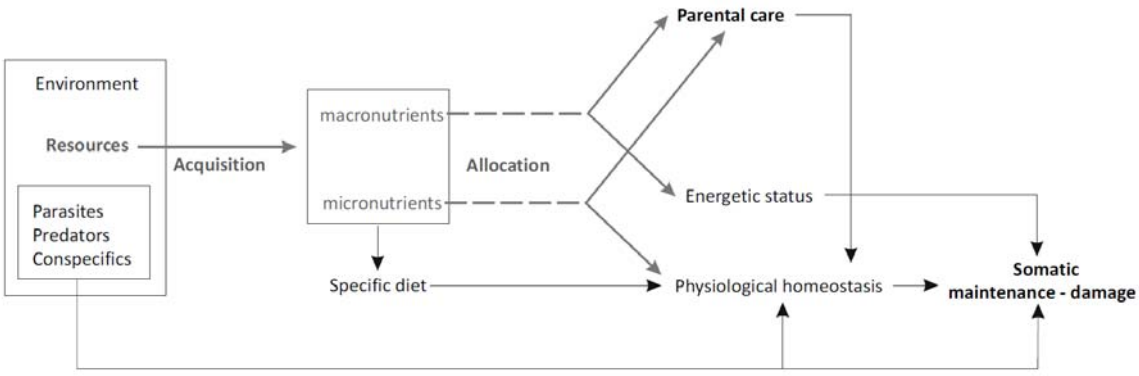

Table 1. Support for a link between parental care, physiological costs, infections and fitness.

Mechanistic Cost	Order	Species	Parental Care	Exp/Corr	Capt/Wild	PC->PHC	PC→FC Reprod	PC→FC Surv	PHC→FC Reprod	PHC→FC Surv	References
Energy expenditure	*Aves*	*Falco tinnunculus*	Brood care and feeding	Exp	Wild	m (f: UT)	n.s.(b)	∗(b)	UT	UT	Dijkstra et al. 1990
		Falco tinnunculus	Brood care and feeding	Exp/Corr	Wild	b	UT	n.s.(b)	UT	∗ (b)	Deeremberg et al. 1995
Body mass loss	*Actinopterygii*	*Ambloplites rupestris*	Brood guarding	Exp/Corr	Wild	m (f: NA)	UT	∗	UT	∗	Sabat et al. 1994
	Reptilia	*Vipera verus*	Egg production and gestation	Corr	Wild	f (m: NA)	NA	∗	UT	∗	Madsen and Shine 1993
		Urosaurus ornatus	Egg production	Exp/Corr	Wild	f (m: NA)	∗	∗	∗	UT	Landwer 1994‡
		Anolis sagrei	Egg production	Exp	Wild	f (m: NA)	UT	∗	UT	UT	Cox and Calsbeek 2010‡
			Egg production	Exp	Wild	f (m: NA)	UT	∗	UT	UT	Cox et al. 2010‡
	Aves	*Stercorarius skua*	Egg production	Exp	Wild	f (m: UT)	∗	n.s.	UT	UT	Kalmbach et al. 2004
		Somateria mollisima	Incubation	Exp	Wild	f (m: NA)	∗	n.s.	UT	UT	Hanssen et al. 2005
		Rissa tridactyla	Incubation, brood care and feeding	Exp	Wild	b	n.s.(b)	∗(b)	UT	UT	Golet et al. 1998
		Rissa tridactyla	Incubation, brood care and feeding	Exp/Corr	Wild	b	∗(b)	∗(b)	∗(b)	n.s.(b)	Golet et al. 2004
		Rissa tridactyla	Brood care and feeding	Exp	Wild	f (m: n.s.)	UT	∗(f, m: n.s.)	UT	UT	Jacobsen et al. 1995
		Branta c. canadensis	Brood care and feeding	Corr	Wild	f (m: n.s.)	∗(b)	n.s.(b)	UT	UT	Lessells 1986
		Larus glaucescens	Brood care and feeding	Exp/Corr	Wild	b	n.s.(b)	∗(b)	UT	n.s.(b)	Reid 1987
		Falco tinnunculus	Brood care and feeding	Exp	Wild	f (m: n.s.)	n.s.	∗(b)	UT	UT	Dijkstra et al. 1990
		Streptopelia risoria	Brood care and feeding	Exp	Capt	b	∗(b)	UT	UT	UT	ten Cate et al. 1993
		Ficedula albicollis	Brood care and feeding	Exp	Wild	b	∗ (f, mUT)	n.s.(f, mUT)	UT	UT	Török et al. 2004
		Cyanistes caeruleus	Brood care and feeding	Exp	Wild	f (m: n.s.)	∗(b)	∗(f, m n.s.)	UT	∗(f, m n.s.)	Nur 1984, Nur 1988†
		Cyanistes caeruleus	Brood care and feeding	Exp	Wild	f (m: UT)	UT	∗	UT	n.s.	Stjernman et al. 2004
		Parus major	Brood care and feeding	Exp/Corr	Wild	b	∗(b)	UT	UT	∗(f, mUT)	Tinbergen and Verhulst 2000
Loss of energy body stores	*Reptilia*	*Eulamprus tympanum*	Overall reproduction	Corr	Capt/Wild	f (m: NA)	UT	n.s.	∗	UT	Doughty and Shine 1998
	Aves	*Stercorarius skua*	Egg production	Exp	Wild	f (m: NA)	∗	n.s.	UT	UT	Kalmbach et al. 2004
Micronutrient adjustment	*Insecta*	*Drosophila melanogaster*	Egg production	Exp	Capt	f (m: NA)	UT	UT	UT	*	Grandison et al. 2009
Physiological stress	*Aves*	*Rissa tridactyla*	Incubation, brood care and feeding	Exp	Wild	b	∗(b)	∗(b)	UT	n.s.(b)	Golet et al. 2004††
Oxidative stress	*Insecta*	*Drosophila melanogaster*	Egg production	Exp	Capt	f (m: NA)	UT	∗	NA	∗	Salmon et al. 2001?
		Drosophila melanogaster	Egg production	Exp	Capt	f (m: NA)	UT	∗	NA	∗	Wang et al. 2001?

	Aves	*Taniopygia guttata*	Overall reproduction	Corr	Capt	b	UT	UT	UT	∗(b)	Alonso-Alvarez et al. 2006#?
Immunosuppression	*Reptilia*	*Anolis sagrei*	Egg production	Exp	Wild	f (m: NA)	UT	∗	UT	UT	Cox et al. 2010
	Aves	*Somateria mollissima*	Incubation	Exp	Wild	f (m: NA)	∗	n.s.	UT	UT	Hanssen et al. 2005
		Tachycineta bicolor	Brood care and feeding	Exp/Corr	Wild	f (m: UT)	UT	UT	UT	∗	Ardia et al. 2003

To create this table a systematic simultaneous search (Web of Science, Thompson Reuters) of the term `fitness´ plus the truncated term `cost´ plus parental care (in any of its potential terms), and plus each potential mechanistic cost (e.g. energetic and non-energetic costs, predation, etc) was performed, using different truncated combinations. This search produced about 500 references.

Abbreviations and notes:

PC→PHC: Parental care inducing the physiological cost

PC→FC: Parental care inducing fitness costs (reproductive cost or reduced survival)

PHC→FC: link between physiological and fitness costs

m: male; f: female; b: both sexes.

NA: not applies

UT: untested

‡ Body growth as measure of body mass variability

†Both studies report findings on the same dataset

††The survival cost of the manipulation was not detected in the subsample where stress was tested

Resistance to oxidative stress was negatively associated with the number of previous breeding events, and predicted subsequent short-term longevity

? Design limitations (see section 3.3.2)

$Individuals whose parental effort was manipulated were not the same that those tested for fitness effects