Do maternal effects influence phenotypic traits in a cooperatively breeding mammal?

Submitted by

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Thesis Summary

Animals within populations show considerable variation in physiological and behavioural traits. These phenotypic differences can be profoundly influenced by the ecological and social conditions experienced in early life. Usually, adverse early life conditions, such as low food availability, will constrain offspring development. However, recent studies suggest that in some contexts suboptimal developmental conditions may be associated with fitness benefits. In this thesis I consider the ultimate and proximate mechanisms underlying individual variation in a cooperatively breeding mammal. I use hormone samples and the long-term dataset from the banded mongoose research project to investigate the social factors that influence early life stress and how patterns of reproductive investment change with maternal age. In chapter one I review current theories on the function of glucocorticoid (GC) hormones and give an overview of maternal stress and its effect on offspring development and physiology. In addition, I discuss the influence of maternal effects on cooperatively breeding systems. In chapter two, I introduce the study population and provide a general outline of the methods used in data collection and analysis. In chapter three, I test how maternal effects, specifically maternal rank, influence GC concentrations in banded mongoose pups and whether elevated early life GC is associated with mortality risk. I also consider whether the amount of cooperative care received by offspring in early life affects their GC concentrations. In chapter four, I use a larger dataset to consider how maternal effects influence adult life-history traits. I examine the influence that maternal age has on offspring survival, longevity and reproductive success and consider whether any effects might be sex-specific. I also ask whether age-related optimal maternal investment strategies can influence offspring sex ratios. In chapter five, I synthesis my findings and attempt to draw on the wider implications and make suggestions for future study.

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Author's Declaration

All chapters in this thesis were written by Dave Seager with comments from Michael Cant, who provided guidance throughout. The wild study population of banded mongooses (*Mungo mungo*) is part of a long-term project run by Michael Cant from the University of Exeter (Cornwall Campus). Michael Cant's research is supported by funding from the European Research Council Starting Grant (309249) and Natural Environment Research Council (UK) Standard Grant (NE/J010278/1).

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All research procedures were approved by the Uganda Wildlife Authority and Uganda National Council for Science and Technology, and adhered to the Guidelines for the Treatment of Animals in Behavioural Research and Teaching, published by the Association for the Study of Animal Behaviour. All research was approved by the Ethical Review Committee of the University of Exeter.

All photographs in this thesis were taken by and are the property of Dave Seager.

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Chapter 1: General Introduction

1.1 Abstract

Individuals within populations show considerable variation in phenotypic traits, including their response to external stressors. Physiological stress is vital in coordinating organismal response to changing social or ecological conditions but can prove maladaptive in the event of prolonged exposure. Although understanding of the processes underlying phenotypic variation remains limited, conditions during development are likely to profoundly influence adult phenotype, including the neuroendocrine stress response. Being born in periods with low resources is traditionally associated with poor outcomes for offspring, but recent work suggests that a challenging rearing environment can become adaptive in specific contexts. The proximate mechanisms mediating such adaptive processes are difficult to establish but it is predicted that maternal hormonal signalling may provide a link between the in-utero environment and offspring phenotype. This review summarises work considering the physiological function of the neuroendocrine stress response and how it can be influenced by ecological and social conditions. I also examine non-genetic drivers of phenotypic variation, in particular maternal effects, and how they can influence the stress hormone concentrations of offspring. I particularly focus on the potential effects of offspring exposure to maternally derived stress hormones and how this 'maternal stress' may mediate transgenerational phenotypic plasticity. As cooperatively breeding species are highlighted as good models for studying maternal effects I lastly consider how the care provided by non-breeding helpers may influence the effect of maternal condition on developing offspring.

1.2 Introduction

Individuals within species, even where closely related, can show profound differences in lifespan, fitness and behaviour (Bolnick et al 2011, Fay et al 2017). That variation in individual quality (or heterogeneity) influences life-history and reproductive traits forms a theoretical basis of evolution by natural selection (Darwin 1859). To understand phenotypic variation within diverse populations it is important to consider both proximate and ultimate causal mechanisms (Tinbergen 1963, Mayr 1974). Many studies have considered how processes such as plasticity, maternal effects and gene modification can influence phenotypic development (Anacker et al 2014, Jablonka & Raz 2009, Jenkins et al 2014). Of key interest is understanding how organisms adjust their physiology or behaviour in response to social or environmental conditions. Endocrine systems represent good candidate mechanisms for mediating response to external stressors as hormones act quickly and can influence several behavioural phenotypes simultaneously (Sanderson 2012). Hormonal profiles may differ markedly within species (Knapp & Moore 1997) or even social groups (Carlson et al 2006a, 2006b) and can provide valuable insight into the underlying selective pressures and trade-offs that drive phenotypic variation.

The purpose of this review is to summarise current research into factors underlying between-individual variation of phenotypic traits and consider how it may apply to group-living species. I focus on variation in stress hormone concentrations during infancy in a cooperatively breeding mammal, but the concepts proposed may be applied to other phenotypic traits or reproductive systems. I begin in section 1.3 by introducing stress as a biological concept and discussing the challenges imposed on organisms by ecological and social variability. I then consider the physiological mechanisms by which organisms maintain internal stability when faced with external stressors with a particular focus on the neuroendocrine stress response. I conclude this section by examining the frequently reported link between elevated glucocorticoid (GC) concentrations and sub-optimal environmental conditions.

In section 1.4, I consider the role of maternal effects in generating individual variation of phenotypic traits, as a mother's condition is predicted to strongly influence the development and fitness of her offspring. I discuss the human and laboratory studies

that report an association between maternal nutritional state during crucial developmental periods and the development of some adult-onset diseases. This research suggests that an individual's developing stress physiology is vulnerable to impairment from inadequate provision of early life care, which may be linked to poor maternal state. I also highlight some wild systems where poor early life conditions are associated with improved fitness if selection pressure results in weaker animals failing to successfully breed. I conclude the section by considering the potential influence of paternal and grandparental effects on offspring development.

Section 1.5 considers exposure to maternally derived stress hormones during pregnancy (maternal stress) and its potential impact on offspring development. Elevated maternal stress concentrations are generally regarded as costly to offspring as maternal investment may be reduced through impaired physical state or ability to provide care. I examine the physiological mechanisms by which maternal stress is predicted to affect offspring phenotype. Of key interest are lab studies reporting that epigenetic mechanisms can impair offspring physiology as hormone receptor genes are modified by the quality of early life care.

Section 1.6 describes recent work that re-evaluates the view that maternal stress is exclusively maladaptive to offspring. These studies propose that maternal stress may become adaptive in specific contexts, particularly where conditions during pregnancy are likely to match with those experienced in early life. If mothers use pre-natal environmental cues to influence offspring phenotype this may represent a form of developmental plasticity known as Predictive Adaptive Response (PAR). Maternal stress is predicted as a good candidate mechanism for regulating PARs and I consider the trade-offs that may be associated with phenotypic plasticity. I also consider PARs in a wider context, specifically how offspring may respond if maternal cues do not match early life conditions and the potential role of developmental plasticity in adapting to human induced environmental change.

Section 1.7 begins with a brief description of cooperative breeding as a reproductive strategy and highlights why it represents a good system for testing maternal effects. Of key interest here are reports that mothers might obtain information about the social environment and adjust reproductive investment depending on the amount of

care available for their offspring from helpers. I end by discussing whether low quality cooperative care could impair development of offspring neuroendocrine system in the same way that poor maternal care is predicted to.

1.3 Mechanisms and functions of physiological stress.

Stress is broadly defined as an organism's neuroendocrine response to changing conditions. External stimuli that trigger stress are known as stressors and can include intrinsic or extrinsic environmental challenges (Raulo & Dantzer 2018). The short-term physiological reaction to an external stressor is known as acute stress and may trigger a behavioural response to a perceived threat, after which the organism will quickly return to its normal or baseline state. However, stress may become chronic when the demands (or perceived demands) imposed by external stressors exceed the organism's ability to maintain internal stability or homeostasis. This can prove physiologically costly to individuals by increasing biological damage and impairing survival or competitive ability (Epel et al 2004). In wild animals, quantifying stress levels can illustrate the challenges implicit in adapting to changing conditions (Romero et al 2009). In addition, measuring behavioural and physiological responses to external stimuli may reveal why some individuals withstand challenging periods better than others. Recent studies suggest that in competitive or risky environments stress can have positive fitness outcomes whilst remaining physiologically costly (Dantzer et al 2013). However, the effects of extended periods of high stress remain profound. In humans associated disorders include hypertension and eczema (Cohen et al 2007) and animal studies have identified additional problems including impaired growth, immunity and reproductive function (Sapolsky et al 2000, Wingfield et al 1998).

Vertebrate stress response is controlled by the hypothalamic-pituitary-adrenal (HPA) axis. External stimuli trigger the hypothalamus to release corticotrophin-releasing hormone (CRH) which stimulates secretion of adrenocorticotrophic hormone (ACTH) from the pituitary gland. This in turn triggers secretion of glucocorticoids (GCs, such as cortisol and corticosterone) from the adrenal cortex into the bloodstream. GCs are often described as "stress hormones" due to their role in coordinating physiological responses to stressors (Romero 2002, Wingfield 2005). This includes facilitating the

conversion of fat and protein reserves into useable energy whilst temporarily suppressing the immune system. The HPA axis is highly sensitive to environmental variability as unexpected stimuli will trigger the release of GCs. If these stressors prove harmless, hormones already in the bloodstream inhibit further secretion. This negative feedback ensures that hormone concentrations rapidly return to normal and do not reach potentially damaging levels. Release of GCs may prompt behavioural responses that prioritise survival in response to risk. Studies suggest that the developing HPA axis is vulnerable to impairment by intrinsic factors such as resource availability and maternal stress both pre and post-birth (Liu et al 1997). Therefore, individual differences in HPA function can be partly explained by early life conditions and may account for some degree of heterogeneity within populations.

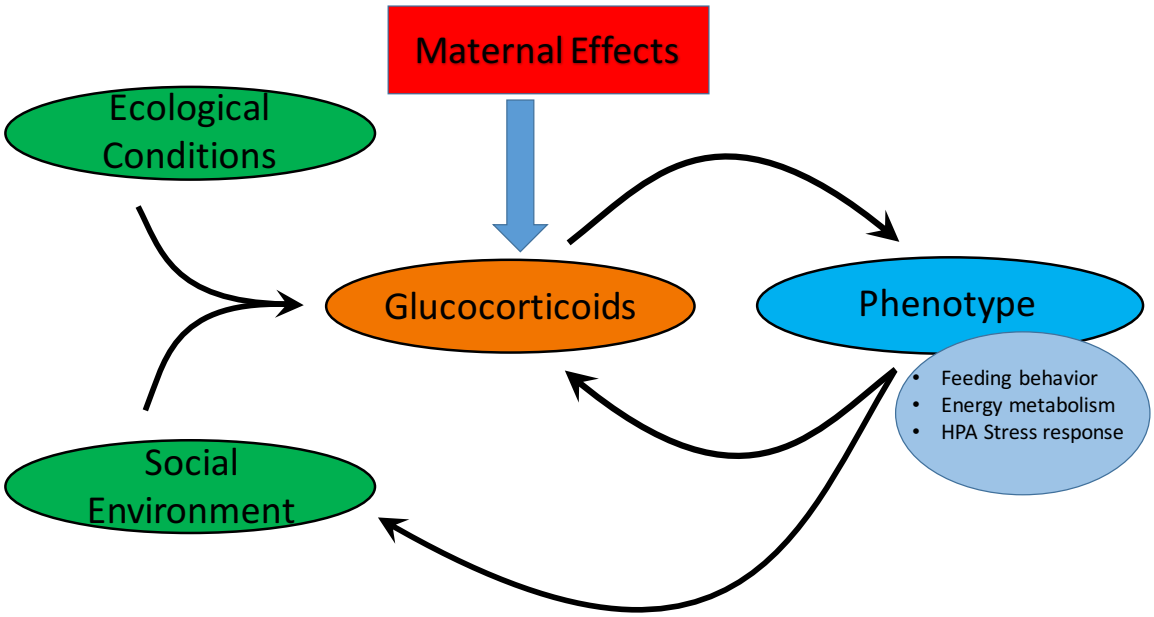

Figure 1; Graphical illustration showing how phenotypic responses to ecological and social conditions may be mediated by glucocorticoid concentrations. External stressors trigger the secretion of GCs, which then affect organism phenotype through behavioural or metabolic changes. Here I am interested in the influence of maternal effects on offspring GC levels and how they may influence phenotypic traits.

The process of achieving homeostasis through physiological or behavioural change is known as allostasis (Sterling & Eyer 1988, McEwen & Wingfield 2003). Environmentally induced changes to energy demand may be associated with predictable seasonal or life-history variation or as an emergency response to unforeseeable threats. An ability to adapt to unpredictable conditions is vital in adjusting to social or environmental variation (McEwen & Wingfield 2010). In group-

living species social and ecological factors are known to affect hormone levels and behaviour (Goymann & Wingfield 2004, Rubenstein 2007, Rubenstein & Shen 2009). GCs play a key role in allostasis by mediating circulating glucose levels (Landys et al 2006, Romero 2004) and are vital in regulating energetically costly activities such as breeding and migration (Romero 2002, Love et al 2012). As such, GCs are predicted to influence state-mediated trade-offs (Love et al 2005) and act as mechanisms underlying life-history evolution (Zera & Harshman 2001). Hormonal measurements can therefore provide insights into the impact of adaptive responses on individual-level fitness and performance, which may be positive, negative or neutral (Breuner et al 2008) depending on ecological context.

Many studies report elevated hormone levels in response to ecological variation (Sheriff et al 2011, Wasser et al 2011). Relatively high GCs may be associated with poor physical state (Romero & Wikelski 2001) when adverse conditions increase local resource competition or food shortages lead to nutritional stress (Compagnucci et al 2002). If energy demands exceed available resources, circulating GC concentrations are predicted to rise in order to regain energetic homeostasis (McEwen & Wingfield 2003). Thus elevated early life GC levels could be associated with both short-term (increased mortality linked to poor physical condition) and long-term costs (reduced fitness, particularly in relation to competitors raised in better nutritional or social conditions). In addition, the impact of these costs will be modulated by individual genotype (de Kloet et al 2005, Lightman 2008) as well as non-genetic processes such as plasticity and maternal effects.

1.4 Maternal effects as drivers of phenotypic variation.

Maternal effects occur when the mother's environment or physical state affects offspring phenotype. Early life social or ecological conditions can influence parental investment strategies and affect developing morphology, physiology and behaviour, with effects persisting into adulthood (Mousseau & Fox 1998, Mousseau et al 2009). The impact on offspring may be positive or negative, depending on ecological context (Groothius et al 2005, Monaghan 2008) and can become adaptive if they improve either offspring or maternal fitness (Marshall & Uller 2007). The degree to

which offspring phenotypic traits are affected may depend on population-level plasticity or heritability of maternal strategies (Badyaev 2008), and levels of parent-offspring conflict (Trivers 1974). In general, resource rich conditions in early-life are predicted to benefit offspring, reported “silver spoon effects” include improved health, survival (Wong & Kolliker 2014, Lindstrom 1999) and fitness (Nussey et al 2007, Lummaa & Clutton-Brock 2002). Similarly, resource poor developmental conditions have been linked with constraints such as high mortality, reduced lifespan and low reproductive success in long-lived species including humans (Nettle 2014), baboons (Lea et al 2015, Tung et al 2016) and elephants (Mumby et al 2015). Offspring born in these sub-optimal developmental conditions may be unable to, or selected against, later compensating for their bad start in life (Metcalfe & Monaghan 2001).

Alternatively, individuals born in apparently adverse early life conditions may show improved survival or reproductive success. Many human and lab studies associate dietary restriction with increased lifespan in the absence of malnutrition (Colman 2009, Masaro 2006). In wild systems this may represent survivor bias if infant mortality is higher in nutritionally poor conditions that impose strong viability selection and high quality individuals are more likely to survive (Chen & Maklakov 2012). For example, Roe deer (*Capreolus capeolus*) showed a sex-specific response to high juvenile mortality, with increased female but reduced male, adult survival (Garrett et al 2015). Sex-biased maternal effects also occur in red deer (*Cervus* elaphus) where females suffer greater reproductive costs when exposed to low temperatures in early life than males (Kruuk et al 1999). Harsh early life conditions can also produce sex-dependent fitness benefits in birds, for example, male superb starlings (*Lamprotonis superbus*) hatched during dry periods were more likely to breed in later life (Rubenstein 2016). Similarly, male great tits (*Parus major)* born in poor years showed improved lifespan and reproductive success, possibly due to stronger early life selection favouring more competitive individuals (Wilkin & Sheldon 2009). Where early life conditions are associated with sex-specific fitness benefits, maternal investment decisions may be dependent on offspring sex. In banded mongooses, males that experienced more variable ecological conditions in early life lived longer and had higher lifetime reproductive success. In contrast, variation in mean conditions influenced males’ relative fertility and lifespan in opposite directions and resulted in no overall effect on reproductive success, suggesting a life-history trade-

off (Marshall et al 2017). These effects were not seen in females who suffered higher mortality when rainfall was low (Marshall et al 2016).

The presence of sex-specific maternal effects suggests that selection may act differently on male and female offspring. For example, maternal stress may have sex-specific impacts on offspring (Schmidt et al 2012, St-Cyr et al 2017) with sons seemingly more susceptible to the effects of exposure to high maternal GC than daughters (Love et al 2005). Differences between the sexes might occur even without differential early life survival in species where traits associated with survival or competitive ability are more closely linked to maternal condition in one sex than the other. Similarly, sexually antagonistic selection may provide a mechanism for maintaining phenotypic variation if males and females show different optimal traits (Bonduriansky & Chenoweth 2009, Cox & Calsbeek 2009). Alternatively, offspring may adaptively adjust life-history trajectories to achieve similar fitness to those born in better conditions (Nettle & Bateson 2015, Taborsky 2006). For example, seychelles warblers (*Acrocephalus sechellensis*) raised in poor environments show reduced lifespan but breed younger to match competitors born in better conditions (Cartwright et al 2013, Hammers et al 2014). Likewise, in humans, women that suffer early life stress may reproduce earlier than those raised in more stable conditions (Chisholm et al 2005, Moffitt et al 1992), although early menarche is also linked to general patterns of health and nutrition (Kaplowitz et al 2001).

Studying the impacts of maternal effects may further our understanding of the origins of human disease. Some adult-onset disorders are associated with the in-utero environment (Barker 1994) and poor maternal physical state can disrupt metabolic activity and predispose children to increased risk of strokes or heart disease in later life. Diabetes is linked to glucose intolerance and inadequate maternal nutrition may lead to offspring with fewer insulin producing pancreatic cells (Hales 1997). Similarly, low protein maternal diet in rats is associated with constrained processing of glucose in offspring livers (Burns 1997). Such later life diseases may represent a trade-off, coordinated by secretion of GCs, to preserve resources through nutritionally poor early life conditions. From an evolutionary perspective the costs of such trade-offs are mitigated as organisms often die prior to reaching old age. The thrifty phenotype hypothesis proposes that a malnourished foetus is maternally programmed for a

nutritionally poor environment and accordingly sets biochemical parameters to conserve energy and store fat (Hales & Barker 2001). This represents a form of plasticity as offspring traits are modified to match developmental conditions. If food is scarce these individuals may survive better than competitors whose metabolisms utilize, rather than store energy. However, problems may arise if thrifty offspring encounter plentiful post-natal conditions as the predisposition to store fat may heighten the risk of lifestyle diseases in later life (Hales & Barker 1992, 2001).

In wild systems individuals may suffer additional constraints if poor maternal physical state disrupts normal patterns of parental care. Offspring may respond to food shortages by reducing investment in growth or development to forego starvation risk. Low resource availability in early life has been linked with impaired growth (Emack et al 2008), immune function (Palmer 2011), skills acquisition (Berghanel et al 2015) and cognitive development (Coe & Lubach 2008). Poor maternal nutrition can constrain offspring as pre-natal investment is reduced in response to limited resources (Klaus et al 2013, Hinde & Milligan 2011) or by compromising the mother's ability to provide care. Development of the HPA axis is particularly vulnerable to naturally occurring variation in care effort (Drury et al 2016) as early life care provides an essential buffer against the physiological damage associated with stress (Gunnar et al 2015, Hostinar et al 2014). Both rodent (Liu et al 2000, Ladd et al 2004) and primate (Sanchez 2006, Sanchez et al 2015) studies link neglectful care with HPA hyperactivity in response to stressors. Subsequent improvement in care quality may reverse any negative effects (Champagne et al 2003, Burton et al 2007) and, in humans is associated with improved behaviour (Dozier et al 2008, Fisher et al 2007). When analysing these effects, it is important to consider differences in normal patterns of care between species, particularly regarding cooperative species where mothers are not always the principal providers of care.

In addition to maternal effects, studies have considered the potential of paternal (Braun & Champagne 2014) and grandparental (Rando 2012, Burton & Metcalfe 2014) effects to influence offspring development. Paternal stress levels may affect offspring phenotype via sperm (Rodgers et al 2013, Evans et al 2017) and depend on whether fathers provide offspring care or not (McGhee & Bell 2014). When male mice were trained to associate a stressor with a specific odour, their pups showed

increased behavioural sensitivity towards that smell even without previous exposure to it (Dias & Ressier 2014). This suggests that paternal experience of specific stressors may transfer to offspring via signalling. Future work could consider the nature of information transferred and how offspring respond when parental signals are in conflict (Chapman et al 2003) or agreement (Leimar & McNamara 2015).

1.5 Maternal stress during pregnancy

A key factor underlying phenotypic variation is exposure to maternally derived stress hormones (maternal stress) and the influence it has on offspring development. Reproduction is energetically costly (Williams 1966, Blount et al 2015) and often associated with high stress hormone concentrations (Bonier et al 2011, Love et al 2014). Chronically elevated GCs can increase maternal anxiety and constrain the ability to provide adequate care or provisioning (Cottrell et al 2012, Baker et al 2008). The offspring of chronically stressed mothers may themselves have higher GC concentrations (Feder et al 2009) and associated fitness costs such as reduced survival or reproductive success (Stearns 1992, Stier et al 2012). Exposure to chronic stress in early life can affect physiology, morphology and behaviour (Dalmaz et al 2015, Meaney et al 1994) and has been linked with aggression, anxiety and depressive behaviours in rodents and primates (Heim & Nemeroff 2001, McCormick & Green 2013). Maternal GC levels during gestation may be influenced by extrinsic factors such as population density (Dantzer 2013), predation risk (Coslovsky & Richner 2011), anthropogenic disturbance (Ellenberg et al 2007) or food availability. This can influence offspring phenotype (Hayward & Wingfield 2004, Meylan & Clobert 2004, 2005), disrupt development of stress physiology (Harris & Secki 2011, Storm & Lima 2010) and restrict size or growth (Sheriff & Love 2013), which is usually maladaptive. There is even potential for transgenerational impacts as maternal GC concentrations are known to influence development of the HPA axis in grandchildren (Bertram et al 2008, Matthews & Phillips 2012).

Maternal stress is generally seen as costly in both humans and animals due to potential impairment of the developing HPA axis and associated health problems (Hanson & Gluckman 2014, Moisiadis & Matthews 2014). The mechanisms by which

maternal stress influences offspring phenotype can work either pre or post-birth. GC levels during pregnancy can affect offspring either through prolonged exposure (Matthews 2002) or via acute stressors at key developmental stages (Kapoor & Matthews 2005, Kapoor 2009). Similarly, in early life, offspring may be affected by maternal GCs directly via lactation (Sullivan et al 2011) or through a stress induced reduction in the quality of parental care (Champagne & Meaney 2007). Such effects are described as 'maternal programming', which appears to assume that offspring have no recourse to any potential outcomes (Monaghan & Spencer 2014). In contrast, studies suggest that offspring can modulate the impact of low-level exposure through various mechanisms (Paitz & Bowden 2013, Paitz 2016) leading to potential "parent-offspring conflict" (Uller & Pen 2011, Muller et al 2007). However, it seems unlikely that these mechanisms can fully mitigate the damaging effects of chronic maternal stress (Lesage et al 2001, Lucassen 2009).

In order to generate individual phenotypic variation maternal effects should trigger changes on which selection can act. Laboratory studies (Weaver et al 2004, Meaney 2001) suggest that epigenetic mechanisms, such as DNA methylation, can pass non-genetic information from parents to offspring. Rats whose mothers were fed a low protein diet had different patterns of gene methylation from those fed a normal diet, changing the metabolic profile of the liver (Lillycrop et al 2005). Similarly, in rats, DNA methylation may provide the mechanism by which maternal GC levels impair the neuroendocrine stress response of offspring (Weaver et al 2004). Here the number of GC receptors in the hippocampus correlates with response to external stressors and is linked to the quality of early life care, which triggers changes to the promoter region of the GC receptor gene (Mueller & Bale 2008). Initially these sites are methylated in all pups, but those that receive intense maternal grooming lose their methylation, which is retained in offspring that receive less intensive care. Pups with methylated genes show higher GC concentrations and anxiety in later life, which impairs their ability to provide adequate care to their own offspring. Subsequent cross-fostering experiments suggested that individual differences were due to levels of care rather than maternal identity (Weaver et al 2004).

1.6 An adaptive perspective on maternal stress

Recent work questions the idea that maternal stress exclusively leads to maladaptive outcomes due to its role in mediating transgenerational phenotypic plasticity (Sheriff et al 2018). Offspring phenotypes such as low birth weight, slow growth and anxiety were previously regarded as an unavoidable consequence of elevated maternal GC. However, in specific conditions and contexts maternal stress may become adaptive (Del Giudice 2014, Belsky et al 2015). The environmental matching hypothesis (Love & Williams 2008, Monaghan 2008) suggests that where pre and post-natal conditions match, usually in challenging environments, phenotypes associated with maternal stress may become adaptive (Marshall & Uller 2007, Uller 2008, but see Uller et al 2013). For example, light birth weights are usually linked with low survival (Allen et al 2008) or competitive ability, but in nutritionally poor conditions could be associated with positive outcomes if individual energetic demands are lower. GCs have potential to modulate such effects as secretion regulates physiological processes, including metabolic rate, in response to external stressors (Haase 2016, Sapolsky 2000). In constrained environments developing organisms may adjust energy expenditure and metabolism in response to food intake (Pontzer 2015, 2017). Thus the adaptive value of phenotypic variation is often context dependent, particularly in wild studies where behavioural responses depend on social conditions (Giesing et al 2011, McGhee et al 2012). Organisms may use environmental cues to adaptively adjust their phenotype (Levins 1968, Jablonka et al 1995) with the population level variation being known as a “reaction norm” (Stearns & Koella 1986, Carter et al 2017). Phenotypic plasticity may allow for adaptation to ecological variation without genomic alteration (Bateson & Gluckmann 2011) via epigenetic changes to DNA expression (Godfrey et al 2007). This may occur when social or environmental conditions during pregnancy match those in early life (Bateson 2001). Plasticity is predicted to evolve in variable rather than stable conditions, which favour more fixed phenotypes (DeWitt et al 1998, Kuijper & Hoyle 2015). A developmental trajectory where phenotype is matched to environmental cues is termed a Predictive Adaptive Responses (PAR) (Gluckman & Hanson 2004) and in mammals, may be coordinated by maternally derived in-utero hormonal signalling (Lee & Zucker 1988).

PARs were first formulated in human studies looking at the association between childhood events and later life disease risk (Bateson et al 2004, Godfray 2010). They have since been considered across taxa and adaptively match offspring phenotype to future environments via maternal cues that reflect conditions during gestation. For example, if predation risk during pregnancy is high, mothers may produce young with enhanced defensive physiology such as camouflage (Rowell 1972), armour (Laforsch 2006), or behavioural adaptations (Mathis et al 2008). Offspring anxiety is linked to maternal stress and may be adaptive in risky environments (Mateo 2007) or arctic systems with cyclical predator-prey interactions (Sheriff 2010) or variable intra-specific competition. Variation in resource availability changes population densities and may represent an important agent of natural selection (Wade & Kalisz 1990, MacColl 2011). In red squirrels (*Tamiasciurus hudsonicus*), maternal GCs increase in response to population density, with territorial vocalisations acting as a cue. Offspring born in high densities grow faster to boost the probability of survival to reproductive maturity (Dantzer et al 2013). Accelerated early life growth is adaptive in high density conditions but is linked with shortened lifespan (Descamps et al 2008) and therefore is not favoured in years with lower density (McAdam et al 2003).

From an evolutionary perspective PARs prioritise survival only to reproductive age (Bateson et al 2014). Adapting to risky or nutritionally poor conditions may represent a life-history trade-off if fast growth or early maturation are linked to reduced longevity or fitness (Metcalfe & Monaghan 2001). Early maturation in response to adverse conditions has been observed in both rats (Slobada et al 2009) and humans (Belsky 2012). Wild studies reveal both positive (Chin et al 2009) and negative (Hayward & Wingfield 2004, Meylan & Clobert 2005) effects of maternal GC on growth. In aspic vipers (*Vipera* aspis), maternal water deprivation and elevated GC concentrations are associated with rapid offspring growth (Dupoue et al 2016). Likewise, in lizards, predation risk increased maternal stress and is linked to large offspring size, particularly tail length (Bestion et al 2014). House Wren (*Trogodytes aedon)* chicks injected with corticosterone prior to hatching were lighter initially but heavier at fledging, suggesting fast compensatory growth (Strange et al 2016). The treated chicks begged more intensely and mothers with experimentally elevated GC increased investment and produced fledglings with improved body condition (Bowers

et al 2016). These studies suggest that in certain contexts, maternal stress can act as a cue to prompt accelerated growth which may confer advantages to offspring in terms of predator avoidance, competitive ability or dispersal (Arendt 1997).

Hormonal in-utero signalling may represent a mechanism for prioritising growth to prepare for competitive early life environments where food supply is unpredictable. In rats, when maternal diet is restricted pups are smaller at birth (Jones & Friedman 1982) but gain weight faster and show increased appetite (Vickers 2000). Although these individuals are generally less active (Vickers 2003) they show an increased willingness to search for more reliable food sources (Miles 2009). Poor pre-natal nutrition may prepare offspring for a low resource environment by inducing phenotypes with lower muscle, a higher set point for satiety and a dietary preference for fat. In plentiful post-natal conditions this phenotype is associated with obesity and heightened disease risk. Human studies link maternal condition to traits such as body composition, metabolic control, reproductive maturation and behaviour (Godfrey 2006, Moritz & Cullen-McEwen 2006, Slobada et al 2007). This presents significant health consequences as diseases like hypertension and type 2 diabetes are more prevalent in individuals that are born small and subsequently follow western lifestyles with abundant nutrition (Patel et al 2006, Ebrahim et al 2010).

Adaptive plasticity may extend into the post-natal environment if early life conditions help organisms adapt to variation in later life (West-Eberhard 2003, Snell-Rood 2012). There is evidence both that good early life conditions can negate the phenotypic effects of maternally derived stress (Francis et al 1999) and that they cannot (Bian et al 2015, Sheriff et al 2015). Theoretical models suggest that the developmental period during which external cues mediate plasticity is determined by environmental variation and species life-history traits (Panchanathan & Frankenhuis 2016). For PARs, a potential field of interest is how phenotypic response is modified by the potential for conflicting information being received from the mother and the early life environment (Kuijper & Hoyle 2015). Another question is to what extent maternally derived stress can allow species to adapt to human induced ecological change. Rapidly acting or novel stressors can set evolutionary traps (Schlaepfer et al 2002), especially where they compromise maternal ability to recognise novel events as dangerous (Sih et al 2010, Trimmer et al 2017). Significant interest has focused

on climatic change and the potential for environmental stressors to become more frequent or variable. In chinook salmon (*Oncorhynchus* tshawytscha), exposure to pre-natal stress appears to improve offspring ability to respond to drought conditions in early life (Capelle 2016). Some human activities may expose organisms to novel predators and maternal predation risk is linked to reduced offspring size and weight (Sheriff et al 2009, Zanette et al 2011). Studies suggest that species may be able to adjust anti-predator responses to include closely related novel predators (Griffin et al 2001, Ferrari et al 2007) and it is possible that plasticity in predation response is linked to maternal stress.

1.7 Maternal effects in cooperatively breeding species.

Cooperatively breeding species (Clutton-Brock 2009, Hatchwell 2009), where adults help raise offspring other than their own, represent good systems for testing maternal effects (Russell & Lummaa 2009). Groups usually consist of a dominant breeding pair, who monopolise reproduction, and younger subordinate individuals that help care for offspring (Griffin et al 2003, Nelson-Flower at al 2011, although some species are more egalitarian, Koenig et al 1995, Packer et al 2001). Helpers are often previous offspring that forego dispersal, delaying their own breeding opportunities (Bourke 1997), although they may also be unrelated immigrants. Within social groups there is marked phenotypic variation, even between individuals of similar sex, age or dominance status which is partly explained by differences in maternal condition and the rearing environment. Therefore, patterns of maternal investment will vary depending on offspring sex, available care provision and levels of intra-group competition. Social rank can strongly influence GC concentrations (Creel 2001, Creel et al 2013) with subordinate individuals often showing elevated stress due to receiving aggression from dominants (Abbott et al 2003, Hacklander et al 2003). Where elevated GC reduces reproductive success (Sanderson et al 2015) such aggression may be associated with fitness costs and represent a form of stress-related reproductive suppression (Young et al 2006).

Cooperatively breeding mammals may be good candidates for evolving PARs if mothers can predict the competitiveness of the early life environment. Maternal cues

may be drawn from hormonal concentrations, which vary depending on social group composition and ecological conditions during pregnancy, and investment may be adjusted accordingly. Within groups, reproductive opportunities vary depending on immigration, extra group mating or co-breeding (Cockburn 2004, Russell 2004). In addition, sex-biased dispersal, care or immigration may influence competition and relative maternal investment. When daughters remain in the natal group as helpers breeding is costly as they have fewer remaining resources for provision of social care and their young may compete with either their mother or future siblings. This may drive aggressive reproductive competition (Clutton-Brock & Huchard 2013) which increases maternal stress and hormonal signalling may prime development towards phenotypes more favoured in competitive environments. Mothers, therefore may face conflict between conserving resources for the future, and maximising current offspring fitness. Therefore, patterns of maternal investment may drive phenotypic variation. Most maternal strategies will represent a balance of these two factors depending on context, often determined by the scope of future reproductive opportunities, care availability and nutritional state during pregnancy.

In social species, phenotypic variation is partly due to the quality of care provided by helpers (Savage et al 2015). Mothers may tactically adjust investment to match conditions (Cunningham & Russell 2000) by increasing pre-birth investment to compensate for poor rearing conditions with fewer helpers (Bolund et al 2009) or reducing resource allocation to transfer costs onto other carers (Russell et al 2007). In cooperative breeders, parents may respond to the presence of helpers by either reducing provisioning (load-lightening, see Crick 1992) or maintaining provisioning effort (additive effects) (Hatchwell 1999). In general, the presence of helpers is expected to affect the scale of maternal effects (Beery & Kaufer 2015) but this may also depend on which of the above strategies is favoured. In systems where mothers are relieved of the burden of care pre-natal conditions may be crucial in mediating maternal investment decisions. Studies of bi-parental systems suggest that the potential costs of maternal stress may be overcome by the partner (Hinde 2006, Johnstone & Hinde 2006). This may extend to cooperatively breeders if offspring are buffered from pre-natal stress in systems where helpers relieve mothers of the burden of exclusive care. In communally breeding degus, high maternal GCs are

associated with impaired offspring stress physiology, but the negative effects are buffered by the presence of unstressed carers in the group (Bauer et al 2015). Similarly, studies of cooperatively breeding birds suggest that the presence of helpers can compensate for reduced maternal investment in eggs and provisioning (Russell et al 2008, Brouwer et al 2014).

Many lab studies associate poor maternal care during crucial developmental periods with impaired stress physiology. However, it remains unclear whether lower standards of cooperative care have similar effects where mothers are not the primary caregivers. In cooperatively breeding marmosets, exposure to early life stressors increased anxiety, altered the HPA stress response and impaired social interactions (Solomon & French 1996). Cues from various classes of caregiver (fathers, siblings and mothers) were all found to influence HPA response during both development and adulthood (Birnie et al 2013). Offspring that received higher rates of rejection from family members showed higher cortisol in response to social separation. Stress physiology is sensitive to social cues and family relationships can either buffer HPA response (Rukstalis & French 2005) or intensify HPA activation during intragroup conflict (Smith & French 1997). The role of GCs in maintaining energetic stability suggests that they may mediate care provision by helpers, as well as parents. GC concentrations have been both positively (Carlson et al 2006) and negatively (Sanderson et al 2014) associated with cooperative care provision and some studies have reported that such effects may be dependent on helper sex (Reynauld & Schradin 2015, Dantzer et al 2017).

1.8 Thesis aims

The aim of this thesis is to investigate the ultimate and proximate mechanisms underlying individual phenotypic variation in a cooperatively breeding mammal. To achieve this, I use the long-term dataset collected by the banded mongoose research project to analysis maternal investment patterns and phenotypic differences that may not be visible in short-term studies. By using a mixed approach, I hope to provide novel insights into the association between individual behavior and physiology within a social context, and enrich our understanding of the evolutionary

processes underpinning sociality and cooperatively breeding systems.
Chapter two introduces the study population and provides a general outline of the methods used in data collection and analysis.
Chapter three tests how maternal effects, in particular maternal rank, and levels of cooperative care influence GC concentrations in banded mongoose pups. In addition, I ask whether elevated GC levels in early life increase the probability of an individual surviving to either nutritional independence at three months of age or maturity at one year.
Chapter four uses a larger dataset to consider how early life conditions and maternal effects influence adult life-history traits. Specifically, I test whether maternal age has sex-specific effects on survival, longevity and reproductive success and whether these patterns reveal condition-dependent maternal resource allocation strategies and influence offspring sex ratios.
Chapter five synthesises my findings and attempts to draw on the wider implications and make suggestions for future study.

Chapter 2: General Methods

2.1 Summary

This section provides a summary of all data collection and analysis methods used in this thesis. I start by introducing the field site, banded mongoose system and data collection, including the behavioural observations that were used to estimate helper care effort. I then briefly summarise the genetic techniques that were used to establish maternal identity and the methods of faecal sample collection and hormonal extraction. All of the above data is collected to maintain the long-term dataset and was not conducted by me. I conclude this section by describing the statistical analyse that I used throughout this thesis.

2.2 Study site

This study was carried out using a habituated population of banded mongooses located on the Mweya Peninsula in Queen Elizabeth National Park, Uganda (figure 1) which comprises a mix of open grassland and thick scrub. This population has been studied since the 1970's but the current research project was started in 1995 by Michael Cant and Tim Clutton-Brock. This study uses a combination of faecal samples from five packs collected by Jenny Sanderson for her PhD thesis and analyses from the long-term dataset. The climate is equatorial with little seasonal fluctuation in temperature or day length. Annual rainfall is typically 800-900mm per year and there are two dry periods in January-February and June-July (figure 2). Meteorological data was collected at a weather station on the field site.

2.3 Study System

Banded mongooses (*Mungos mungo)* are cooperatively breeding mammals that live in mixed-sex groups of between approximately 8 and 40 individuals (Cant et al 2016). Adults reach sexual maturity at around one year of age (Cant et al 2013) and all group members are capable of breeding. Reproductive opportunities are skewed toward older animals (Nichols et al, 2010, 2012) and females usually start breeding at a younger age than males, who tend to live longer (Vitikainen et al 2016). Females

have higher mortality rates throughout life, particularly in periods of low rainfall (Marshall et al 2016), leading to male-biased sex ratios within groups.

a. b. c.

Figure1. Location of the Banded Mongoose Research Project study site. Field site location in (a) Africa and (b) Uganda is indicated with red stars. (c) Map of the Mweya Peninsula. The field site is shown with a red dotted line, the ridge separating the upper and Lower Peninsula is shown with blue dots (from Sanderson 2012).

Unlike in cooperative species where dominant pairs monopolise reproduction (including closely related meerkats (*Suricata suricata*) (O'Rhian et al 2000) and dwarf mongooses (*Helogale parvula)* (Keane et al 1994)) banded mongooses are plural breeders. Packs have up to four litters per year which are highly synchronized (within but not between groups), with most females in the group reproducing in each breeding attempt and giving birth to a large communal litter on the same day (Cant 2000, Hodge 2011). The evolution of synchronous breeding leads to large cohorts of similarly aged individuals and intense intragroup competition for reproductive opportunities in both sexes. Mothers may kill the pups of rival breeders where birth is asynchronous (Cant et al 2014) and there is no evidence that parents can discriminate their offspring from others in the communal litter (Vitikainen et al 2017). This constrains the effectiveness of infanticide as a mechanism of reproductive control (Clutton-Brock et al 2001). Instead, in response to reproductive competition, older females violently evict younger females from the group (Cant et al 2010, Thompson et al 2016), implying an age-based dominance hierarchy. The threat of expulsion may represent a covert form of suppression as younger breeding females show higher stress hormone levels during pregnancy and lower reproductive success (Sanderson et al 2015a). There is evidence of predictive adaptive response (PAR) in banded mongooses (Inzani et al 2016) as maternal investment, measured

by foetus size, increased when pups were predicted to face a competitive early life environment. Larger size is expected to confer a competitive advantage and may increase the amount of escorting care received (Vitikainen et al 2017) and thus maternal investment appears to vary according to the social conditions experienced during pregnancy. Where reproductive suppression is largely ineffective and pups are born in competitive environments investment may be adjusted to maximise offspring fitness, rather than conserving resources for future litters. Thus, maternal decisions could be dependent on pre-natal cues as mothers are not burdened by early life care and can effectively de-couple pre and post-birth investment.

(a) (b)

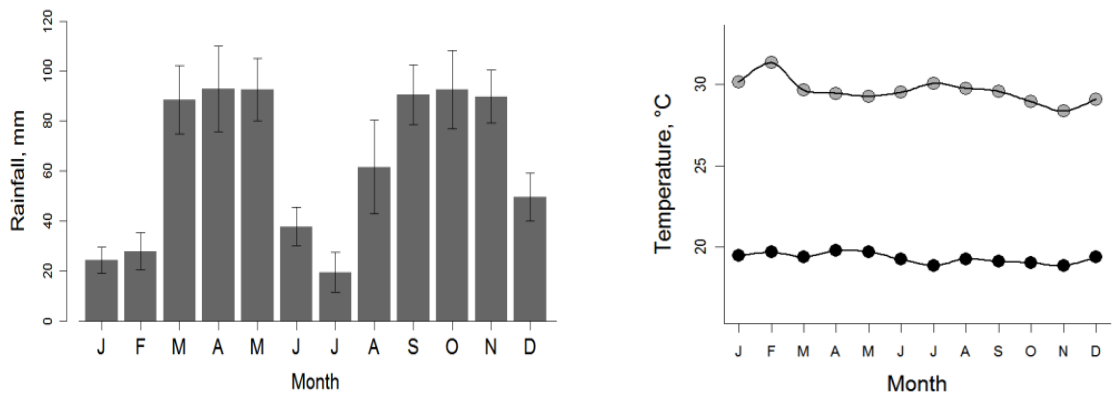

Figure 2. Annual meteorological data for Mweya, Queen Elizabeth National Park. (a) monthly rainfall, means and standard error. (b) Daily maximum and minimum temperature, means. Data from 1999 to 2012 provided by UWA (from Sanderson 2012).

Gestation lasts for around 60 days and litters are cared for by most adult group members. Banded mongooses exhibit two distinct forms of cooperative care, “babysitting” (figure 3a) and “escorting” (figure 3c). Litters remain underground for their first month or so and are cared for by babysitters, who remain at the burrow during group foraging trips to guard against predators or rival packs. Following emergence pups move with the group and compete for escorts, with whom they form a ‘one on one’ caring relationship (Hodge et al 2009). Although pups seem to initiate this bond it is maintained by the adult who will respond to both experimental presentation of the pup (Gilchrist et al 2008) and playbacks of their specific distress calls (Muller & Manser 2008). The escort provides food to the pup until nutritional

independence at around three months (Gilchrist, 2004). There can be significant variation in the care received by each pup (Hodge 2007) and escorting provides important benefits, including higher provisioning, faster growth, lower mortality and earlier reproductive maturity in females (Hodge 2005). Most group members over the age of 6 months help with care, although subordinate males contribute more to both babysitting (Cant 2003) and escorting (Gilchrist & Russell 2007). This is probably due to sex-specific care costs (Clutton-Brock et al 2000) as relatedness does not explain the variation in helping behaviour (Nichols et al 2012). Both forms of care are associated with weight loss and females suffer relatively higher costs than males as maternal weight is associated with breeding success (Hodge et al 2009). Reproductive skew is more pronounced amongst males who have more opportunity to regain weight between breeding attempts. Male pups generally receive more care than female pups partly because carers tend to assort with offspring by sex (Vitikainen et al 2017). This sex-specific care assortment may impact individual development and help explain the observed male-biased sex ratios. As such, pre-birth social group composition may inform mothers of the likelihood of pups receiving care and allow them to adjust investment accordingly.

Figure 3; Some examples of banded mongoose social behaviour, a) an adult babysitting, b) competition between pups during the escorting period, c) an adult escorting, d) a pack defensive response.

2.4 Data Collection

Packs were visited at least three times a week, the regularity of visits allowed group composition to be constantly monitored. During the later stages of pregnancy groups were visited more frequently to establish exact birthdates for litters. The mongooses were habituated to close observation and trained to step onto portable electronic scales to obtain weight measurements. To identify specific individuals all mongooses were captured within three weeks of emergence from the natal burrow and then every 3 – 6 months until leaving the study through death or dispersal. Trapping allowed general health checks and morphometric measurements, and was used to assess pregnancy in adult females. Captures were conducted using baited tomahawk traps (67 x 23 x 23 cm; Tomahawk Live Trap Co., Tomahawk, WI, USA) and following trapping, individuals were anaesthetised using isoflurane (for full protocols see Jordan et al 2010, 2011). The research was conducted under licence from the Uganda National Council for Science and Technology, and all procedures were approved by the Uganda Wildlife Authority. During an individual's first capture a 2mm skin sample was taken for genetic analysis (Nichols et al. 2010) and the animal was microchipped for identification purposes. To allow individual identification during behavioural observations pups were marked with a unique combination of patches using hair dye and adults were marked with a unique shave on their back. For radio-tracking purposes individuals in each pack were fitted with a radio collar weighing 26-30g (Sirtrack Ltd, Havelock North, New Zealand).

2.5 Quantifying escort care

The amount of care received by pups from escorts was calculated following the methods of Vitikainen et al (2017). During the escorting period, groups were visited on average 12 times for a minimum of 20 minutes (the duration of one focal observation). Adults were scored as escorts if associated closely with the focal pup and were seen within 50cm of the same pup for more than 50% of the observation period (Vitikainen et al 2017). Escorting effort received per pup was calculated as the proportion of days that a pup was recorded with an escort out of the total number of days where escorting was recorded by any group member (7-21 observation days per breeding attempt). This gave a single escorting score (as a proportion) for each

pup in a litter which was used to represent care received. Previous studies suggest that this association score is a reliable summary of quantitative nearest-neighbour data (Gilchrist & Russell 2007).

2.6 Genetic analysis

Due to the birth synchrony in this species, maternity cannot be determined via observations and therefore genetic analysis was used to assign parentage (Nichols et al 2010, Sanderson et al 2015b). To perform DNA extraction, the skin samples collected during capture (usually tail-tips) were incubated on a rotating wheel in 330 ml of lysis solution (10 mM Tris HCl (pH 8.0), 1 mM EDTA, 1% SDS, 50 mg/ ml of proteinaseK), first at 55˚C for 2 hours and then at 37˚C overnight. DNA was purified from the digested tissue sample using an equal phenol:chloroform purification followed by an ethanol and ammonium acetate DNA precipitation. Additional DNA extraction was done using DNA extraction kits (Qiagen Tissue and Blood Kit). Samples were genotyped at up to 43 microsatellite loci, isolated from a variety of carnivore species, including the banded mongoose.

Pre 2010 Genotyping

Details of genotyping procedures followed prior to 2010 are given in Nichols et al (2010). Briefly, genotyping was conducted using a panel of 14 microsatellite loci, isolated from a variety of carnivore species. PCRs were carried out in 11 ml reaction volumes, using approximately 20 ng of genomic DNA, 0.2 mM each of forward and reverse primers, 10 mM Tris, pH8, 50 mM KCl, 0.01% Tween 20, 0.01% gelatine, 0.01% nonidet P40, 0.025 units of Taq polymerase, 1.5-2.52 mM magnesium chloride and 0.01 uCi (a33P)-dCTP. The following PCR conditions were used with each primer pair: an initial denaturing step of 94˚C for 4 minutes, followed by 35 cycles of 94˚C denaturization for 45 seconds, 50-60˚C annealing for 30 seconds and 72˚C extension for 30 seconds, followed by a final elongation of 5 minutes at 72˚C. PCR products were resolved by electrophoresis on standard 6% polyacrylamide gels and were visualized using a phosphorimager. Bands were scored manually.

Post 2010 Genotyping

After 2010, genotyping was conducted (Sanderson et al 2015b) using multiplex PCRs (Qiagen Multiplex PCR Kit, UK) with fluorescent-labelled forward primers and was visualized through fragment size analysis on an ABI 3730 DNA Analyzer. PCR conditions followed the Qiagen Multiplex PCR Kit recommendations (but were conducted in 12 µL reactions), with an annealing temperature of 57°C. Full details of the 43 microsatellites used in this study along- side primer sequences, multiplex sets and PCR conditions are given in Sanderson et al 2015b (Appendix S1.1).

2.7 Parentage Analysis

The extracted DNA samples were used to create a pedigree to infer parentage within the study population. The final pedigree used Masterbayes 2.51 (Hadfield et al. 2006) and Colony 2.0.5.7 (Jones & Wang 2010) as well as field observations and individual genotypes. In brief, Masterbayes was used to assign parents to offspring (i.e. individuals that were observed being born into the population). All females (aged >6 months) present in the natal group at birth were included as candidate mothers, and all males (aged >6 months) present in the study population at conception were included as candidate fathers to allow for extra-group mating (Nichols et al 2015). The following phenotypic predictors of parentage were also included: whether or not a female was recorded as giving birth, if a male was in the offspring's natal group, and the age and quadratic age of both males and females. In addition, the number of unsampled candidate mothers and fathers was estimated in the parentage assignment model. Genotyping error rates were calculated manually from samples genotyped in duplicate following Hoffman & Amos (2005). Allele frequencies were calculated in Cervus 3.0.7 (Kalinowski et al. 2007) using the full genotype data set. Genotyping error rates and allele frequencies were both provided in the model specification. The Markov chain Monte Carlo estimation chain was run for 1 500 000 iterations with a thinning interval of 500 and a burn-in of 500 000. No further prior distributions were specified, and default improper priors were used. Successive samples from the posterior distribution had low autocorrelation ($r < 0.01$).

Secondly, sibships were constructed in Colony by partitioning all genotyped individuals (including offspring, founders and immigrants) into full and half-sibship

groups with or without parentage assignments, using a maximum-likelihood method. The same candidate parent criteria were used as above to generate candidate father list, candidate mother list, paternal exclusion list and maternal exclusion list as input into Colony. No maternal or paternal sibships were excluded. A weak sibship prior of 1.5 for both maternal and paternal average sibship size was included to limit false-positive sibship assignments, and the probabilities that the true mother and father were in the candidate lists were both set as 0.8 (Sanderson et al 2015b). Parentage assignment was accepted with ≥0.8 probability in both Masterbayes and Colony. Masterbayes parentage assignments were accepted first (note that no ungenotyped individuals were confidently assigned parentage), and Colony parentage assignments were then added where Masterbayes had failed to assign parentage.

Using the same panel of genetic markers for parentage assignment and for calculating levels of relatedness has been shown to bias paternity assignments towards unrelated fathers in some cases (Wang 2010). We minimized the probability of encountering such biases by using a large panel of markers for parentage analysis (43 microsatellites) which allowed for high confidence of parentage assignment in almost all cases (between April 2003 and September 2013 91% and 88% were assigned paternity at ≥0.8 and ≥0.95, respectively). Furthermore, where possible, we verified our genetic data using behavioural observations of mate-guarding patterns, which are not subject to such biases.

2.8 Faecal sample collection and extraction

All faecal samples were taken between May 2010 and December 2013 and then transported to the UK for analysis. Where possible, faecal samples were collected during the morning latrine session soon after emergence from the burrow. Faecal samples were collected by hand into small plastic bags (4” x 2”), labelled with the mongoose identity, date and time, and placed on ice in a Thermos flask. Samples were then transferred to a -20 freezer within 5 hours of collection. Collection time and time to freezer were non-significant predictors of faecal glucocorticoid metabolite (fGC) concentrate ions (Sanderson 2012). Over-marking of faeces is common in banded mongooses (Muller & Manser 2008) and samples were only collected where certain that no over-marking occurred. In addition, to avoid interference with group scent marking behaviour only half of each sample was collected.

Material transfer permits were obtained from the Uganda Wildlife Authority (UWA) and the Uganda Council for Science and Technology (UNCST), with UK import permits being obtained from DEFRA. Frozen faecal samples were transported back to the UK on wet ice in a cool box. Samples were transferred to Entebbe airport by road, flown back to the UK as checked-in luggage, and transported to the University of Exeter in Cornwall by train. In total, this journey lasted a maximum of 36 hours and the samples were still chilled when they arrived at their final destination.

Hormone extraction was carried out either at Chester Zoo Endocrinology Lab (CZEL) or at the University of Exeter (UofE). Samples extracted at the UofE were then transferred to CZEL on ice for assay. We extracted a subset of samples (n=20) at both CZEL and UofE to ensure the location of extraction did not affect the hormone results. The glucocorticoid assays from samples extracted in both CZEL and UofE were highly correlated (Pearson's correlation: $T = 10.66$, $p < 0.001$) and there was no significant effect of extraction location on fGC measures (GLMM: $\chi 2 = 2.72$, $p=0.26$), therefore results from both locations were pooled in the analyses.

Hormone extraction was done using a wet-weight shaking process adapted from Walker et al. (2002) following thawing and manual homogenisation. In brief, 0.5 g of faecal material was combined with 90% methanol, shaken overnight at room temperature and centrifuged for 20 minutes. The methanol fraction was decanted and evaporated to dryness. Faecal extracts were re-suspended in 1ml methanol and stored at -20°C until analysis.

2.9 Hormonal Assay; Methods and Validation

Faecal glucocorticoid metabolite (fGC) concentrations were analysed using modified enzyme immunoassays (EIA; Young et al, 2004, adapted from Munro & Stabenfeldt, 1984). Each EIA utilised an antibody (polyclonal corticosterone CJM006 supplied by CJ Munro, University of California, Davis, CA), horseradish peroxidase conjugated label (corticosterone; prepared according to Munro & Stabenfeldt, 1984) and standards (corticosterone; Sigma-Aldrich, UK).

The modified assay procedures for the corticosterone EIA were as follows:

- antiserum was diluted at 1:15,000 in coating buffer (0.05 M $NaHCO_3$, pH 9.6), loaded 50µl per well on a 96-well Nunc-Immuno Maxisorp microtiter plate (Thermo-Fisher Scientific), and covered with a plate sealer and left overnight at 4°C.
- Plates were washed five times (0.15 M NaCl, 0.05% Tween 20).
- Standards (corticosterone, 3.9 – 1000 pg per well) or samples were diluted 1:20 in EIA buffer (0.1 M $NaPO_4$, 0.149 M NaCl, 0.1% bovine serum albumin, pH 7.0) and loaded 50µl per well.
- The horseradish peroxidase conjugate was diluted in EIA buffer to 1:70,000 and added 50µl per well. Following incubation in the dark for 2 hours at RT, plates were washed 5 times and incubated with 100µl per well of RT substrate (0.4 mM 2,2'-azino-di-(3-ethylbenzthiazoline sulfonic acid) diammonium salt, 1.6 mM H_2O_2, 0.05 M citrate, pH 4.0) and left to develop at RT in the dark and measured at 405 nm at optical density 0.8 to 1.0.

The corticosterone antiserum CJM006 was found to cross-react with Corticosterone 100%, Deoxycorticosterone 14.25%, Progesterone 2.65%, Tetrahydrocorticosterone 0.90%, Testosterone 0.64%, Cortisol 0.23%, Prednisolone 0.07%, 11-desoxycortisol 0.03%, Prednisone < 0.01%, Cortisone < 0.01% and Estradiol < 0.01% (Watson et al, 2013). The intra and inter-assay coefficients of variation for the corticosterone assay were 7.52 and 6.33% (C1 and C2) and 8.66 and 10.47% (C1 and C2) respectively.

The corticosterone assays were validated for measuring corticosterone metabolites in female and male banded mongoose faeces by parallelism, accuracy check, and ACTH challenge. Serial dilutions of male faecal extract yielded a displacement curve parallel to the standard curve (corticosterone: sample % binding = 11.713 + 0.651 (standard % binding), R^2 178 = 0.9957, $F_{1,7} = 1631.26$, $p < 0.001$). There was no evidence of matrix interference in male corticosterone assays, as addition of diluted faecal extract to standards did not alter the amount expected (corticosterone: Observed = 31.074 + 1.522 (Expected), $R^2 = 0.997$, $F_{1,7} = 2342$, $p = 0.06$). Serial dilutions of female faecal extract yielded a displacement curve parallel to the standard curve (corticosterone: sample % binding = 11.713 + 0.651 (standard % binding), R^2 178 = 0.969, $F_{1,7} = 218.64$, $p = 0.683$). There was no evidence of

matrix interference in assays of female corticosterone, as addition of diluted faecal extract to standards did not alter the amount expected (corticosterone: Observed = 31.074 + 1.522 (Expected), R2 = 0.9954, F1,7 = 1512.316, p = 0.4329). The physiological validity of using this EIA assay technique to measure fGC concentrations in banded mongoose samples was established by demonstrating a cause-and-effect relationship between exogenous administration of corticotrophin (ACTH; one intramuscular injection of 13µl of 1mg / ml synthetic ACTH [Tetracosactide; Synacthen], n = 3 males and 3 females) and the subsequent excretion of fGC metabolites in the faeces. fGC levels were higher in the two days following injection than the two days prior to injection (Mann-Whitney; n = 26 and 25, $p < 0.001$) (one female failed to show an increase in fGC concentration, though this may be due to missed samples). Peak fGC elevation occurred 6.67 ± 0.21 hours (mean ± SE, n = 5) after ACTH administration.

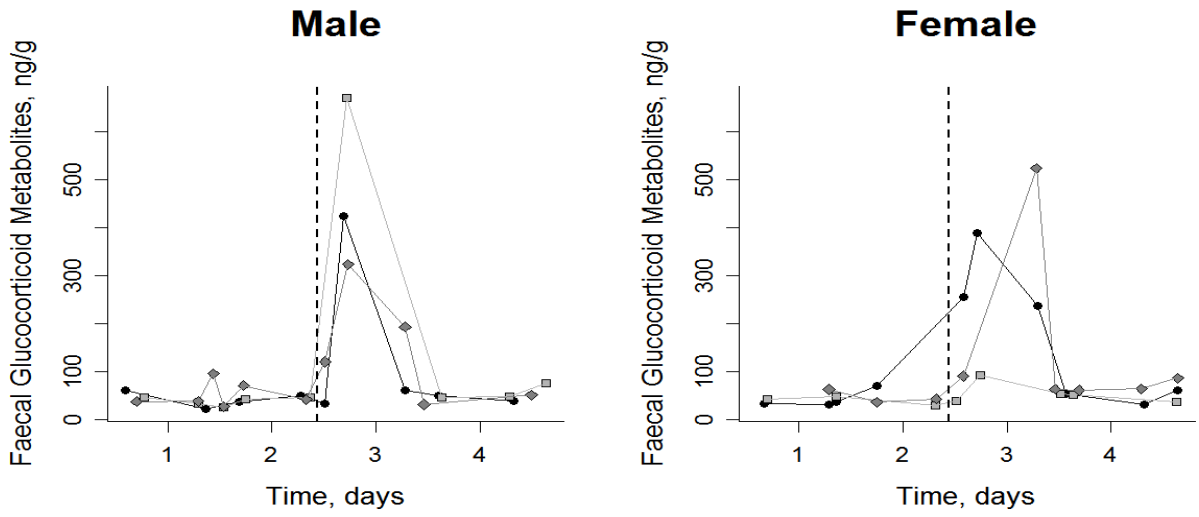

Figure 4. Faecal glucocorticoid metabolite (fGC) concentrations from male and female banded mongooses before and after administration of synthetic ACTH (Tetracosactide; Synacthen). Each solid line represents a single individual. The vertical dashed line represents time of ACTH administration (from Sanderson 2012).

2.10 Statistical Analysis and model selection

Statistical analyses were performed in R (R Core Team 2016). Linear and general linear mixed models (LMM's and GLMM's) were fitted using the lme4 package (Bates et al. 2015). In all analyses, we assessed the significance of each fixed effect

by comparing the likelihood ratio of the maximal model to that of the model without the fixed effect (Bates et al 2015). We present parameter estimates and SEs from maximal models, rather than removing nonsignificant fixed effects due to problems associated with stepwise model reduction (Forstmeier & Schielzeth 2011). We did, however, remove non-significant interactions to allow the significance of the main effects to be tested (Enquist 2005). Multivariate statistics were used to control for repeated sampling of individuals, litters and social groups within the population. Where possible parametric tests were used and at times logarithmic transformations were needed to achieve normality of error. All models were checked using residuals and were found to meet the assumptions for parametric tests.

Chapter 3: Factors influencing early life stress in banded mongoose pups.

Abstract

Conditions experienced during development can profoundly influence offspring phenotype. Maternal effects may be mediated by prenatal hormone exposure and can shape individual variation via non-genetic inheritance of phenotypic traits. Mammalian laboratory studies suggest that maternal stress can constrain the quality of parental care and trigger lasting physiological changes. There is less information on how maternal effects influence development in wild mammals, particularly in cooperative breeders where offspring receive a large fraction of investment from non-parental carers. I investigated whether the amount of cooperative care received influenced the stress levels of banded mongoose pups, and whether highly stressed phenotypes were associated with reduced pup survival. I found no evidence that levels of cooperative care affected early life glucocorticoid (GC) concentrations. However, I did find a sex-specific within-individual effect of maternal age on pup GC. The GC levels of daughters were found to decline over a mother's reproductive lifespan, whereas that of sons increased. Elevated early life stress was also associated with lower immediate, but not delayed, survival rates. Further investigation is needed to establish the social, behavioural or demographic consequences of early life stress in this species and whether early life GC levels can have sex-specific effects on offspring fitness. The results highlight how maternal age and rank in cooperatively breeding groups can have major effects on the stress physiology and survival of developing offspring, and that these effects may persist long after maternal investment has ended.

Introduction

Cooperatively breeding species, where adults help raise offspring other than their own, have proved to be excellent models for understanding the evolution of cooperation and conflict in social animals (Koenig et al 2016). However, these studies have also revealed unexplained behavioural and physiological variation, even among individuals of similar age, sex, relatedness and dominance status (Solomon & French 1997). A potential explanation for such differences is maternal effects, by which a mother's condition or environment can influence offspring phenotype (Bernado 1996, Wolf et al 1998). The link between maternal and offspring phenotype may be mediated by prenatal hormone exposure, which can influence physiology and behaviour through non-genetic inheritance of phenotypic traits (Sapolsky 2005, Dioniak et al 2006). Maternal effects are predicted to exert a strong influence in cooperative species (Russell & Lummaa 2009), where the quality of the rearing environment depends on group social structure and the quality of care provided by both parents and non-parental helpers.

The maternal environment can profoundly influence offspring development and phenotype (Mousseau & Fox 1998, Mousseau 2009). These effects can be positive or negative depending on ecological and social conditions (Groothius et al 2005, Monaghan 2008). Favourable early life conditions, known as 'silver spoon' effects (Grafen 1988), generally have a positive influence on offspring fitness (Wong & Kolliker 2014). By contrast mothers in poor condition have fewer available resources to invest in their offspring, who may suffer developmental constraints (Gluckman & Hanson 2014). In addition, increased physiological stress may constrain maternal ability to provide adequate offspring care or nutrition (Liu 1997). Mammalian lab studies suggest that poor maternal care can alter the expression of genes regulating behavioural and hormonal stress responses with effects that persist into adulthood (Meaney 2001, Weaver et al 2004). Elevated stress constrains maternal ability to provide care and triggers changes in DNA methylation patterns at the promoter region of the glucocorticoid (GC) receptor gene (Mueller & Bale 2008). Pups that receive less intensive licking and grooming in early life are associated with maladaptive phenotypes and an impaired ability to downregulate GC's and cope with external stressors. These epigenetic changes may trigger intergenerational effects if

mothers that receive less care during early life then show higher stress during their own pregnancies and produce offspring with impaired stress physiology (Champagne & Meaney 2007).

Much research into the vulnerability of biological systems to stress has focused on the HPA (hypothalamic-pituitary-adrenal) axis. This is a major neuroendocrine system that helps organisms maintain internal stability and regulate physiological processes. The HPA axis mediates organism response to changing conditions and prompts endocrine glands to secrete steroid hormones, including glucocorticoids such as cortisol and corticosterone, that control physiological stress response. GCs facilitate energy release in response to demanding or dangerous activities and have been proposed as a major mechanism mediating maternal effects in mammals (Dantzer et al 2013). In addition to their role as modulators of phenotypic and behavioural variation, GCs mediate day-to-day homeostasis and play a key role in the body's response to external stressors (Sapolsky 2000). Although the secretion of stress hormones is adaptive in competitive or risky environments, over-secretion of GCs may inflict physiological costs in the long term. Chronic stress is associated with severe physiological problems including impaired digestion, growth, immunity and reproduction (Wingfield 1998). Early life and prenatal conditions can modify baseline hormone levels and HPA reactivity with effects that persist into adulthood. Studies have reported correlation between elevated GC concentrations and poor body condition (Romero & Wikelski 2001, Love et al 2005) but there is little consistent evidence that high baseline GC negatively affects fitness (but see Bonier et al 2009).

In contrast to the deleterious effects of chronic stress, in certain circumstances elevated stress hormones may be associated with fitness benefits (Escribano-Avila et al 2013), especially in energy demanding life-stages such as growth (Chin 2009) or reproduction (Bonier 2011). When offspring are born in adverse conditions, maternal stress may become adaptive in the short-term if acting to conservatively constrain development (Hales & Barker 1992). In the longer-term, maternal stress has been posited to function as a form of predictive adaptive response (PAR) (Gluckmann et al 2005), allowing mothers and offspring to improve the fit between offspring phenotype and the anticipated post-natal environment. According to the PAR hypothesis, mothers take predictive cues from the environment during

pregnancy and adjust developmental trajectories according to the conditions that offspring are expected to encounter in early life. Maternal GC represents a good candidate mechanism for mediating such cues (Bateson et al 2015), and phenotypic plasticity is predicted to occur via epigenetic mechanisms such as methylation rather than genomic alterations to the developing organism (Tammen et al 2013). For example, in periods of high density red squirrel (*Tamiasciurus hudsonicus*) mothers show elevated GC levels and produce faster growing offspring that gain an advantage over slower growing competitors (Dantzer et al 2013). Thus maternal stress can be adaptive where it promotes rapid in-utero offspring growth in response to reliable signals that offspring will face a highly competitive early life environment.

A powerful compliment to laboratory studies is observation of wild animals in their natural environment. True developmental trade-offs may only be realised when mothers and offspring are exposed to natural predators, pathogens and environmentally mediated rates of resource availability and competition. Patterns in wild populations can be used to test whether the observed impacts of maternal effects are as predicted by adaptive hypotheses. Studies of cooperatively breeding mammals are particularly useful (Lummaa & Russell 2009) because these species are prime candidates for the evolution of PARs (Inzani et al 2016). Maternal effects are important because competition between breeding females can be intense as their offspring will compete locally for helpers and resources. The degree of early life competition faced by offspring may therefore be predictable from group structure during pregnancy and mothers may use social cues to adaptively prime unborn offspring for their postnatal environment (Dloniak et al 2006).

Here I investigate the influence of social and ecological conditions on the hormonal mechanisms regulating maternal effects in wild banded mongooses (*Mungos mungo*). Banded mongooses are cooperatively breeding mammals that live in mixed-sex groups of between 8 and 40 individuals. Adults reach sexual maturity at around one year of age and most group members are capable of breeding (Cant et al 2013). Reproductive opportunities, however are skewed towards older individuals (Nichols et al 2010, 2012) and females reproduce at a younger age than males, although males usually live longer (Vitikainen et al 2016). Groups may breed up to four times per year and reproduction is highly synchronized within, but not between groups,

with multiple females giving birth to a large communal litter, usually on the same day (Cant 2000, Hodge 2011). Pups remain underground for their first month or so, after which they join the group on foraging trips away from the den. During this period pups compete intensely for particular adult helpers, with which they form a one on one "escorting" relationship (Gilchrist et al 2008, Hodge 2009). The escorting period lasts for around 60 days until pups reach nutritional independence at 3 months (Gilchrist 2004, Gilchrist & Russell, 2007). Being escorted provides clear benefits to pups, including higher provisioning rates, faster growth, lower mortality and earlier female reproductive maturity (Hodge 2005, Vitikainen et al 2017). Given the importance of the escorting relationship, variation in the level of care received may represent an important source of individual heterogeneity in banded mongoose pups.

The evolution of synchronous breeding results in groups containing large cohorts of similarly aged individuals and intense intragroup competition (Hodge et al 2011). There is no evidence that parents can discriminate their offspring from others in the communal litter (Vitikainen 2017). This reduces the effectiveness of infanticide as a form of reproductive control (Clutton-Brock et al 2001). Instead, to limit reproductive competition older females violently evict younger females from the group (Cant et al 2010, Thompson et al 2016). This threat of violence may represent a covert form of stress-related suppression as the remaining subordinate females show elevated stress levels during pregnancy and lower reproductive success (Sanderson et al 2015a). There is evidence that mothers increase prenatal investment by producing larger foetuses in response to competitive early life environments, as predicted by the PAR hypothesis (Inzani et al 2016). This response was particularly strong in adverse ecological conditions when food availability is limited and large size could confer an advantage over littermates. As such, pre-natal investment may adaptively vary according to conditions to improve the probability of offspring survival.

I monitored baseline cortisol secretion in banded mongoose pups via non-invasive faecal glucocorticoid metabolite measures in order to address three main questions.

1. **Do pups from lower ranked mothers have higher baseline GC?**

Maternal effects may influence offspring phenotype through age-related changes to maternal condition or investment strategies (Williams 1966, Stearns 1992). Here, low ranked mothers suffer from elevated stress during pregnancy and lower reproductive success (Sanderson et al 2015a). Studies suggest that exposure to high levels of maternal stress during development may impose constraints on offspring (Meaney et al 1994). I therefore predict that pups from mothers of low rank will show higher baseline GC than those from more highly ranked mothers, who are predicted to show lower stress levels in mid and late pregnancy.

2. **Does the care effort received by pups during the escort period influence early life baseline GC concentrations?**

Being escorted provides clear benefits to pups (Hodge 2005) including higher provisioning rates and faster growth. Given the evidence linking poor quality maternal care to gene modification and impaired HPA axis function (Weaver 2004, Liu 1997) I ask if levels of cooperative care influence early life stress physiology. I predict that pups receiving less care will have elevated baseline GC as a result of reduced provisioning and associated poor body condition.

3. **Does early life GC influence mortality risk?**

Elevated GC levels have been linked with impaired physical condition (Romero & Wikelski 2001, Love et al 2005) and, if chronically high, may be associated with multiple long-term physiological problems (Cohen et al 2007). I predict that elevated early life GC will be associated with poor physical condition and lower survival to nutritional independence. As elevated hormone concentrations may increase susceptibility to disease and be associated with long-term physiological constraints, early life stress may also be associated with a higher risk of mortality in the subsequent developmental stage. Therefore, I also test the effect of early life GC level on the probability of survival between 90 days and maturity at one year.

Materials and methods

Data Collection

This study was conducted on the Mweya Peninsula in Queen Elizabeth National Park, Uganda where the resident banded mongoose population is part of a long-term study (see Cant et al 2016 for further details). Each of the five sampled groups was visited at least three times a week and are habituated to close proximity of observation. Each visit lasted at least 20 minutes during which presence or absence of all individuals was noted. During the later stages of pregnancy groups were visited more frequently to establish exact birthdates for litters. To identify specific individuals all mongooses were captured within three weeks of emergence and then regularly recaptured until leaving the study through death or dispersal (for trapping protocols see Jordan et al 2010, 2011). During an individual's first capture a skin sample was taken for genetic analysis (Nichols et al. 2010) and the animal was microchipped for identification purposes. To allow individual identification during behavioural observations pups were marked with a unique combination of patches using hair dye and adults were marked with a unique shave on their back. For radio-tracking purposes individuals in each pack were fitted with a radio collar weighing 26-30g (Sirtrack Ltd, Havelock North, New Zealand). Weather data was collected by the Mweya weather station, and cumulative rainfall during the 30 days prior to either sampling or birth was used as a proxy of resource availability (Nichols et al 2012, Marshall et al 2016).

We collected 155 faecal samples from 79 mongoose pups (aged less than 90 days) in five social groups between May 2010 and December 2013. Faecal samples are predicted to estimate GC concentrations over the previous couple of days and may represent a mixture of baseline hormone secretion and response to acute stressors. Samples were then transported to the UK for analysis using material transfer permits obtained from the Uganda Wildlife Authority (UWA) and the Uganda Council for Science and Technology (UNCST), with UK import permits being obtained from DEFRA. Where possible, faecal samples were collected during the morning latrine session soon after emergence from the burrow. Faecal samples were collected by hand into small plastic bags (10cm x 5cm), labelled with the mongoose identity, date

and time, and placed on ice in a Thermos flask. Samples were then transferred to a -20 freezer within five hours of collection. Collection time and time to freezer were non-significant predictors of faecal glucocorticoid metabolite (fGC) concentration (Sanderson 2012). Over-marking of faeces is common in banded mongooses (Muller & Manser 2008) and samples were only collected where certain that no over-marking occurred. In addition, to avoid interference with group scent marking behaviour only half of each sample was collected. Hormone extraction was carried out at either Chester Zoo Endocrinology Lab (CZEL) or the University of Exeter in Cornwall (UofE) and used a wet-weight shaking process (adapted from Walker et al 2002) following sample thawing and manual homogenisation. Faecal glucocorticoid metabolite (fGC) concentrations were analysed using modified enzyme immunoassays (Young et al 2004, adapted from Munro & Stabenfeldt 1984). All assays were conducted at CZEL.

Genetic and Parentage Analysis

Due to the extreme birth synchrony in this species, maternity cannot be determined via observations and therefore genetic samples were used to assign parentage (Nichols et al 2010, Sanderson et al 2015b). DNA from tissue samples was used to construct a pedigree using 43 polymorphic microsatellite markers to estimate relatedness and assign parentage. The final pedigree used the programmes Masterbayes 2.51 (Hadfield et al 2006) and Colony 2.0.5.7 (Jones & Wang 2010) to infer parentage. Of the maternal assignments in this study 94% were made at greater than 90% confidence. For full details of DNA extraction, genotyping, parentage assignment and pedigree construction see Sanderson et al 2015b.

Measuring Escort Effort received

Banded mongooses exhibit a unique form of cooperative care known as escorting. Adult helpers provide exclusive care for individual pups during the developmental period between emergence at around 30 days and nutritional independence at around 90 days, although this time period will vary with each breeding attempt. As I am interested in early life fGC concentrations any faecal samples collected during the pup's first 90 days of life were used. Therefore, in all pups the escort period will

lie completely within our hormone sampling period. All adult members of a group may escort but subordinate males provide the majority of care. Escorting effort received by pups was calculated following the methods of Sanderson (2012) and Vitikainen et al (2017). Briefly, during the escorting period, groups were visited on average 12 times for a minimum of 20 minutes (the duration of one focal observation). Adults were scored as escorts if associated closely with the focal pup and were seen within 50cm of the same pup for more than 50% of the observation period (Vitikainen et al 2017). Previous studies have shown that this association score is a reliable summary of quantitative nearest-neighbour data (Gilchrist 2001, Gilchrist & Russell 2007). Escorting effort received per pup was calculated as the proportion of days that a pup was recorded with an escort out of the total number of days where escorting was recorded by any group member (7-21 observation days per breeding attempt). The escorting period was deemed to have finished when no adults were recorded as escorting. This gave a single escorting score (as a proportion) for each pup which was used to represent care received and can provide an accurate representation of the variability in escorting care between individuals.

Statistical Analysis and model selection

Statistical analyses were performed in R (R Core Team 2016). Linear and general linear mixed models (LMM's and GLMM's) were fitted using the lme4 package (Bates et al. 2015). Significance of terms was determined using likelihood ratio tests and non-significant interactions were dropped from final models to allow significance testing of the main terms (Engqvist 2005). Multivariate statistics were used to control for repeated sampling of individuals within groups. As reproductive conflict intensifies with increasing number of breeding females (Cant et al 2010) the total number of adult females in the group was fitted as a predictor variable. Likewise the number of pups present in the group at birth (emerged litter size plus any pups from previous litters that were under three months at birth) was included as a measure of early life competition. Total number of adult males was also fitted because males are the primary providers of social care, and group social structure may influence early life competition or maternal strategies. I did not include total group size as a predictor variable due to potential co-linearity. Rainfall is included as a predictor as it provides

a good proxy for food availability (see Marshall et al 2016). Likewise maternal weight is included in the model as it provides a good indicator of maternal condition which can strongly influence offspring phenotype. All models were checked using residuals and were found to meet the necessary assumptions for parametric tests.

Does maternal age rank influence offspring GC?

To test whether social and environmental factors influence early life GC levels a LMM with gausian error structure was fitted using log-transformed fGC concentration as the response variable (fGC followed a lognormal distribution). Maternal age rank at the time of offspring birth was fitted as an explanatory variables along with total rainfall in the month prior to sampling, the total number of pups in the group at emergence, pup age at sample, pup sex, the total number of adult males and the total number of adult females in the group at birth. Maternal ID, pack, individual ID and litter were included as random effects. Due to the behavioural and demographic differences observed between the sexes in this species interactions effects were also tested between all explanatory variables and offspring sex. Interaction effects that were not significant were excluded from the final model to allow for significance testing of the main terms. Explanatory variables and interaction effects were considered to be significant if $p = < 0.05$.

Where significant results are reported for age-related parameters such as maternal rank or pup age at sample I tested the relative importance of within versus between-individual effects (Van de Pol & Wright (2009)). This method allows the effect sizes in the fitted model to be separated to account for variation attributable to within (for example individual phenotypic plasticity) versus between (fixed responses based on individual or class) individuals. For pup age at sample this involved calculating the mean (average age when individual is sampled) and delta age (age at sample – mean age at sample) for each sample recorded during the escort period. This gives some insight as to whether an individual's fGC concentration changes over the escort period. As maternal age rank is closely correlated with maternal age we used a similar method to calculate mean (the average age at which each mother gives birth calculated from all her genotyped pups) and delta maternal age (maternal age at birth-mean age at which she gives birth over her lifespan). This allows us to

establish if fGC varies over a female's reproductive lifespan. In both cases mean age provides the between individual and delta age the between individual effect.

Does escorting effort received influence offspring GC?

To test whether the amount of care that an individual received during the escort period influenced fGC concentrations the above model was repeated with a smaller subset of samples taken only from within each groups recorded escort period. This represented the period between when escorting behaviour was first observed at the group and when no pups in the group was recorded in the presence of an escort. We added as an explanatory variable whether the sample was taken within the recorded period when that individual was observed as being escorted.

Do GC concentrations influence early life mortality risk?

To test whether early life GC concentrations influenced mortality risk two separate GLMM's were fitted using binomial error structure and logit link function. The response variable was pup survival, firstly from birth to 90 days, the end of the care period and nutritional independence, and secondly from 90 days to one year, approximately the age of reproductive maturity. In the second model individuals that were sampled and then died during the escorting period were excluded from the analysis. As each pup appeared only once in this dataset, repeat sampling was replaced with a mean GC value for each individual from all their collected samples during the escorting period. As a result, our measure of rainfall was changed from the month pre sample to the month pre-birth. The log-transformed mean fGC value was fitted as an explanatory variable along with maternal rank, maternal weight, pup sex, rainfall, total number of pups in the group at emergence and the number of adult males and females in the group at birth. Maternal ID, pack and litter were included as random effects.

Results

Does maternal age rank influence pup GC?

Model 1 revealed a significant interaction between maternal rank and pup sex (LMM, n=155, β ± s.e = -0.19 ± 0.07, $\chi 2$ = 9.07, p = 0.003) (model 1 in table 1 and fig 1a), suggesting that the effect of maternal rank on offspring fGC depended on sex. Female pups showed increasing fGC concentrations with maternal age rank (or as mothers became more subordinate), whereas male offspring showed a decline. In model 2, where rank was changed to within (delta maternal age) and between (mean maternal age) individual age effects there was a significant interaction between delta maternal age and pup sex on pup fGC (LMM, n=150, β ± s.e = 0.37 ± 0.13, $\chi 2$ = 6.97, p = 0.008) (model 2 in table 1 and fig 1b) but no effect of mean maternal age (LMM, n=150, β ± s.e = 0.04 ± 0.06, $\chi 2$ = 0.09, p = 0.77). Again the effect of delta maternal age on pup fGC depended on sex with the hormone levels of female pups declining more strongly over maternal reproductive lifespan than those of male pups.

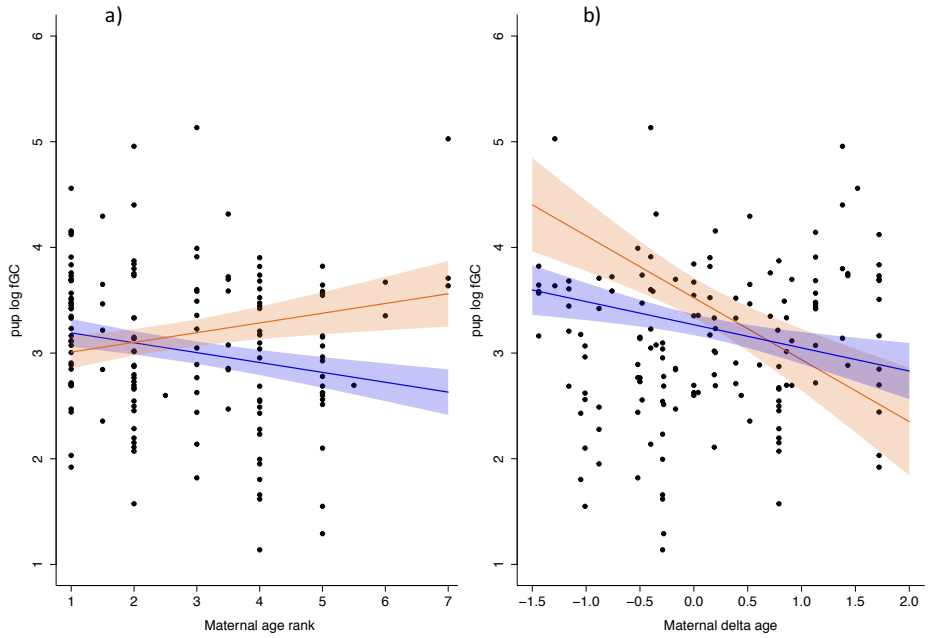

Figure 1. Offspring fGC as a function of maternal age rank (panel a) and maternal delta age (panel b). The interaction effect of both maternal age rank and delta age with sex significantly correlates with on pup fGC levels. Lines show predicted estimates and shaded areas represent confidence intervals. For both plots, orange shows female pups, and blue shows male pups and each point represents a fecal sample.

In model 1 offspring fGC concentrations were found to decline with pup age (LMM, n=155, β ± s.e = -0.009 ± 0.004, $\chi 2$ = 8.07, p = 0.004) (see model 1 in table 1). In model 2 where pup age was changed to within (delta age) and between (mean age) individual effects, offspring fGC concentration declined with mean age (LMM, n=150,

β ± s.e = -0.02 ± 0.006, $\chi 2$ = 14.51, p <0.001) (figure 2a) but not delta age (LMM, n=150, β ± s.e = -0.001 ± 0.005, $\chi 2$ = 0.05, p = 0.82) (figure 2b) (see model 2 in table 1). Pups sampled at a younger age tended to have higher fGC concentrations but there was no within individual decline in hormone levels during the escorting period. This suggests that individuals with high early life fGC may be selectively lost from the population during the escorting period.

	Response Variable	**Fixed Effect**	***β± s.e***	**$\chi 2$**	**P**	
Model 1	(log) pup fGC	Intercept	4.42 ± 1.03			
		Maternal Rank	0.09 ± 0.06	-	-	
		Pup sex	0.37 ± 0.21	-	-	
		Rainfall	**0.005 ± 0.002**	**7.62**	**0.006**	**
		No. adult females	**-0.10 ± 0.04**	**8.19**	**0.004**	**
		Maternal weight	-0.0005 ± 0.0007	0.15	0.70	
		No. pups	-0.02 ± 0.01	3.26	0.07	.
		No. adult males	**0.06 ± 0.02**	**12.27**	**0.0005**	***
		Pup age at sample	**-0.009 ± 0.004**	**8.07**	**0.004**	**
		Maternal age * Pup sex	**-0.19 ± 0.07**	**9.07**	**0.003**	**
		Number of observations	Samples = 155, Individuals = 79, Packs = 5			
Model 2	(log) pup fGC	Intercept	5.41 ± 1.01			
		Maternal delta age	-0.58 ± 0.25	-	-	
		Maternal mean age	0.04 ± 0.06	0.09	0.77	
		Pup. sex	-0.26 ± 0.11	-	-	
		Rainfall	**0.004 ± 0.002**	**5.40**	**0.02**	*
		Maternal weight	-0.0004 ± 0.0006	1.20	0.27	
		No. adult females	**-0.08 ± 0.04**	**6.26**	**0.01**	*
		No. pups	-0.02 ± 0.01	2.79	0.09	.
		No. adult males	**0.06 ± 0.02**	**11.77**	**0.0006**	***
		Pup delta age	-0.001 ± 0.005	0.05	0.82	
		Pup mean age	**-0.02 ± 0.006**	**14.51**	**0.0001**	***
		Maternal delta age * Pup sex	**0.37 ± 0.13**	**6.97**	**0.008**	**
		Number of observations	Samples = 150, Individuals = 75, Packs = 5			

Table 1. Output from a 2 LMMs predicting the effects of social and ecological conditions on pup faecal glucocorticoid (fGC) concentrations. Model 1 differs from model 2 in that both pup age at sample and maternal age rank were replaced with within and between individual age effects. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors (p=<0.05) are highlighted in bold.

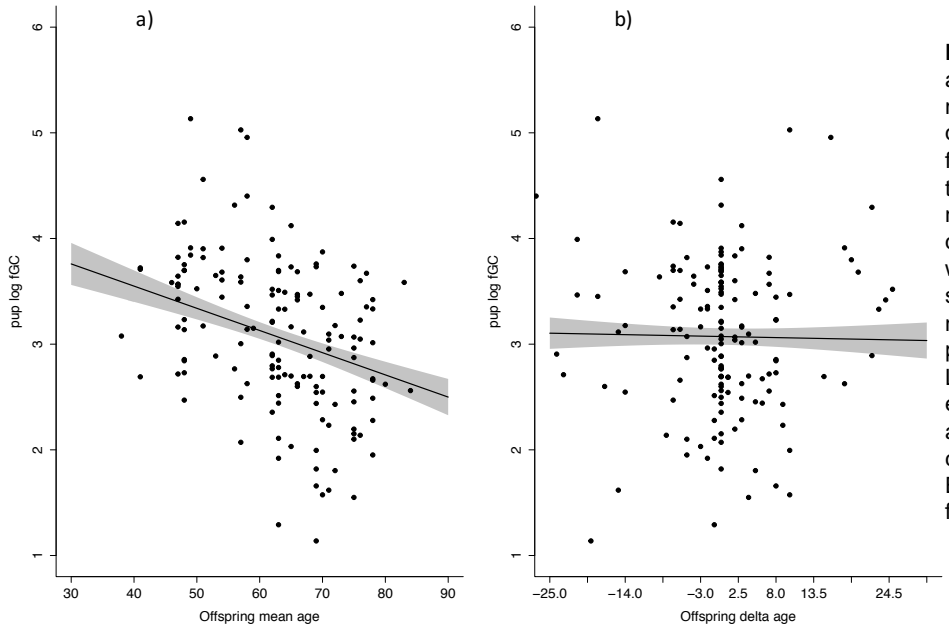

Figure 2. Offspring fGC as a function of offspring mean (panel a) and delta age when the faecal sample was taken (panel b). There is no correlation between delta age and pup fGC whereas mean age shows a significant negative correlation with pup fGC concentrations. Lines show predicted estimates and shaded areas represent confidence intervals. Each point represents a fecal sample.

Both models show that other social and ecological factors can significantly influence early life fGC concentrations. The number of females in the group at birth is negatively correlated with pup fGC (LMM, n=155, β ± s.e = -0.10 ± 0.04, $\chi 2$ = 8.19, p = 0.004) (fig 3a), whereas the number of males is positively correlated (LMM, n=155, β ± s.e = 0.06 ± 0.02, $\chi 2$ = 12.27, p = < 0.001) (fig. 3b). In addition, pup fGC levels are positively correlated with pre-sample rainfall (LMM, n=155, β ± s.e = 0.005 ± 0.002, $\chi 2$ = 7.62, p = 0.006) (fig 3c) and there is a non-significant negative correlation with the number of pups in the group at birth (LMM, n=155, β ± s.e = -0.02 ± 0.01, $\chi 2$ = 3.26, p = 0.07) (fig 3d).

Do levels of social care influence pup GC?

There was no relationship between total escort effort and fGC concentration in the smaller subset of pups that were sampled whilst their group was escorting (LMM, n=108, β ± s.e = 0.49 ± 0.36, $\chi 2$ = 2.05, p = 0.15) (table 2).

Response Variable	Fixed Effect	β± s.e	$\chi 2$	P
(log) pup fGC	Intercept	4.43 ± 1.20	-	-
	Maternal Rank	0.11 ± 0.08	-	-
	Pup sex	0.75 ± 0.44	-	-

Maternal weight	-0.0003 ± 0.0007	0.19	0.66
Escorting Effort	0.49 ± 0.36	2.05	0.15
Within pup esc period	-0.19 ± 0.20	0.98	0.32
Rainfall	0.003 ± 0.003	0.76	0.38
No. adult females	-0.08 ± 0.05	3.58	0.06 .
No.pups	**-0.02 ± 0.01**	**4.19**	**0.04 ***
No. adult males	**0.06 ± 0.02**	**8.49**	**0.003 ****
Pup age	**-0.02 ± 0.007**	**7.18**	**0.007 ****
Maternal rank * Pup sex	**-0.16 ± 0.09**	**3.93**	**0.048 ***
Number of observations	Samples = 108, Individuals = 57, Packs = 5		

Table 2. Output from a LMM predicting the effects of levels of social care on pup faecal glucocorticoid (fGC) concentrations. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors (p=<0.05) are shown in bold.

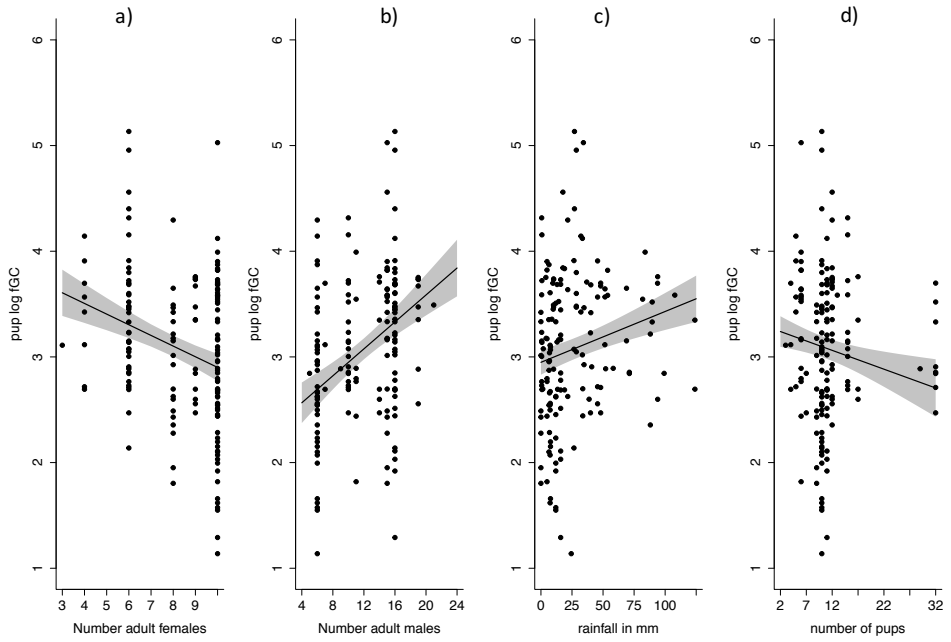

Figure 3. The influence of other social and ecological factors on pup fGC concentrations. The total number of females (panel a), males (panel b) and pups (panel d) in the natal group at offspring birth and rainfall in the month prior to fecal sample (panel c) as a function of pup fGC. Lines represent predicted estimates from LMM's and shaded areas represent confidence intervals. Each data point represents a fecal sample.

Does pup GC influence early life mortality rate?

Of the 79 individuals that were sampled for fGC levels during the escort period only 12 (15%) died before reaching three months of age (8 males and 4 females). My analyse reveals a negative correlation between mean pup fGC and survival to 90 days (GLMM, n=79, β ± s.e = -1.60 ± 0.79, $\chi 2 = 4.56$, $p = 0.03$) (table 3 and fig 4a). This suggests that pups with higher fGC levels are less likely to survive to nutritional independence. In addition, there is a non-significant positive correlation between rainfall and pup survival (GLMM, n=79, β ± s.e = 0.71 ± 0.40, $\chi 2 = 3.25$, $p = 0.07$).

Response Variable	Fixed Effect	$\beta \pm s.e$	$\chi 2$	P
Survival to 90 days	Intercept	3.67 ± 3.57		
	Maternal rank	-0.07 ± 0.29	0.06	0.81
	Escort level	0.69 ± 1.54	0.20	0.66
	Pup sex	-1.05 ± 0.79	1.92	0.17
	Rainfall (standardised)	0.71 ± 0.40	3.25	0.07 .
	No. adult females	0.20 ± 0.24	0.72	0.40
	Maternal weight (standardised)	-0.40 ± 0.56	0.52	0.47
	No. pups	0.09 ± 0.07	1.74	0.19
	No. adult males	0.11 ± 0.13	0.77	0.38
	Mean early life GC (log)	**-1.60 ± 0.79**	**4.56**	**0.03 ***
	Number of observations	Individuals = 79, Packs = 5		

Table 3. Output from a GLMM predicting the effect of social and ecological conditions on pup survival to nutritional independence at 90 days. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors are highlighted in bold.

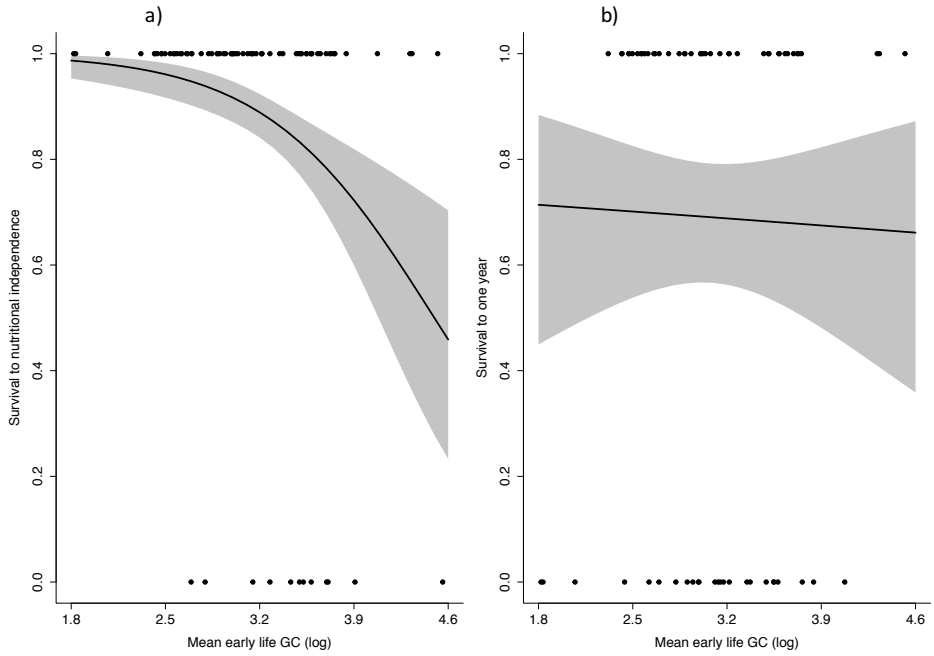

Figure 4. The probability of a pup surviving to 90 days (panel a) and 1 year (panel b) as a function of offspring mean fGC during the escorting period (30 to days days). The effect of mean pup fGC was a significant predictor of survival to 90 days but not one year. Lines show predicted estimates and shaded areas represent confidence intervals. In both plots each point represents an individual .

In contrast, there is no evidence that early life fGC influences survival probability to one year (GLMM, n=67, β ± s.e = -0.11 ± 0.76, $\chi 2$ = 0.01, p = 0.92) (table 4 and fig 4b). Both the number of pups in the group (GLMM, n=67, β ± s.e = 0.38 ± 0.17, $\chi 2$ = 9.36, p = 0.002) and maternal weight (GLMM, n=67, β ± s.e = -1.66 ± 0.79, $\chi 2$ = 5.83, p = 0.02) are significant predictors of survival probability to one year amongst this sampled group. When there are more pups in the group or their mothers are of

low weight then individuals are less likely to die between 3 and 12 months of age. In total, 37 (47%) of the 79 pups sampled failed to survive to one year, of which 19 were male and 18 were female, with 25 (32%) dying between 3 and 12 months of age.

Response Variable	Fixed Effect	$\beta \pm s.e$	$\chi 2$	P	
Survival to 1 year	Intercept	-3.82 ± 4.12			
	Maternal rank	-0.02 ± 0.28	0.001	0.97	
	Escort level	0.76 ± 1.86	0.16	0.69	
	Pup sex	0.88 ± 0.74	1.51	0.22	
	Rainfall (standardized)	0.57 ± 0.63	0.88	0.35	
	No. adult females	-0.21 ± 0.35	0.32	0.57	
	No. adult males	0.11 ± 0.14	0.63	0.43	
	Maternal weight (standardised)	-1.66 ± 0.79	5.83	0.02	*
	No. pups	0.38 ± 0.17	9.36	0.002	**
	Mean early life GC (log)	-0.11 ± 0.76	0.01	0.92	
	Number of observations	Individuals = 67, Packs = 5			

Table 4. Output from a GLMM predicting the effect of social and ecological conditions on pup survival to one year. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors are highlighted in bold.

Discussion

Although poor early life conditions are predicted to constrain offspring development, particularly in competitive social environments, I found no evidence that the level of escorting received affected pup GC concentrations in early life. This fails to support my prediction that lower levels of cooperative care would be associated with higher pup stress. Maternal rank did influence offspring stress levels, though not as predicted. Rather than the elevated maternal stress reported for younger mothers transferring to offspring, there was a sex-specific effect of maternal rank on pup GC. As maternal rank decreases the fGC concentrations of female pups increases, whereas that of males decreases. The analysis also shows that as mothers age the fGC of their pups decreases and again this effect is sex-specific, with the response being stronger in daughters than sons. Although it remains unclear whether or in what context early life GC levels are adaptive, this suggest that there may be age-related changes in maternal allocation strategies. However, such interactions may not directly constitute maternal effects, the influence of which on phenotypic traits remain difficult to disentangle from the impact of external conditions, particularly given the sex differences reported in patterns of care (Vitikainen et al 2017). Interestingly, both low rainfall and high competition in early life as associated with relatively low fGC concentrations. This is unexpected given that both are predicted to be associated with reduced food availability and such stressors are often associated with elevated glucocorticoids. It therefore seems likely that our fGC measurements give an indication of baseline hormone levels, which might be associated with energy consumption and growth, as well as acute stressors.

There was also evidence that elevated fGC levels during early life correlate with increased mortality risk. This appears to be supported by the within-individual analysis of pup age which suggests that pups with elevated fGC are selectively lost from the population. However, there is no association between fGC concentrations and mortality risk during the subsequent period between nutritional independence and adulthood. This suggest that elevated early life GC may be associated with immediate but not delayed survival costs. This is quite a striking result given that only 15% of our sampled pups died during the escorting period, a far lower pup

mortality rate than expected from Nichols et al 2011 where 47% of pups were found to die before three months. Our sample is inevitably already skewed towards pups that lived long enough to have a faecal sample collected. Due to this apparent underrepresentation of early life mortality it does not seem prudent to discuss any of the other variables that were found to predict either survival to 90 days or adulthood.

My initial prediction that lower levels of cooperative care would be associated with high GC was based on the benefits derived from being escorted (Hodge 2005). The extent of these benefits renders it likely that poor care is accompanied by physiological costs, especially when pups compete with littermates that receive more frequent provisioning. If offspring do suffer costs, then my failure to detect them may have several explanations. First, cooperative care may help buffer against the maladaptive outcomes associated with poor maternal condition. Indeed, a recent study (Marshall et al in prep) suggests that, under some conditions, escorts may preferentially target weaker or lighter pups for care, although it is more usual for heavier pups to receive more attention (Vitikainen et al 2017). Alternatively, escorting may occur too late in development to influence stress physiology, with the earlier babysitting period potentially being more critical. Finally, the multi-functional nature of GCs, especially their role in mediating growth rates and energetic demand (Sapolsky 2000), makes any direct correlation with care hard to detect in a longitudinal study. My prediction of high stress in pups that receive less care may be obscured by a similar increase in the baseline GC concentrations of well provisioned pups that grow faster. Therefore, a controlled experimental approach may be needed to isolate the relative costs of variable care within litters. In addition, alternative physiological markers, like oxidative damage, may better indicate costs that only appear in later life. Allocating resources towards traits that accelerate growth may trade-off with allocating resources to traits, like somatic repair, that enhance lifespan (Kirkwood 1977, English & Uller 2016). Therefore, any effects of poor early life care may manifest themselves in adulthood (Nettle et al 2015, Andrews et al 2017), rather than be expressed as variation in early life hormone levels.

Many of the studies linking poor maternal care with developmental impairment in offspring have identified maternal stress as a key mechanism (Meaney 2001, Stier et

al 2012). Post-natal care is energetically expensive (Clutton-Brock et al 1989) and by removing this burden exclusively from mothers, cooperative care may limit the maladaptive effects of maternal stress on offspring. In communally breeding degus (*Octodon degus*), maternal stress was found to affect HPA axis development but deleterious effects were limited by the presence of non-parental carers (Bauer et al 2015). Laboratory studies that associate stress-induced maternal care quality with epigenetic changes to offspring stress physiology (Weaver et al 2004, 2005) measure care in the first few weeks of life. Escorting begins after emergence from the burrow at around four weeks and therefore may take place too late in development to affect offspring stress physiology. Similarly, patterns of DNA methylation in cooperatively breeding superb starling (*Lamprotonis superbus*) chicks did not change between one week of age and adulthood (Rubenstein et al 2016). Prior to escorting, and during the period when pups may be susceptible to gene modification, another form of cooperative care, babysitting, is exhibited. Although, like escorting, babysitting provides benefits to pups (Cant 2003) it takes place within the den and therefore within-litter variation is hard to quantify. The majority of both forms of care is provided by non-breeders (Gilchrist & Russell 2007) and may allow mothers to effectively uncouple pre and post-natal investment. As such, females can concentrate resources on the gestation period rather than allocating reserves of energy for pup care. During early life, allolactation represents the main source of pup nutrition (Gilchrist 2006) and may further reduce the burden of post-natal investment on energetically constrained mothers. This division of reproductive labour within groups suggests that pre-natal effects may have a stronger influence on offspring phenotypes than post-natal effects.. In addition, it may enable pups from younger, subordinate mothers to compete with littermates from dominant females despite the constraints faced by younger breeders via reproductive suppression. However, it remains difficult to separate the significance of pre and post-natal effects with the correlational data used in this study.

Elevated GC concentrations are associated with energetically demanding activities, including growth (Dantzer et al 2013, Strange et al 2016). Here, rather than being a physical indicator of poor condition (Kitayski 1999, Love et al 2005), early life GC levels may reflect the energy resources utilised for growth. GCs are predicted to mediate allostatic mechanisms that ensure internal stability and match energy

demand to food availability, and as such, may be associated with plasticity in growth or life-history trajectories. In constrained environments developing organisms respond to food supply by adjusting energy expenditure and metabolic rates (Pontzer 2015, 2017). Metabolic rate reflects the energetic cost of living and is likely to influence life-history traits, including age at maturity and lifespan (Glazier 2015). Faster metabolic rates are associated with increased energy acquisition and faster rates of somatic growth (Biro & Stamps 2010, Auer et al 2018). However, accelerated growth rates may be associated with biological processes that are costly to survival (Finkel & Holbrook 2000, Monaghan & Haussmann 2006). A key function of GCs is regulation of physiological processes, including metabolic rate (Haase 2016, Sapolsy 2000) in response to external stressors. Here, pup baseline GC levels decline in response to low rainfall and may represent an adaptive response to prioritise survival over growth in nutritionally poor conditions. That pups who receive less care do not also show lower GC might reflect within-litter competitive growth (Huchard et al 2016), or individual behavioural changes such as switching from begging to independent foraging in response to low care. Any early life adaptive growth or behavioural response, whether due to competitive pressure or adverse conditions, is likely to be associated with life-history trade-offs in later life.

This study provides some evidence to support the prediction that pups from lower ranked mothers would show elevated GC concentrations. The fGC concentrations of daughters from lower ranked mothers was higher but those of their sons was not. In considering this result we need to account for both differences between the sexes and the change in pup fGC with maternal rank. Sex-specific fitness responses to challenging early life conditions have been reported in this (Marshall et al 2017) and other species (Rubenstein et al 2016, Wilkin & Sheldon 2009, Garrett et al 2015). Other studies have reported sex-specific responses to maternal stress (St-Cyr et al 2017, Bronson & Bale 2016). Banded mongoose males that experience low rainfall in their first year live longer at a cost of reduced fertility (Marshall et al 2017). Here low food availability may trigger a trade-off between survival and fitness which mirrors studies showing correlation between calorific restriction and longevity (Colman 2009, Masaro 2006). This effect was not found in females, who gain weight faster in good conditions and individuals of low weight suffer increased mortality in poor conditions (Marshall et al 2016). If female offspring are more vulnerable to

nutritionally poor early life conditions, then viability selection may ensure that only the best quality female offspring survive. Therefore, males may show greater plasticity in life-history trajectories in response to early life conditions, whereas, females may be selected to adapt to the conditions experienced in adulthood. As such female reproductive competition may prevent strategies that increase longevity at the expense of growth being favoured.

That selection imposes different pressures on male and female pups is not surprising given the marked demographic and behavioural differences in this species. Females generally breed earlier than males and reproductive success is more closely linked to early life weight, whereas males tend to live longer (Vitikainen el at 2016). These patterns may result in stronger selection in females than males for traits that enhance competitive ability. Where such traits are associated with weight gain, individuals are expected to have higher energetic expenditure, which could be associated with elevated GC concentrations. Although there is no evidence that mothers can identify their own offspring and direct attention towards them patterns of care may help explain our sex-specific interaction. The escorting effort received by pups shows marked variation between the sexes (Vitikainen et al 2017). Male pups receive more care than females as escorts associate with pups by sex and adult males provide more care and usually outnumber adult females in packs. As mothers are more likely to escort when they have offspring in the litter but care provision declines in heavier older females this may leave the daughters of more dominant mothers with fewer available female escorts than those of lower ranked mothers. Similarly, lower ranked females may only breed in more plentiful conditions and have more resources available for offspring care. As such, female pups might have to compete more intensely for early life care then males. This competition is likely to favour larger pups and although both male and female pups that are heavier at emergence receive more care the response is stronger in females. Therefore, being of heavy weight at emergence may provide greater benefit to female than male pups due to the competitive advantage that it provides over littermates.

Other factors underlying the apparent change in investment strategies with maternal age may be more difficult to disentangle. In banded mongooses, mothers of low rank are more likely to abort their litters (Inzani 2016), be evicted (Cant et al 2010), and

have lower reproductive success (Sanderson et al 2015a). Despite this strong selection pressure, there is no evidence that the surviving pups of younger females suffer from developmental constraints in early life. If the elevated GC concentrations found in daughters from subordinate mothers is linked with increased weight or growth rate, this again may be evidence of viability selection. The strong pre-natal selection on low ranked females resulting from suppression and competition may insure that only those in good condition reproduce. Where traits associated with maternal quality are more closely linked to female than male fitness then those traits might be expressed more in female then male offspring. In this species the higher female mortality risk in sub-optimal conditions and the possible increased competition for escorts may result in investment being directed towards daughters when mothers are in good physical state. With this in mind it is strongly recommended that future studies should consider the influence of both offspring fGC concentrations and maternal rank on pup weight at emergence and growth rates.

Alternatively, studies suggest that the extent of maternal effects on offspring phenotype might be affected by age-related changes in maternal condition or allocation (Curio 1983, Stearns 1992). The age-related deterioration of organismal function or fitness is known as senescence and studies across taxa link increasing maternal age with decreasing offspring performance (Moorad & Nussey 2016). The observed within-individual decline in offspring GC levels, which is stronger in daughters than sons could be due to senescent decline of traits that correlate with female fitness. Markers of oxidative damage (a mechanism underlying life-history trade-offs between survival and reproduction (Blount et al 2015)) are known to vary with maternal age (Vitikainen et al 2017). High levels of oxidative damage are associated with lower survival and were consistently higher in females than males, although pregnant females showed relatively low levels, suggesting that mothers may shield their offspring from physiological costs. This effect was less marked in older than younger mothers, suggesting that shielding mechanisms may suffer from senescent decline. More damage during pregnancy predicted lower reproductive success and again this effect was stronger in older breeders. Similar effects are reported in birds, where older mothers may be limited in their provision of Antioxidants to eggs with adverse effects on offspring development (Beckman & Ames 1998, Toruk et al 2007). Where females suffer higher costs than males, and

oxidative damage reduces reproductive success, maternal shielding may provide stronger fitness benefits to daughters than sons. Similarly, if shielding ability diminishes with age, then daughters of younger, lower ranked mothers may receive greater benefits. The high GC levels observed in daughters from subordinate mothers may provide a competitive advantage in terms of growth and fitness, despite the mortality risks of chronically elevated stress. As such, maternal shielding may enable the daughters of younger mothers to maintain fast growth (and associated high GC concentrations) without suffering the maladaptive effects of oxidative damage or chronic stress.

Pre-natal social conditions may also influence maternal investment decisions and offspring phenotype. Here increasing numbers of adult female group-mates predicted lower pup GC, with a contrasting trend for males where increasing numbers predicted higher pup GC. If we infer that the number of females indicates levels of reproductive competition and the number of males indicates care availability, then low pup GC levels may represent variable weight or growth rate in different social conditions. Maternally derived phenotypic changes that prepare pups for early life environments and are predictable from social cues may represent a form of adaptive plasticity (Bateson et al 2015) known as predictive adaptive response (PAR) (Gluckmann et al 2004). There is evidence for PARs in this species as maternal prenatal investment increases with the number of potential breeders in the group (Inzani et al 2016). This response was particularly strong in poor ecological conditions. The role of GCs in allostasis make them good candidate mechanisms for mediating plasticity via maternal hormonal signalling (Dloniak et al 2006). In human studies, the thrifty phenotype hypothesis (Hales & Barker 1992) suggests that early-life metabolic adaptations increase survival chances by selecting appropriate growth trajectories in response to environmental cues (Wells 2007). Recent studies suggest that maternal stress in early pregnancy may recalibrate life-history trajectories towards faster growth and earlier reproduction (Berghanel et al 2017). Here we see downregulation of stress physiology in response to adverse conditions and sex-specific upregulation in response to maternal rank, suggesting some plasticity in investment strategies, possibly prompted by pre-natal cues. That the number of males predicts higher GC levels may also represent a predictive cue, perhaps triggering reduced maternal investment in response to increased care availability

(Savage et al 2015).

In summary, despite the benefits associated with escorting, levels of cooperative care did not influence GC concentrations in early life. This lack of effect may be explained by de-coupling of pre and post-natal maternal investment as non-parental carers ensure that energy allocation towards offspring is minimised post birth. Therefore, pre-natal maternal effects assume greater importance and play a significant role in determining pup phenotype. More research is needed to establish links between maternal stress during pregnancy and offspring growth trajectories, and whether these effects are sex-specific. Maternal age had a sex-specific effect on pup GC and work is needed to establish the fitness consequences of early life stress, and whether they differ between the sexes. Additional research should focus on age-related maternal decline and whether senescence has a stronger selective impact on daughters than sons. Finally, we revealed that high early life GC is associated with an immediate, but not a delayed increase in mortality risk, this might reflect energetic deficits associated with immediate causes of mortality, such as disease or starvation.

Chapter 4: Sex-specific maternal effects and offspring sex ratio in a cooperative mammal.

Abstract

Patterns of reproductive investment strongly influence both offspring and maternal fitness. Where maternal traits affect the fitness of sons and daughters differently, mothers may strategically direct resources towards the sex with stronger fitness benefits or adaptively adjust offspring sex-ratio. In many species, maternal aging is associated with declining offspring performance and mothers might be selected to make sex-specific age-related adjustments in resource allocation. Currently little is known about how optimal investment strategies shift over maternal lifespan or how they influence offspring development and fitness. Banded mongooses (*Mungos mungo*) are a cooperatively breeding mammal in which demographic and behavioural traits differ widely between the sexes. Here I test whether the effect of maternal age on performance related life-history traits differs between the sexes and whether offspring sex-ratio is influenced by maternal age. I found no evidence that maternal age has sex-specific effects on offspring survival or lifespan. However, the effect of maternal age on lifetime reproductive success did differ between the sexes, with lifetime reproductive success of daughters, but not sons, declining with maternal age. Maternal age also affected offspring sex-ratio, with the proportion of daughters produced declining with maternal age. This study suggests that maternal effects can have sex-specific impacts on traits that are associated with individual quality. These effects may prompt mothers to strategically allocate resources to the sex favoured by selection, with the potential for biased offspring sex-ratios to arise.

Introduction

Life-history theory predicts that with advancing age reproductive effort should increase as future reproductive potential decreases (Williams 1966, Daunt et al 1999). This age-related change in resource allocation has been associated with increasing litter size and offspring survival in mammals (Atkins-Regan 2005). In contrast, many studies have reported a decrease in offspring quality as mothers age, known as maternal senescence (Moorad & Nussey 2016). Senescence is the age-related decline of organism function and fitness and is traditionally defined as a weakening in the strength of natural selection to preserve survival and reproduction with age (Hamilton 1966, Williams 1957). Most research on ageing has considered the impact on either organism survival (Charlesworth 2001) or fertility (Nussey et al 2006) and has paid little attention to the influence of social or maternal effects (but see Keller & Genoud 1997). Negative correlations between maternal age and the health, lifespan or fitness of offspring have been reported across taxa, including in humans (Mahy 2003), mammals (Descamps et al 2008) and birds (Froy et al 2013). These deleterious effects on offspring quality are linked with maternal resource allocation, either pre-birth, through age-linked physiological constraints, or post-birth, through a reduced ability to provide adequate offspring care (Bogdanova et al 2006, Nussey et al 2009, Beamonte-Barrientos et al 2010).

Parents face selection to adjust reproductive investment according to their available resources (Clutton-Brock et al 1981). If social or environmental conditions influence the fitness of males and females differently, parents may be selected to vary resource allocation depending on offspring sex (West & Sheldon 2002, West 2009). Where optimal investment strategies differ there is potential for adaptive variation in offspring sex-ratios (McCleod & Clutton-Brock 2013, Nichols et al 2014). Traditional sex allocation theory predicts that an even sex-ratio at breeding age should be favoured by selection. Thus investment may be skewed towards the rarer sex, or that suffering higher mortality during development (Fisher 1930). Later work suggests that, where maternal traits influence offspring fitness, sex-ratios may be biased towards the sex with higher reproductive skew (Trivers & Willard 1973). In many species, the sex that invests less heavily in reproduction, usually males,

competes for breeding opportunities and shows stronger secondary sexual characteristics (Trivers 1972). Therefore, favourable early life conditions, or silver spoon effects (Grafen 1988), should benefit the competitive sex more as they obtain greater fitness benefits from being of good quality. Thus offspring sex-ratio may vary with maternal state as mothers in good condition invest more resources in individuals of the competitive sex. Studies suggest that offspring sex-ratio can be affected by other parameters related to maternal condition including age (Saltz 2001) and social rank (Meikle et al 1984, Sheldon & West 2004). In species where maternal rank is inherited, like some baboons, rank correlates with breeding success and dominant females produce an excess of daughters who are themselves more successful (Altmann 1980, Silk 1993). Whereas, in polygynous species like red deer (*Cervus elaphus*), high ranking females produce an excess of sons as male fitness is strongly linked with competitive ability (Clutton-Brock et al 1981, 1984).

The Trivers-Willard theory has been tested across taxa, including in birds (Wiebe & Bortollotti 1992), mammals (Sheldon & West 2004) and humans (Ruckstuhl et al 2010) but has received mixed support (Hewison & Gaillard 1999, Brown 2001). In some cases, empirical data does not match theoretical predictions and it remains unclear whether mothers can physiologically determine offspring sex (Williams 1979, Uller et al 2007). To account for the effect of sex-specific traits on adaptive offspring sex-ratios, theoretical work predicts that reproductive value (the fraction of the population descended from an individual) may represent a better measure of fitness than the number of offspring produced (Leimar 1996, Schindler et al 2015). Of particular importance is how reproductive value can change in response to sex differences in life-history traits, particularly early life mortality and growth or age-related reproductive output in adulthood. Schindler (2015) suggested that the predicted sex-ratio of a sexually dimorphic species (Bighorn sheep (*Ovis canadensis*)) could be altered by increasing early life and size based mortality rates. In this species demographic rates are dependent on individual weight and age and the sexes show different mortality schedules throughout life. Likewise, in a non-sexually dimorphic species (Columbian ground squirrel *Urocitellis columbianus*) an overproduction of daughters was expected from good quality mothers to maximise reproductive value. Models showed that the predicted sex-ratio could be reversed by

increasing early life male mortality, as this was associated with an increase in the reproductive value of sons as males became the rarer sex.

In some cooperative species females face stronger reproductive competition than males (Clutton-Brock et al 2006, Bennett & Faulkes 2000). Here traits that enhance competitive ability may have a stronger effect on female than male fitness returns and thus mothers in good condition are predicted to invest more resources in female offspring and produce an excess of daughters. Interactions between relatives in cooperative groups may influence sex-allocation decisions in other ways. The local resource competition model (Clark 1978, Silk & Brown 2008) predicts that females may suffer fitness costs by producing daughters that act as future competitors for local resources. As such, offspring sex-ratios may be biased towards the dispersing sex, although the strength of any effect will vary with maternal age or length of reproductive tenure. Older breeders may not be selected to adjust sex-ratios if they are likely to die before their daughters become reproductively mature. Similarly, in cooperative species sex-ratio adjustment may enhance local resources (Emlen et al 1986) by producing an excess of the more helpful (Komdeur et al 1997, Creel et al 1998) or cooperative sex (West et al 2009). The adaptive value of this adjustment will depend on the benefits provided by helpers, with species where helpers do not benefit offspring fitness unlikely to show an observable bias (Griffin et al 2005).

Where life-history traits differ between the sexes the selection pressures faced by males and females may also differ (Wilkin & Sheldon 2009, Kruuk et al 1998). Sexually-antagonistic selection can be a mechanism for maintaining individual variation when males and females show different optimal traits (Bonduriansky & Chenoweth 2009, Cox & Calsbeek 2009). In banded mongooses, females suffer higher mortality than males (Marshall et al 2016) with individuals of low weight particularly vulnerable, especially in adverse conditions. Sex differences in mortality rates may be associated with sensitivity to ecological conditions (Conradt et al 2000, Coulson et al 2001) with the larger sex, being more vulnerable to adverse conditions due to increased energetic demands (Clutton-Brock et al 1985, Isaac 2005). Banded mongooses do not show pronounced sexual dimorphism and it is suggested that the energetic requirements of reproduction may account for higher female mortality (Marshall et al 2016). Males generally breed at an older age than females and their

reproductive success is less closely associated with early life weight (Hodge 2005). Therefore, males may show greater plasticity in life-history strategies than females (Marshall et al 2017) as the fitness costs associated with slower growth or later reproductive maturity are less. These demographic differences may contribute to the male dominated sex-ratios observed in groups (Cant et al 2016) and explain why males generally provide a greater share of energetically costly cooperative care (Sanderson et al 2014).

Little is known about how optimal investment strategies may shift over maternal lifespan or how they influence offspring development and fitness. The demographic and behavioural sex differences seen in banded mongooses make them a good system to investigate such questions. Banded mongooses are cooperatively breeding mammals that live in mixed sex groups of between approximately 8 and 40 individuals. They reach sexual maturity at around one year and although all adults are capable of breeding (Cant et al 2013), reproductive opportunities are skewed towards older individuals (Nichols et al 2010, 2012). Packs have up to four litters per year which are highly synchronized within (but not between) groups, with multiple females giving birth to a large communal litter on the same day (Cant 2000, Hodge 2011). The evolution of synchronous breeding results in large cohorts of similarly aged individuals and intense competition within groups (Hodge et al 2011). To control reproductive competition older females violently evict younger females from the group (Cant et al 2010, Thompson et al 2016). Mothers increase pre-natal investment when their pups are likely to face a competitive environment (Inzani et al 2016), suggesting that resource allocation may adaptively vary according to social and ecological conditions. Previous studies show that older females reproductively suppress younger females, particularly in adverse conditions (Nichols et al 2012). This suppression leads to younger mothers having elevated stress levels during pregnancy and lower reproductive success (Sanderson et al 2015a) but it remains unclear whether their pups suffer fitness costs.

My previous chapter found that maternal rank, which is closely correlated with maternal age, has a sex-specific effect on pup glucocorticoid (GC) concentrations. GCs are known as “stress hormones” due to their functional role in energy metabolism and the physiological response to external stressors. The baseline GC of

daughters decreases over a mother's reproductive lifespan, whereas that of sons increased, though the effect appears stronger in female offspring. Here I test whether this age-related sex difference in early life hormonal profiles represents evidence of a shift in optimal sex allocation strategies over maternal lifespan. Although the influence of early life GC on fitness remains unclear pup survival rates were also found to suffer a decline over maternal lifespan. Therefore, I will use the banded mongoose dataset to investigate two key questions.

1. **Does the effect of maternal age on offspring life-history traits differ between the sexes?**

Early life conditions and their effect on levels of parental investment can influence offspring phenotypic traits (Lindström 1999, Metcalfe & Monaghan 2003) and these effects can differ between the sexes (Clutton-Brock et al 1985). I predict that the fitness of female pups will be more strongly affected by maternal effects than that of male pups. Therefore, due to age-related changes in maternal condition, the fitness of daughters will decline with maternal age, whereas that of sons will not. To test this, I will measure whether the effect of maternal age on the probability of survival, lifespan and lifetime reproductive success of banded mongoose pups differs by sex.

2. **Does maternal age influence offspring sex-ratio?**

In species where traits that enhance competitive ability benefit one sex more than the other mothers in good condition may gain fitness benefits from adjusting offspring sex-ratio (Trivers & Willard 1973). In banded mongooses, females have higher mortality rates, particularly when they are of low weight (Marshall et al 2016). In my previous chapter I speculated that the fitness of female pups may suffer greater costs from constrained maternal investment than that of male pups. Therefore, if viability selection leads to only high quality younger females breeding they may produce more daughters than older mothers. As such, I predict that resource allocation may vary over female reproductive lifespan and the proportion of daughters produced may decline with maternal age.

Materials and Methods

Data Collection

This study was conducted on the Mweya Peninsula in Queen Elizabeth National Park, Uganda where the resident banded mongoose population is part of a long-term study (see Cant et al 2016 for further details). The data used for this study was collected between May 2000 and December 2013, with records of mortality and reproductive success included up until May 2017. Each of the sampled groups was visited at least three times a week and were habituated to close proximity of observation. Each visit lasted at least 20 minutes during which presence or absence of all individuals was noted. During the later stages of pregnancy groups were visited more frequently to establish exact birthdates for litters. To identify specific individuals all mongooses were captured within three weeks of emergence and then recaptured regularly until leaving the study through death or dispersal (for trapping protocols see Jordan et al. 2010, 2011). During an individual's first capture a skin sample was taken for genetic analysis (Nichols et al. 2010) and the animal was microchipped for identification purposes. To allow individual identification during behavioural observations pups were marked with a unique combination of patches using hair dye and adults were marked with a unique shave on their back. For radio-tracking purposes individuals in each pack were fitted with a radio collar weighing 26-30g (Sirtrack Ltd, Havelock North, New Zealand). Weather data was collected by the Mweya weather station, and cumulative rainfall during the 30 days prior to either sampling or birth was used as a proxy of resource availability (Nichols et al 2012, Marshall et al 2016).

Genetic and Parentage Analysis

Due to the extreme birth synchrony in this species, maternity cannot be determined via observations and therefore genetic samples were used to assign parentage (Nichols et al 2010, Sanderson et al 2015b). DNA from tissue samples was used to construct a pedigree using 43 polymorphic microsatellite markers to estimate relatedness and assign parentage. The final pedigree used the programmes Masterbayes 2.51 (Hadfield et al 2006) and Colony 2.0.5.7 (Jones & Wang 2010) to

infer parentage. Of the maternal assignments in this study 94% were made at greater than 90% confidence. For full details of DNA extraction, genotyping, parentage assignment and pedigree construction see Sanderson et al 2015b.

Statistical Analysis and model selection

Statistical analyses were done in R (R Core Team 2015) with linear and generalised linear mixed models (LMMs and GLMMs) fitted using the lme4 package (Bates et al. 2015). Significance of terms was determined using likelihood ratio tests and non-significant interactions were dropped from final models to allow significance testing of the main terms (Engqvist 2005). Multivariate statistics were used to control for repeated sampling of individuals within groups. The explanatory variables differ from the model in the previous chapter as when investigating offspring sex-ratio I used group sex ratio as an indicator of maternal competition and total group size as a measure of group competitive ability. All models were checked using residuals and were found to meet the necessary assumptions for parametric tests. Variables were reported as significant if $p < 0.05$.

Does the effect of maternal age on offspring life-history traits differ between the sexes?

A dataset was compiled of all individuals for which I had details of maternal ID and precise age at birth. To examine the effect of maternal age on infant mortality a similar approach was used as in the previous chapter. A GLMM was fitted using a binomial structure and logit link function with pup survival to one year as the response variable. The key explanatory variable was the interaction between maternal age and offspring sex and was fitted along with rainfall in the month pre-birth, group size, group sex ratio and the number of pups in the group at birth. Maternal ID, pack and litter ID were included as random effects.

To test whether maternal age had a sex-specific influence on lifespan, a LMM was fitted with a gausian error structure with log-transformed individual lifespan as the

response variable. All individuals that died within one year were excluded from the dataset. I used the same explanatory and random variables as in the above model.

Individual lifetime reproductive success was calculated using the banded mongoose project pedigree. Here the analysis was conducted in two stages; first the factors affecting whether an individual bred successfully were tested and for those individuals that bred a second model examined the factors influencing the total number of offspring produced. In both models the interaction between maternal age and sex was the main explanatory variable and the other explanatory and random variables are as above. To test whether maternal age influenced the chance of reproducing of males and females differently a GLMM was fitted using a binomial structure and logit link function with whether an individual bred as the response variable. Those individuals that failed to breed were then excluded from the dataset and a GLMM using a poisson distribution was constructed to test whether maternal age influenced the number of offspring produced differently depending on sex. As the number of offspring is expected to increase with the number of opportunities to breed we include (log)lifespan as an offset function.

Does maternal age influence offspring sex ratio?

To examine whether maternal effects influenced offspring sex ratio a GLMM with binomial error structure and logit link function was fitted. The response variable was offspring sex and the main explanatory variable was maternal age. Adult sex ratio, group size, number of pups at birth and rainfall were fitted as additional explanatory variables and maternal ID, litter and pack were fitted as random variables.

Results

Survival to one year was significantly correlated with group size (GLMM, n=901, β ± s.e = -0.04 ± 0.02, χ2 = 4.24, p = 0.04) (see table 1 and figure 1a) and rainfall in the month pre-birth (GLMM, n=901, β ± s.e = 0.42 ± 0.13, χ2 = 9.87, p = 0.002) (see table 1 and figure 1b). The probability of a pup surviving its first year was higher when pre-birth rainfall was high and when they were born in smaller groups. In our sample, 71% of individuals died during their first year, with females (74%) suffering higher mortality than males (67%) although sex was a marginally non-significant predictor of survival to adulthood (GLMM, n=901, β ± s.e = 0.34 ± 0.18, χ2 = 3.69, p = 0.054) (see table 1). There was no sex-specific effect of maternal age on survival to one year (GLMM, n=901, β ± s.e = 0.0004 ± 0.09, χ2 = 0.0007, p = 1.00).

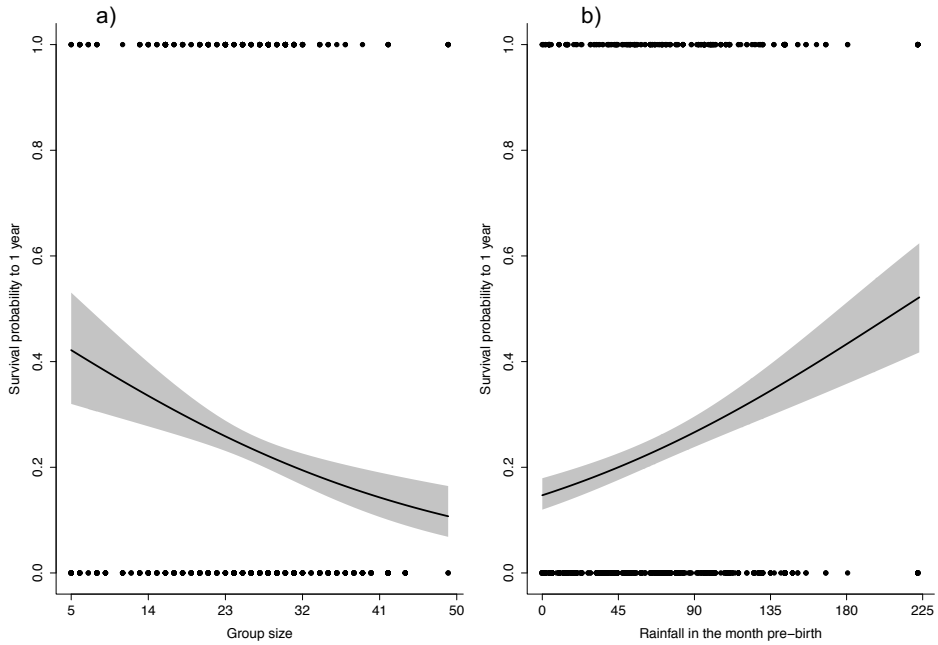

Figure 1. Probability that offspring survived to one year as a function of a) group size and b) rainfall in the month before birth. Both group size and rainfall had significant influences on survival probability Lines show the predicted estimate and shaded areas represent confidence intervals. Each point represents an individual.

Response Variable	Fixed Effect	*β± s.e*	χ2	P	
Survival to 1 year	Intercept	0.25 ± 0.73			
	Maternal age	0.06 ± 0.05	1.33	0.25	
	Pup sex	0.34 ± 0.18	3.71	0.054	.
	Rainfall (standardized)	**0.42 ± 0.13**	**9.87**	**0.002**	**
	Group sex ratio	-1.56 ± 0.99	2.48	0.12	
	Group Size	**-0.04 ± 0.02**	**4.24**	**0.04**	*

	Number of pups	0.01 ± 0.02	0.14	0.71
	Number of observations	Individuals = 901, Packs = 13		

Table 1. Output from a GLMM predicting the effects of early life social and ecological conditions on offspring survival to one year. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors of early life mortality are highlighted in bold.

The mean ± sd adult lifespan was 1236 ± 734 days (range = 367-4512 days) but there was no sex-specific effect of maternal age on lifespan (LMM, n=265, β ± s.e = 0.06 ± 0.03, $\chi 2$ = 2.66, p = 0.10). None of the other tested variables were found to predict lifespan (see table 2).

Response Variable	**Fixed Effect**	***β± s.e***	**$\chi 2$**	**P**
Adult lifespan (log)	Intercept	1.03 ± 0.25		
	Maternal age	0.02 ± 0.02	0.86	035
	Pup sex	-0.02 ± 0.07	0.12	0.73
	Rainfall (standardized)	-0.01 ± 0.01	0.02	0.88
	Group sex ratio	-0.51 ± 0.33	2.29	0.13
	Number of pups	0.01 ± 0.01	0.13	0.72
	Group size	0.01 ± 0.01	0.86	0.35
	Number of observations	Individuals = 265, Packs = 12		

Table 2. Output from a LMM predicting the effects of early life social and ecological conditions on lifespan of all adults that survive to one year. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors of lifespan are highlighted in bold.

Whether or not an individual bred during their lifetime was strongly correlated with sex (GLMM, n=238, β ± s.e = -1.67 ± 0.38, $\chi 2$ = 27.90, p <0.001) but there was no sex-specific effect of maternal age (GLMM, n=238, β ± s.e = -0.006 ± 0.16, $\chi 2$ = 0.002, p = 0.97) (figure 2a). Females (68%) were found to be significantly more likely to successfully breed than males (36%) if they reached adulthood.

Response Variable	**Fixed Effect**	***β± s.e***	**$\chi 2$**	**P**	
Does individual breed	Intercept	0.03 ± 1.09			
	Maternal age	0.12 ± 0.09	1.61	0.20	
	Pup sex	**-1.67 ± 0.38**	**27.90**	**< 0.001**	*******
	Rainfall (standardized)	-0.001 ± 0.17	0.0001	0.99	
	Group sex ratio	-1.45 ± 1.46	1.04	0.31	
	Group Size	0.05 ± 0.03	2.26	0.13	
	Number of pups	-0.02 ± 0.03	0.35	0.55	
	Number of observations	Individuals = 238, Packs = 10			

Table 3. Output from a GLMM predicting the effects of early life social and ecological conditions on whether an individual was assigned as a parent during their lifetime. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors of whether an individual bred are highlighted in bold.

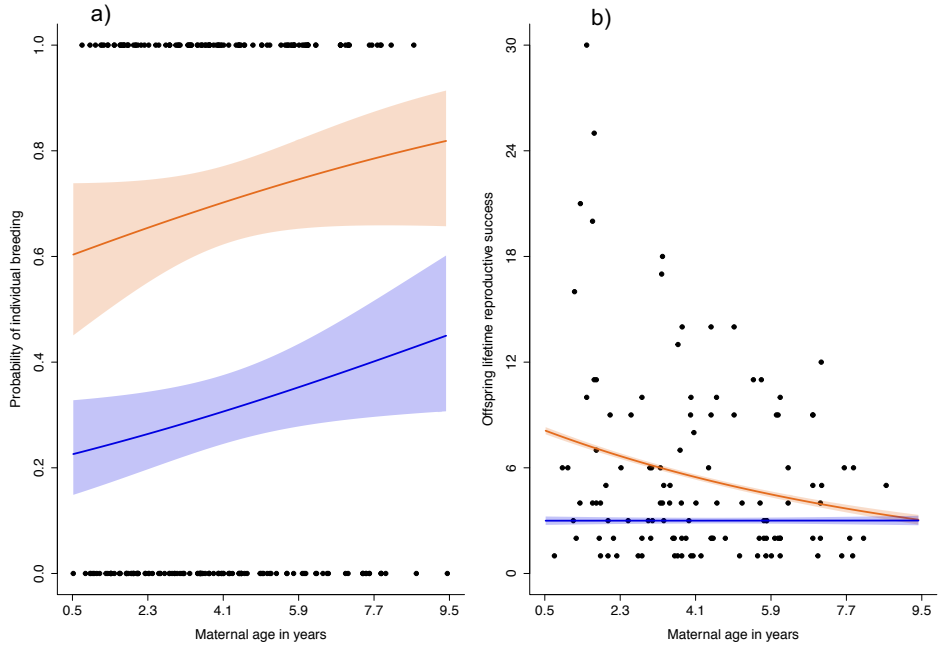

Figure 2. The probability of an individual breeding successfully in their lifetime (panel a) and individual lifetime reproductive success (panel b) as a function of maternal age. The interaction between maternal age and sex is significant in panel b but not in panel a. Lines represent predicted estimates and shaded areas represent confidence intervals. Orange represents females and blue represents males. Each point represents an individual.

The mean ± sd number of pups produced per female over their lifespan was 5.60 ± 5.32 (n=117, range = 1-30). There is a significant sex-specific effect of maternal age on lifetime reproductive success (GLMM, n=117, β ± s.e = 0.110 ± 0.05, $\chi 2$ = 4.44, p = 0.04) (see table 4 and figure 2b). The lifetime reproductive success of female offspring declined with maternal age whereas that of male offspring did not.

Response Variable	Fixed Effect	β± s.e	$\chi 2$	P	
Number of offspring	Intercept	5.445 ± 0.42			
	Maternal age	-0.11 ± 0.04	-	-	
	Pup sex	-1.05 ± 0.24	-	-	
	Rainfall (standardized)	-0.04 ± 0.06	0.36	0.55	
	Group sex ratio	-0.30 ± 0.58	0.27	0.61	
	Group Size	0.003 ± 0.01	0.07	0.79	
	Number of pups	0.01 ± 0.01	0.33	0.57	
	Maternal age * Pup sex	**0.11 ± 0.05**	**4.44**	**0.04**	*
	Number of observations	Individuals = 117, Packs = 8			

Table 4. Output from a GLMM predicting the effects of early life social and ecological conditions on lifetime reproductive success. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors of lifetime reproductive success are highlighted in bold.

This suggests that although females (mean ± sd number of offspring = 5.97 ± 5.89, range=1-30, n = 68) appear to have higher lifetime reproductive success than males (mean ± sd number of offspring = 5.08 ± 4.42, range=1-18, n = 49) this effect depends on maternal age.

Response Variable	Fixed Effect	$\beta \pm s.e$	$\chi 2$	P	
Offspring sex	Intercept	0.10 ± 0.43			
	Maternal age	**0.07 ± 0.04**	**4.13**	**0.04**	*
	Rainfall (standardized)	0.08 ± 0.07	1.44	0.23	
	Group sex ratio	-0.23 ± 0.57	0.16	0.69	
	Number of pups	-0.001 ± 0.01	0.002	0.96	
	Group Size	-0.01 ± 0.01	0.25	0.62	
	Number of observations	Individuals = 901, Packs = 13			

Table 5. Output from a model predicting the effects of early life social and ecological conditions on an individual's sex. Parameter estimates (±standard errors) are shown, along with likelihood-ratio chi-square statistic and p-values. Significant predictors of individual sex are highlighted in bold.

The sex ratio of our sample was 53% male, rising to 59% male for all individuals that survived to one year. The likelihood of an individual being female declined with maternal age (GLMM, n=901, β ± s.e = 0.07 ± 0.04, $\chi 2$ = 4.13, p = 0.04) (see table 5 and figure 3). It should be noted that sample size is large and the effect is marginal so care must be taken drawing conclusions on the biological relevance of this result.

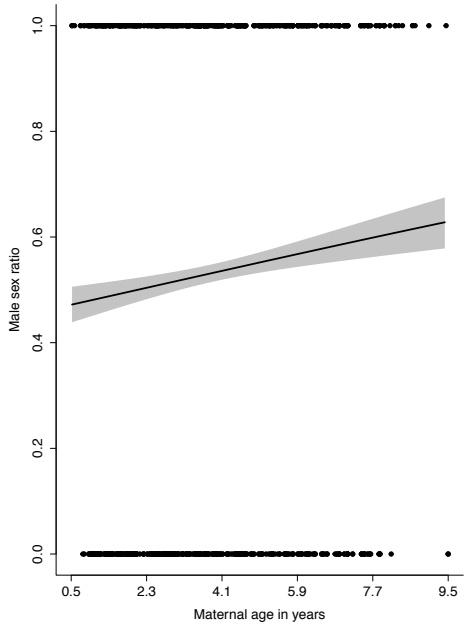

Figure 3. Offspring sex ratio as a function of maternal age showing that younger mothers produce a higher ratio of surviving female offspring than older mothers. The line represents the predicted estimate and the shaded areas show confidence intervals.

Discussion

My results suggest that maternal effects may have sex-specific impacts on offspring fitness. The lifetime reproductive success of daughters declined with maternal age, whereas that of sons did not. There was no evidence of any sex-specific effects of maternal age on survival to maturity, lifespan or whether offspring successfully reproduced. That the daughters of younger mothers show higher lifetime reproductive success supports my initial prediction that the fitness of female pups could be more strongly influenced by maternal age than that of males. This suggests that, despite the reproductive costs suffered by younger mothers, their daughters may sustain a competitive advantage over rivals produced by older females.

In banded mongooses, pre-natal maternal investment is higher in competitive conditions (Inzani et al 2016) when the costs of co-breeding increases (Nichols et al 2012). Theoretical work suggests that a trade-off exists between the size and number of offspring produced by a mother (Lack 1947, Smith & Fretwell 1974). Where maternal age correlates with decreasing resource acquisition, it is predicted that optimal litter size may vary over time (Begon & Parker 1986) due to trade-offs between pup weight and litter size (Hayward et al 2013, Berger et al 2015). The number of foetuses carried by banded mongooses during a breeding attempt increases with maternal age (up to about four years) before senescing in old age (Inzani et al 2016). Therefore, younger mothers may have more resources available to the individual offspring within these smaller litters. There is an association between both female mortality risk (Marshall et al 2016), and reproductive success (Hodge 2005) with individual weight. Pup weight is known to correlate with maternal condition (Hodge 2009) so where maternal investment is associated with traits that favour weight gain it may provide greater fitness benefits to daughters than sons. Future work should attempt to disentangle these effects, in particular to establish whether pup weights decrease with maternal age, as seen in the closely related meerkat (*Suricata* suricata) (Sharp & Clutton-Brock 2009).

That the daughters of younger females have a competitive advantage does not necessarily support the conclusion that optimal allocation strategies change with

maternal age or that female pups suffer greater fitness costs from weak maternal investment than males. The costs suffered by younger mothers in the form of reproductive suppression and competition may impose selection pressures that appear to increase the fitness of their daughters. For example, it might be that only good quality mothers are able to breed at a young age and their offspring are inherently fitter through heritability of female reproductive traits. Similarly, due to younger mothers having higher rates of abortion (Inzani 2016) and being less likely to breed in adverse conditions (Nichols et al 2012) only good quality offspring might survive to reproductive age. Viability selection can lead to sex-specific improvements in performance despite poor early life conditions (Wilkin & Sheldon 2009, Garratt et al 2015). Similarly, studies on both mammals (Weladjii et al 2008), and birds (McCleery et al 2008) suggest that an earlier age at first breeding event is associated with improved performance. Where traits that improve female reproductive success are heritable, the offspring of higher quality females should also show improved individual performance.

Females have high risk of both mortality and eviction during their reproductive lifespan so may benefit from investing heavily in early breeding attempts if conditions are good. Therefore, if traits that benefit female fitness undergo senescent decline, optimal allocation strategies might favour stronger investment in daughters early in maternal reproductive lifespan. Alternatively, younger and older breeders may be following different life-history trajectories which are triggered by early life conditions. Individuals born in poor conditions may breed early to compensate for shortened lifespan (Cartwright et al 2014, Hammers et al 2013) and thus achieve similar reproductive tenure length to those born in relative abundance. Maternal stress during pregnancy is predicted to provide a mechanism for plasticity in life-history trajectories (Berghanel et al 2017). Despite the constraints imposed by high stress levels during late pregnancy, elevated GC concentrations in early pregnancy may be linked to accelerated offspring growth and earlier reproduction at a cost of reduced lifespan (Dmitriew 2011, Schopper et al 2012). Such developmental plasticity seems unlikely here as no significant effect of maternal age rank on GC levels was found prior to or in early pregnancy (Sanderson et al 2015a). In banded mongooses, there may be more scope for plasticity in life-history strategies in males than females due to energetic costs associated with female reproduction (Marshall et al 2017).

This study provides no evidence that maternal age has sex-specific effects on offspring lifespan. This is perhaps unsurprising as longevity is predicted to have significant heritability (Finch & Kirkwood 2000) and be affected by stochastic events such as predation. In banded mongooses, males generally live longer than females (Vitikainen et al 2016) which contrasts with many species, where males have shorter lifespans (Mealey 2000) and higher mortality rates (Clutton-Brock & Isvaren 2007). Here, males but not females, that experience adverse early life conditions live longer at the cost of reduced fertility but not overall reproductive success (Marshall et al 2017). This suggests that males may show greater plasticity in life-history strategies than females, and that any associated trade-offs are mediated by early life conditions. Female reproductive competition might ensure that maternal traits that enhance competitive ability benefit daughters more than sons. Traits that enhance competitive ability are predicted to trade-off against those that increase survival (Clutton-Brock & Isvaran 2007), but here we see no sex-specific effect of maternal age on lifespan. Future work should consider the effect of paternal age on offspring quality to quantify how traits that enhance male fitness may differ from those that enhance female fitness. In banded mongooses a small number of older males generally monopolises paternity within groups (Nichols et al 2010) and longevity may represent a key predictor of male reproductive success. As such, sons sired by older males may receive direct genetic traits that improve longevity from their fathers. In contrast, some studies suggest that sperm quality declines with age (Pizzari et al 2007, Durrant et al 2001) so the effect of male reproductive senescence on offspring performance also merits investigation in this species.

My results provide no evidence of a sex-specific effect of maternal age on offspring survival to maturity, which increased with pre-birth rainfall, and decreased with increasing group size. That low rainfall during pregnancy correlates with low pup survival is consistent with the developmental constraints hypothesis (Monaghan 2008, Lea et al 2015). As rainfall is strongly correlated with food availability, mothers may be energetically constrained by nutritional stress and respond by reducing offspring investment (Bowen 2009, Wells et al 2016), which has been linked with non-adaptive growth plasticity (Berghanel et al 2017). The resulting low birth weight or postnatal body size are strong predictors of reduced early life survival (Lummaa &

Clutton-Brock 2002, Metcalfe & Monaghan 2003). That large group size correlates with higher early life mortality is perhaps more surprising. In cooperative species large group size is often associated with augmentation benefits (Clutton-Brock 2002), including an improved ability to obtain food resources (Creel & Creel 2002) and raise offspring successfully (Clutton-Brock et al 2001). In banded mongooses, there is a strong negative effect of group size on weight and weight gain, which may be linked to a reduction in the size of individual foraging niche (Sheppard et al 2018). In addition, the amount of cooperative care provided by helpers declines with increasing group size so pups in large groups receive less overall care (Vitikainen et al 2017). The reasons why helpers adjust care patterns with group size remains unclear, recruitment may benefit individual carers more in small groups due to potential allee effects (Courchamp et al 1999) and therefore helpers increase investment. In addition, larger packs contain higher levels of intragroup competition so helpers may reduce care to preserve resources for potential dispersal, eviction or reproduction. In banded mongooses, the period between nutritional independence and maturity may be associated with high mortality due to selection pressures associated with competition for resources and independent foraging. Such pressures may increase mortality in individuals of relatively low weight, which is associated with reduced competitive ability and increased mortality, especially in females (Hodge 2005, Marshall et al 2016).

My results suggest that maternal effects can influence offspring sex-ratio, as the proportion of females produced declined with maternal age. It is predicted that mothers in good condition will produce a higher proportion of the sex with higher early life mortality or a stronger association between competitive ability and fitness (Schindler et al 2015, West 2009). The biased sex-ratios here appear to be driven by the higher relative fitness of daughters from young mothers rather than as a response to local resource competition (Clark 1978) or enhancement (Komdeur et al 1997). In banded mongooses, sex-ratio at birth is roughly even (51%, see Cant et al 2016), so there is no evidence for any pre-birth adjustment of sex-ratio towards males as either the more helpful sex, or to minimise local resource competition between mothers daughters. A point of interest here is to what extent the skewing of adult sex-ratios towards males might be influenced by patterns of cooperative care and how they affect infant mortality. The use of maternal “condition” in the Trivers-

Willard hypothesis may refer to the resources available to provide adequate offspring care (Carranza 2002). In banded mongooses, males generally contribute more to pup care but females provide more escorting when they have mothered pups in the litter and allocate care to female pups when there are fewer adult females in the group (Vitikainen et al 2017). Thus sex-biased care patterns may mean that female pups receive less care, and suffer higher mortality, when local resource competition is high. Although there is no evidence that mothers can recognise their own pups, these patterns of care suggest that they may provide more care to their daughters than sons. Therefore, additional care costs may make daughters more expensive to raise than sons and older mothers that lack the resources to provide care may produce sons that are more likely to be cared for by other group members.

It remains unclear whether variation in offspring sex-ratios with age is an adaptive maternal strategy, or the processes by which adjustment can occur (Williams 1979). Recently proposed hormonally-mediated mechanisms of maternal sex-determination may explain why young mothers produce an excess of daughters (Helle et al 2012, Douhard 2017). Maternal testosterone levels may influence the developing oocyte and increase its propensity to be male (Grant & Charnley 2010). In social species with defined rank hierarchies, dominance may be associated with higher testosterone levels. Evidence however is mixed, with some studies reporting that mothers with high testosterone produce male-biased sex-ratios (Grant & Irwin 2005, Helle et al 2008), but others reporting no such association (Diez et al 2009, Garcia-Herreros et al 2010). Banded mongooses form age-based hierarchies within groups but it is unclear whether testosterone levels increase with maternal age. A more likely mechanism for offspring sex-determination is sex-biased embryonic mortality and abortion in response to maternal GC levels (Krackow 1995). Male offspring are generally more susceptible to oxidative stress in-utero than females (Navara 2010, Love et al 2005) and maternal stress has been associated with female-biased sex-ratios (Linklater 2007, Ideta et al 2009). In banded mongooses, younger mothers are more stressed during pregnancy (Sanderson et al 2015a) and more likely to abort their litters (Inzani 2016). Elevated GC levels are associated with a higher risk of abortion in meerkats (Young et al 2006) and here younger mothers may lose litters in response to low rainfall to reduce the threat of eviction from the group. Future research should investigate the possible association between maternal stress and

offspring sex ratios. If found, this would help underpin the observed effects of maternal age on offspring sex ratios with a fuller mechanistic understanding.

In summary, I show that the effect of maternal age on offspring fitness can differ depending on sex. The lifetime reproductive success of female offspring declined with maternal age, whereas that of males did not. There were no sex-specific effects of maternal age on survival to maturity, lifespan or whether offspring reproduced successfully in their lifetime. My results also suggest that sex-specific maternal effects may be associated with age-related offspring sex-ratio adjustment. The proportion of daughters produced decreased with maternal age which may represent a reverse Trivers-Willard effect if maternal condition declines with age and younger mothers allocate more resources to female offspring. This sex-ratio adjustment may be adaptive if daughters receive greater fitness benefits from maternal investment than sons. In this species, female mortality and reproductive success are more closely associated with individual weight than that of males and therefore increasing resources to daughters may benefit maternal fitness. However, this study provides little evidence that this sex-ratio adjustment is adaptive and it may simply represent a mechanistic by-product of the elevated stress experienced by younger mothers during pregnancy.

Chapter 5: Conclusion

This study investigated how conditions during development influenced early life stress hormone levels in banded mongoose pups. The wider contextual aim was to consider how maternal effects might generate individual level variation of phenotypic traits in cooperatively breeding species. I examined how maternal dominance rank, and the amount of alloparental care received, affected offspring glucocorticoid (GC) concentrations. I predicted that, as escorting increases both provisioning and growth rates (Hodge 2005), pups that received less care would have higher GC levels. Given that low ranked mothers show higher stress during pregnancy and lower reproductive success due to suppression by older females (Sanderson et al 2015a), I also predicted that pups from low ranked mothers would have elevated GC levels. Finally, I tested whether early life GC concentrations influence offspring survival, both until nutritional independence at 90 days, and maturity at one year of age.

I found no association between the amount of cooperative care and pup GC levels, therefore no evidence is provided to support my prediction that receiving less care would correlate with higher offspring stress. Despite this it remains a valid prediction that lower care levels and the associated nutritional stress constitute an acute stressor capable of upregulating pup stress response. The lack of effect may be due to the difficulty in disentangling the effects of post-natal cooperative care with those of maternal effects, particularly in systems where care levels depend in part on pre-natal maternal allocation. The evolution of cooperative care has allowed for mothers to effectively decouple their pre and post-natal investment, which may buffer against any developmental constraints linked to maternal stress. In systems where additional carers relieve parents of the burden of exclusive care the presence of helpers may mitigate the transfer of stress from mothers in poor condition to offspring. The “load-lightening” properties of cooperative care have been widely reported (Crick 1992, Hatchwell 1999), and allow for faster recovery of maternal body condition after reproductive events. An ability to minimise reproductive costs may explain why cooperative breeding has evolved in many species that inhabit challenging environments (Lukas & Clutton-Brock 2017, Guindre-Parker & Rubenstein 2018).

Similarly, in systems where maternal care is augmented by helpers, groups may be able to recruit members more rapidly and prosper in competitive environments.

In our system, cooperative care may buffer against maternal stress derived from the social environment, which will vary depending on rank and intragroup competition. The synchronous breeding system limits the effectiveness of infanticide as a means of suppression (Cant 2000). Instead, reproductive competition is controlled by a combination of occasional violent evictions (Thompson et al 2016) and a more covert form of stress-induced suppression (Sanderson et al 2015a), both inflicted by older females. Lower ranked females show higher GC levels during late pregnancy and may suffer greater energetically costs due to their lower body weight and potentially shorter gestation periods (Cant et al 2014). However, if the pups of these stressed mothers are successful at accessing escorts then any maternal constraints may be mitigated. Here, rather than these pups showing developmental constraints we see sex-specific elevated GC concentrations. Although, with the exception of short term pup mortality, the fitness costs or benefits of early life GC remain unclear, I argue that this reflects viability selection resulting from the suppression of lower ranked females. In effect only pups from good quality low ranking females can survive and their daughters tend to have higher GC, which if associated with heavier weight at emergence, may correlate with higher escorting effort received. Although chapter four suggests that the daughters of young mothers have higher reproductive success, further investigation is needed. In particular, future studies should look for links between early life GC levels and adult fitness traits such as longevity or reproductive success, and whether such effects differ between males and females.

Evidence from lab studies suggests that functionality of the stress response in adults is linked to the quality of care received in early life via gene modification at glucocorticoid receptors. Here I find no evidence of any association between pup GC concentrations and early life care. This is perhaps unsurprising given that growth rates represent a major allocation of energy in early life and are expected to influence GC levels. In this case, care is associated with provisioning and growth, which may increase rather than decrease GC concentrations and therefore, any costs linked to poor care will not reflect hormone levels. Faecal GC measures may not be a useful indicator of offspring HPA axis function as they represent a

combination of baseline hormone concentrations and response to acute stressors. As such, future work should look for associations between response to acute stressors (Rich & Romero 2005) and levels of cooperative care rather then consider faecal GC concentrations in pups. This could provide clearer mechanistic understanding of whether development of the neuroendocrine stress response is linked to alloparental care quality in cooperatively breeding species.

Maternal age was found to have a within-individual sex-specific effect on pup stress levels, as mothers grow older the GC concentrations of daughters declines more strongly than in sons. This supports the assertion that female hormone levels are more sensitive to maternally derived effects than males, and thus pup GC levels may have sex-specific fitness consequences. Although this within-individual decline might question the link between viability selection and the sex-specific effect of maternal rank on offspring GC, it may also reflect that subordinate females only breed in more nutritionally optimal conditions. Conversely this could represent age-related changes to maternal condition which influence optimal maternal investment strategies. Due to mortality and reproductive output being more closely linked to weight in females than in males, maternal condition may more strongly effect the fitness of daughters than sons. These effects may be compounded by patterns of escort care if females have to compete more intensely for carers as the care they receive is more strongly linked to weight at emergence than males. As such, any age-related senescence in traits linked to offspring weight may impact the competitive ability of female pups and we might expect younger mothers to direct investment towards their daughters.

Elevated early life GC concentrations were associated with an immediate but not delayed risk of mortality. The probability of dying prior to 90 days was correlated with high GC levels, but the probability of not surviving to maturity was not correlated with GC in the first 90 days. This supports the analysis of the effect of pup age on GC levels suggesting that highly stressed pups are selectively lost from the population during the first 90 days. Any physiological problems associated with early life stress did not appear to increase the risk of death between nutritional independence and maturity. Rather than suggesting that chronically elevated stress is damaging to pups, high GC levels may be a symptom of morbidity, particularly if the cause of death is disease or starvation.

Early life GC concentrations are strongly influenced by both pre-natal social conditions and post-natal food supply. Low rainfall in early life correlates with low pup GC levels, this appear counter-intuitive as low food availability should represent an acute stressor that would increase GC concentrations. Similarly, an increasing number of adult females in the group correlates with low pup GC. As the number of breeding females increases competition within litters should increase, which again may be associated with lower food availability or increased competition for helpers. Alternatively, mothers may take predictive cues about the early life environment from pre-natal social effects and adjust offspring phenotype. Adaptive plasticity in response to stress may allow mothers to prioritise offspring survival by reducing energy expenditure in periods of low food availability. Further investigation here could use within-litter provisioning studies to establish whether GC concentrations reflect food availability or represent a predictive maternal effect. Likewise, examining how patterns of pre-natal competition influence offspring weight and growth rate could highlight in what contexts such effects may become adaptive.

Following the sex-specific effect of maternal rank on pup GC levels, I asked how maternal investment strategies shift with a mother's age and influence offspring development and fitness. I tested whether maternal age had sex-specific effects on offspring survival, lifespan and reproductive success. I predicted that, if maternal condition conferred stronger fitness benefits to one sex, resource allocation might vary with age and skew offspring sex-ratio towards the sex whose fitness was condition-dependent. My results suggest that the effect of maternal age on offspring fitness can differ depending on sex. The lifetime reproductive success of female pups declined with maternal age, whereas that of males did not. There were no sex-specific effects of maternal age on survival, lifespan or whether offspring bred successfully in their lifetime. Although this suggests that daughters of younger females might enjoy an advantage over competitors from older mothers it remains difficult to ascertain whether this is represents adaptive maternal investment. Future work should attempt to link the age-related decline in reproductive success with the sex-specific effect of maternal age on offspring GC. Investigation of pup weights and growth rates might more accurately quantify how optimal resource allocation changes with maternal age.

My results also suggests that maternal effects may be associated with age-related offspring sex-ratio adjustment. The likelihood of an individual being female declined with maternal age, which may represent a reverse Trivers-Willard effect if younger mothers allocate resources to female offspring in response to traits that benefit female fitness undergoing senescent decline. As such, this sex-ratio adjustment may be adaptive if daughters receive greater fitness benefits from maternal investment than sons. Here, female mortality and reproductive success are more closely linked with individual weight than in males and therefore increasing investment to daughters should improve maternal fitness. However, there is little evidence that the sex-ratio adjustment is adaptive and it may simply represent a mechanistic by-product of the elevated stress experienced by younger mothers during pregnancy.

Some plasticity in optimal maternal investment strategies is expected in cooperative breeders due to the complex social system and may be mediated by GC concentrations. Female reproductive opportunities may depend on maternal age or rank as well as ecological and social conditions. For example, intense intergroup competition means that smaller groups are vulnerable to extinction. Therefore, individuals in small groups benefit from rapid recruitment and younger females may be selected to breed when intragroup competition is relatively low. Subsequently, as group size gets larger, local resource competition increases and reproductive suppression by older females may select against younger females reproducing to avoid the threat of eviction. Similarly, as both sexes are largely philopatric, individuals may remain in the natal group for their entire reproductive lifespan and thus be subject to local resource competition from any offspring that they produce.

It remains difficult to establish evidence for the evolution of maternal effects in cooperative breeders using purely correlational data. Particularly when seeking to draw conclusions about the impacts of early life conditions on long term fitness by measuring multifunctional biological markers. In terms of maternal effects, we would expect factors like low maternal rank or adverse ecological conditions to be linked with developmental constraints and elevated GC concentrations. However, our results suggest that viability selection can associate such factors with improved fitness or beneficial phenotypic traits and is likely to account for positive results in correlational studies. In contrast, maternal effects such as physical condition are less

likely to be subject to viability selection and appear more likely to be associated with fitness or developmental costs. In this study, despite there being no direct effect of early life care on offspring GC concentrations the interaction between care availability and maternal effects may prove crucial in estimating how the early life environment may influence offspring fitness. As such, future research should address these questions by stress hormone levels with a phenotypic trait, such as weight at emergence or growth rate, which provides a more tangible link to offspring fitness.

Bibliography

Abbott DH et al. (2003) Are subordinates always stressed? A comparative analysis of rank differences in cortisol levels among primates. *Horm. Behav.* **43**, 67–82.

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

Altmann, J. (1980). *Baboon Mothers and Infants.* Cambridge, Massachussetts: Harvard University Press.

Anacker C., O'Donnell K. J., & Meaney M. J (2014) Early life adversity and the epigenetic programming of hypothalamic-pituitary-adrenal function. *Dialogues Clin. Neurosci.*, 16, pp. 321-333

Andrews C, Nettle D, Larriva M, Gillespie R, Reichert S, Brilot BO, Bedford T, Monaghan P, Spencer KA, Bateson M. (2017) A marker of biological age explains individual variation in the strength of the adult stress response. *R. Soc. open sci.* **4**: 171208. http://dx.doi.org/10.1098/rsos.171208

Arendt, J. D. (1997). Adaptive intrinsic growth rates: an integration across taxa. Q. Rev. Biol. 72, 149-177.

Auer S. K, Dick C. A, Metcalfe N. B., & Reznich D. (2018). Metabolic rate evolves rapidly and in parallel with the pace of life history. *Nature cmmunications.*

Badyaev A.V 2008 Maternal effects as generators of evolutionary change: a reassessment. *Ann. N.Y. Acad. Sci*. 1133, 151–161.

Baker S, Chebli M, Rees S, Lemarec N, Godbout R, Bielajew C. (2008). Effects of gestational stress: 1. Evaluation of maternal and juvenile offspring behavior. *Brain Res* 1213:98–110.

Barker D.J.P. Mothers, babies and disease in later life. In BMJ Publishing Group (1994) London, UK. BMJ Publishing Group

Bates D, Maechler M, Bolker B, Walker S (2015). Fitting Linear Mixed-Effects

Models Using lme4. *J Stat Software* 67: 1–48

Bateson P (2001) Fetal experience and good adult design. *Int. J. Epidemiol*. **30**, 928–934.

Bateson P, Barker D, Clutton-Brock T, Deb D, D'Udine B, Foley RA, Gluckman P, Godfrey K, Kirkwood T, Lahr MM, McNamara J, Metcalfe NB, Monaghan P, Spencer HG & Sultan SE (2004). Developmental plasticity and human health. *Nature* **430**, 419–421.

Bateson P & Gluckman P (2011). *Plasticity, Robustness, Development and Evolution*. Cambridge University Press, Cambridge, UK.

Bateson P, Gluckman P, & Hanson M., (2014). The biology of developmental plasticity and the Predictive Adaptive hypothesis. *The journal of physiology.* 2357-2368.

Bauer C. M., Hayes L. D., Ebensperger L. A., Ramírez-Estrada J., León C., Davis G. T., Romero M. L (2015) Maternal stress and plural breeding with communal care affect development of the endocrine stress response in a wild rodent. *Hormones and Behaviour*. 75, 18-24.

Beamonte-Barrientos, R., Velando, A., Drummond, H. & Torres, R. (2010). Senescence of maternal effects: aging influences egg quality and rearing capacities of a long-lived bird. *American Naturalist* **175**, 469–480

Beckman, K. B., & B. N. Ames. (1998). The free radical theory of aging matures. Physiological Reviews 78:547–581.

Beery AK, Kaufer D. (2015). Stress, social behavior, and resilience: insights from rodents. *Neurobiol Stress* 1:116–27.

Begon, M. & Parker, G. A. (1986). Should egg size and clutch size decrease with age? *Oikos* **47**, 293–302

Belsky J (2012). The development of human reproductive strategies progress and prospects. *Curr Direc Psychol Sci* **21**, 310–316.

Belsky J, Ruttle PL, Boyce WT, Armstrong JM, Essex MJ (2015) Early adversity, elevated stress physiology, accelerated sexual maturation, and poor health in females. *Dev Psychol* 51:816–822.

Bennett NC, Faulkes CG (2000) African Mole-Rats: Ecology and Eusociality (Cambridge Univ Press, Cambridge, UK).

Bernado J. (1996) Maternal effects in animal ecology. *American Zoologist.* 36, 2, 83-105.

C. Bertram, O. Khan, S. Ohri, D.I. Phillips, S.G. Matthews, M.A. Hanson (2008). Transgenerational effects of prenatal nutrient restriction on cardiovascular and HPA function. *J. Physiol*., 586, pp. 2217-2229

Berger, V., Lemaître, J. F., Gaillard, J. M. & Cohas, A. (2015). How do animals optimize the size-number trade-off when aging? Insights from reproductive senescence patterns in marmots. *Ecology* **96**, 46–53.

Berghänel A, Heistermann M, Schülke O, Ostner J (2016) Prenatal stress effects in a wild, long-lived primate: Predictive adaptive responses in an unpredictable environment. *Proc Biol Sci* 283:20161304.

Berghänel A, Heistermann M, Schülke O, Ostner J (2017) Prenatal stress accelerates offspring growth to compensate for reduced maternal investment across mammals. *PNAS*

Bestion E, Teyssier A, Aubret F, Clobert J, Cote J. (2014). Maternal exposure to predator scents: offspring phenotypic adjustment and dispersal. *Proc R Soc Lond B Biol Sci* 281 published online (doi:10.1098/rspb.2014.0701).

Bian J-H, Du S-Y, Wu Y, Cao Y-F, Nie X-H, He H, You Z-B. (2015). Maternal effects and population regulation: maternal density-induced reproduction suppression impairs offspring capacity in response to immediate environment in root voles *Microtus oeconomus*. *J Anim Ecol* 84:326–36.

Birnie, A.K., Taylor, J.H., Cavanaugh, J., French, J.A., (2013). Quality of maternal

and paternal care predicts later stress reactivity in the cooperatively-breeding marmoset (Callithrix geoffroyi). *Psychoneuroendocrinology* 38, 3003–3014.

Biro, P. A, & Stamps, J. A. (2008). Are animal personality traits linked to life-history productivity? *Trends in Ecology & Evolution*, 23, 361–8.

Biro, P. A. & Stamps, J. A. (2010) Do consistent individual differences in metabolic rate promote consistent individual differences in behavior? *Trends Ecol. Evol.* 25, 653–659.

Blount, J. D., Vitikainen, E. I. K., Stott, I. and Cant, M. A. (2015). Oxidative shielding and the cost of reproduction. *Biol. Rev*.

Bogdanova, M. I., Nager, R. & Monaghan, P. (2007). Age of the incubating parents affects nestling survival: an experimental study of the herring gull *Larus argentus*. *Journal of Avian Biology* **38**, 83–93

Bolnick DI, Amarasekare P, Araújo MS, Burger R, Levine JM, Novak M, Rudolf VHW, Schreiber SJ, Urban MC, Vasseur DA (2011) Why intraspecific trait variation matters in community ecology. *Trends in Ecology and Evolution* 26(4): 183–192

Bolund. E, Schielzeth. H., & Forstmeier. W., (2009) Compensatory investment in zebra finches: females lay larger eggs when paired to sexually unattractive males. *Proceedings of the Royal Society B: Biological Sciences*, 276, pp. 707-715

Bonduriansky R., & Chenoweth SF (2009) Intra-locus sexual conflict. *Trends in ecology and evolution*. 24 280-288

Bonier, F., Martin, P.R., Moore, I.T., Wingfield, J.C., (2009). Do baseline glucocorticoids predict fitness? *Trends Ecol. Evol*. 24, 634–642.

Bonier, F., Moore, I. T. & Robertson, R. J. (2011). The stress of parenthood? Increased glucocorticoids in birds with experimentally enlarged broods. *Biol. Lett.* 7, 944-946.

Bourke A.F.G. (1997) Sociality and kin selection in insects. In *Behavioural ecology: an evolutionary approach* Krebs J.R, Davies N.B. pp. 203–227. Eds. Oxford, UK. Blackwell Science.

Bowen WD. (2009) Maternal effects on offspring size and development in pinnipeds. In *Maternal effects in mammals* (eds D Maestripieri, JM Mateo), pp. 104–132. Chicago, IL: University of Chicago Press

Bowers EK, Thompson CF, Sakaluk SK. (2016). Maternal natal environment and breeding territory predict the condition and sex ratio of offspring. *Evol Biol* **44**: 11–20.

Braun K, Champagne FA. (2014). Paternal influences on offspring development: behavioural and epigenetic pathways. *J Neuroendocrinol* 26:697–706.

Breuner CW (2008) Maternal stress, glucocorticoids, and the maternal/fetal match hypothesis. *Hormones and Behavior*, **54**, 485–487.

Bronson SL, Bale TL. (2016). The placenta as a mediator of stress effects on neurodevelopmental reprogramming. *Neuropsychopharmacology* 41:207–18.

Brouwer L, Van de Pol M, Cockburn A. (2014). The role of social environment on parental care: offspring benefit more from the presence of female than male helpers. *J Anim Ecol* 83:491–503.

Brown, G. R. (2001). Sex-biased investment in nonhuman primates: can Trivers & Willard's theory be tested? *Anim. Behav*. 61, 683–694.

Burns SP., Desai M., Cohen RD, Hales CN., Iles RA., Germain JP., Going, TCH & Bailey RA. (1997). Gluconeogenesis, Glucose Handling, and Structural Changes in Livers of the Adult Offspring of Rats Partially Deprived of Protein During Pregnancy and Lactation. J. *Clin. Invest.* 100 (7). 1768-1774.

Burton, C.L., Chatterjee, D., Chatterjee-Chakraborty, M., Lovic, V., Grella, S.L., Steiner, M., Fleming, A.S., (2007). Prenatal restraint stress and motherless rearing disrupts expression of plasticity markers and stress-induced corticosterone release in adult female Sprague–Dawley rats. *Brain Res*. 1158, 28–38.

Burton T, Metcalfe NB. (2014). Can environmental conditions experienced in early life influence future generations? *Proc R Soc Lond B Biol Sci* 281 published online (doi: 10.1098/rspb.2014.0311).

Cant, M. A. (2000). Social control of reproduction in banded mongooses. *Animal Behaviour*, 59, 147– 158.

Cant, M. A., Otali, E., Mwanguhya, F., & Edward, L. (2001). Eviction and dispersal in co-operatively breeding banded mongooses (Mungos mungo). *Journal of the Zoological Society of London*, 254, 155–162.

Cant, M. A. (2003). Patterns of helping effort in co-operatively breeding banded mongooses (Mungos mungo). *Journal of the Zoological Society of London*, 259, 115–121.

Cant MA, Hodge SJ, Bell MBV, Gilchrist JS, Nichols HJ. (2010). Reproductive control via eviction (but not the threat of eviction) in banded mongooses. *Proc. R. Soc. B* **277**, 2219–2226.

Cant, M. A., Vitikainen, E., & Nichols, H. J. (2013). Demography and social evolution of banded mongooses. *Adv. Stud. Behav*. 45, 407-445.

Cant, M. A., Nichols, H. J., Johnstone, R. A., and Hodge, S. J. (2014). Policing of reproduction by hidden threats in a cooperative mammal. *Proc. Natl. Acad. Sci. (USA)*, 111, 326–330.

Cant, M. A., Nichols, H. J., Thompson, F. J., & Vitikainen, E. I. K. (2016). Banded mongooses: demography, life history, and social behavior. In *Cooperative Breeding in Vertebrates*. (eds W.D Koenig & J.L Dickinson) 318-337.

Capelle PM. (2016). Interactive effects of pre- and post-natal stressors on Chinook salmon performance and fitness [dissertation]. University of Windsor.

Carlson, A. A, Manser, M. B., Young, A. J., Russell, A. F., Jordan, N. R., McNeilly, A. S., & Clutton- Brock, T. H. (2006a). Cortisol levels are positively associated with pup-feeding rates in male meerkats. *Proceedings of The Royal Society B*, **273**, 571–7.

Carlson, A. A, Russell, A. F., Young, A. J., Jordan, N. R., McNeilly, A. S., Parlow, A. F., & Clutton- Brock, T. H. (2006b). Elevated prolactin levels immediately precede decisions to babysit by male meerkat helpers. *Hormones and*

Behavior, 50, 94–100.

Carranza J. (2002). What did Trivers and Willard really predict? *Anim Behav* **63**: 1–3.

Carter MJ, Lind MI, Dennis SR, Hentley W, Beckerman AP (2017) Evolution of a predator-induced, non-linear reaction norm. *Proceedings of the Royal Society B.* DOI: 10.1098/rspb.2017.0859

Cartwright, S. J., Nicoll, M. A. C., Jones, C. G., Tatayah, V., & Norris, K. (2014). Anthropogenic natal environmental effects on life histories in a wild bird population. *Current Biology*, *24*, 536–540.

Champagne, F.A., Francis, D.D., Mar, A., Meaney, M.J., (2003). Variations in maternal care in the rat as a mediating influence for the effects of environment on development. *Physiol. Behav*. 79, 359–371.

Champagne FA, Meaney MJ (2007). Transgenerational Effects of Social Environment on Variations in Maternal Care and Behavioral Response to Novelty. *Behav Neurosci*.;121:1353–1363.

Chapman T, Arnqvist G, Bangham J, Rowe L. (2003). Sexual conflict. *Trends Ecol Evol* 18:41–7.

Charlesworth B (2001) Patterns of age-specific means and genetic variances of mortality rates predicted by the mutation-accumulation theory of ageing. *J Theor Biol* 210(1):47–65

Chin E. H., Love O. P., Verspoor J. J., Williams T. D., Rowley K., Burness G. (2009) Juveniles exposed to embryonic corticosterone have enhanced flight performance. *Proc. R. Soc. B* **276**, 499–505.

Clark AB (1978) Sex ratio and local resource competition in a prosimian primate. *Science*, 201. 163-165.

Clutton-Brock TH, Albon SD, Guinness FE. (1981) Parental investment in male and female offspring in polygynous mammals. *Nature* **289**, 487–489.

Clutton-Brock, T. H. (1984). Reproductive effort and terminal investment in iteroparous animals. *American Naturalist* **123**, 212–229.

Clutton-Brock TH, Albon SD, Guinness FE. (1985) Parental investment and sex differences in juvenile mortality in birds and mammals. *Nature* **313**, 131–133.

Clutton-Brock TH, Albon SD, Guinness FE. (1989) Fitness costs of gestation and lactation in wild mammals. *Nature* **337**, 260–262.

Clutton-Brock, T. H., Brotherton, P. N. M., Riain, M. J. O., Griffin, A. S., Gaynor, D., Sharpe, L., Kansky, R., Manser, M. B., & Mcilrath, G. M. (2000). Individual contributions to babysitting in a cooperative mongoose, Suricata suricatta. *Proceedings of The Royal Society B*, **267**, 301–305.

Clutton-Brock, T. H., Brotherton, P. N. M., Russell, A. F., O'Riain, M. J., Gaynor, D., Kansky, R., Griffin, A. S., Manser, M., Sharpe, L., McIlrath, G. M., Small, T., Moss, A. M., Monforts, S. L. (2001). Cooperation, control, and concession in meerkat groups. *Science*, **291**, 478–81.

Clutton-Brock TH, Hodge SJ, Spong G, Russell AF, Jordan NR, Bennett NC, Sharpe LL, Manser MB. (2006) Intrasexual competition and sexual selection in cooperative mammals. *Nature* **444**, 1065–1068.

Clutton-Brock TH & Isvaran K. (2007) Sex differences in ageing in natural populations of vertebrates. *Proc. R. Soc. B* **274**, 3097–3104.

Clutton-Brock TH (2009) Structure and function in mammalian societies. *Philosophical Transactions of the Royal Society of London. Series B*. 364, 3229-3242.

Clutton-Brock TH, Huchard E. (2013) Social competition and selection in males and females. *Phil. Trans. R. Soc. B* **368**, 20130074.

Cockburn A Mating systems and sexual selections. In *Ecology and evolution of cooperative breeding in birds* Koenig W.D, Dickinson J.L 2004pp. 81–101. Eds.

Cambridge, UK. Cambridge University Press.

Cohen, S., Janicki-Deverts, D., Miller, G.E., (2007). Psychological stress and disease. JAMA 298, 1685–1687.

Colman, R. J., Anderson, R. M., Johnson, S. C., Kastman, E. K., Kosmatka, K. J., Beasley, T. M., ... Weindruch, R. (2009). Caloric restriction delays disease onset and mortality in rhesus monkeys. *Science*, *325*, 201–204.

Compagnucci CV., Compagnucci GE., Lomniczi A., Mohn C., Vacas I., Cebral E., Elverdin JC., Friedman SM., Rettori V & Boyer PM. (2002). Effect of Nutritional Stress on the Hypothalamo-Pituitary-Gonadal Axis in the Growing Male Rat. *Neuroimmunomodulation.* 10: 153-162.

Conradt L., Clutton-Brock TH., & Guinness (2000). Sex differences in weather sensitivity can cause habitat segregation: red deer as an example. *Animal Behaviour,* **59,** 1049–1060

Coslovsky M, Richner H. (2011). Predation risk affects offspring growth via maternal effects. *Funct Ecol* 25:878–88.

Cottrell EC, Holmes MC, Livingstone DE, Kenyon CJ, Seckl JR. (2012). Reconciling the nutritional and glucocorticoid hypotheses of fetal programming. *FASEB J* 26:1866–74.

Coulson T., Catchpole EA., Albon SD., Morgan BJT, Pemberton JM., Clutton-Brock TH., Crawley MJ., Grenfell BT (2001). Age, Sex, Density, Winter Weather, and Population Crashes in Soay Sheep. *Science* **292**, 1528.

Courchamp F, Clutton-Brock TH, Grenfell B. (1999). Inverse density dependence and the Allee effect. Trends Ecol Evol. 14:405–410.

Cox RM, Duryea MC, Najarro M, Calsbeek R. (2011). Paternal condition drives progeny sex-ratio bias in a lizard that lacks parental care. *Evolution* **65**: 220–30.

Creel S., Creel N & Monfort SL. (1998) Birth order, estrogen and sex ratio adaptation **in** African wild dogs (*Lycaon pictus*). *Animal Reproduction Science*. 53,

315-320.

Creel S. (2001) Social dominance and stress hormones. *Trends Ecol. Evol.* **16**, 491–497.

Creel S., & Creel N. (2002). The African wild dog: behavior, ecology and conservation. Princeton University Press.

Creel S, Dantzer B, Goymann W, Rubenstein DR. (2013) The ecology of stress: effects of the social environment. *Funct. Ecol.* **27**, 66–80.

Crick HP (1992) Load-lightening in cooperatively breeding birds and the cost of reproduction. *Ibis* **134**, 56–61.

Cunningham E.J.A, Russell A.F (2000) Egg investment is influenced by male attractiveness in mallards. *Nature*. **404**, 74–77.

Curio E (1983) Why do young birds reproduce less well? *Ibis* **125**(3):400–404

Dalmaz C., Noschang C., Krolow R., Raineki C., Lucion A. B., (2015) How postnatal insults may program development: studies in animal models. *Adv. Neurobiol*., 10, pp. 121-147

Dantzer, B., Newman, A. E. M., Boonstra, R., Palme, R., Boutin, S., Humphries, M. M. (2013). Density Triggers Maternal Hormones That Increase Adaptive Offspring Growth in a Wild Mammal. Science 340, 1215-1217.

Dantzer B, Bennett NC, Clutton-Brock TH. (2017) Social conflict and costs of cooperation in meerkats are reflected in measures of stress hormones. *Behav. Ecol.* **28**, 1131–1141.

Darwin C. (1859) *On the origin of species by means of natural selection*. London, UK: John Murray

Daunt F., Wanless S., Harris MP., & Monaghan P (1999). Experimental evidence that age-specific reproductive success is independent of environmental effects. *Proc.*

R. Soc. Lond. B. 266, 1489-1493

de Kloet E. R., Sibug R.M., Helmerhorst F. M., Schmidt M (2005) Stress, genes and the mechanism of programming the brain for later life. *Neurosci. Biobehav. Rev.*, 29, pp. 271-281

Del Giudice M (2014). Life history plasticity in humans: the predictive value of early cues depends on the temporal structure of the environment. *Proc R Soc B* **281**, 20132995.

Descamps S, Boutin S, Berteaux D, Gaillard J.-M. (2008) Age-specific variation in survival, reproductive success and offspring quality in red squirrels: evidence of senescence. *Oikos*. **117**, 1406–1416.

DeWitt TJ, Sih A, Wilson DS. (1998). Costs and limits of phenotypic plasticity. *Trends Ecol Evol* 13:77–81.

Dias BG, Ressier KJ. (2014). Parental olfactory experience influences behavior and neural structure in subsequent generations. *Nat Neurosci* 17:89–96.

Díez C, Bermejo-Alvarez P, Trigal B, Caamaño JN, et al. (2009). Changes in testosterone of temperature during the in vitro oocyte culture do not alter the sex ratio of bovine embryos. *J Exp Zool* **311A**: 448–52.

Dloniak, S. M., French, J. A. & Holekamp, K. E. (2006). Rank-related maternal effects of androgens on behaviour in wild spotted hyenas. Nature. 440 (7088), 1190-1193.

Dmitriew, C. M. (2011). The evolution of growth trajectories what limits growth rate? *Biological Reviews* **86**, 97–116.

Douhard, M. (2017). Offspring sex ratio in mammals and the Trivers-Willard hypothesis: In pursuit of unambiguous evidence. *Bioassays*. 39, 9

Dozier, M., Peloso, E., Lewis, E., Laurenceau, J., Levine, S., (2008). Effects of an attachment- based intervention on the cortisol production of infants and toddlers in foster care. *Dev. Psychopathol*. 20, 845–859.

Drury S. S., Sánchez M. M., Gonzalez A (2016) When mothering goes awry: Challenges and opportunities for utilizing evidence across rodent, nonhuman primate and human studies to better define the biological consequences of negative early caregiving. *Hormones and Behaviour.* 77, 182-192.

Dupoue A., Angelier F., Brischoux F., DeNardo D. F., Trouve C., Parenteau C., & Lourdais O. (2016) Water deprivation increases maternal corticosterone levels and enhances offspring growth in the snake *Vipera aspis*. *Journal of experimental Biology*. 219, 658-667.

Durrant, B. S., Millard, S. E., Zimmerman, D. M. & Lindburg, D. G. (2001). Lifetime semen production in a cheetah (*Acinonyx jubatus*). *Zoo Biology* **20**, 359–366.

Ebrahim S, Kinra S, Bowen L, Andersen E, Ben-Shlomo Y, Lyngdoh T, Ramakrishnan L, Ahuja RC, Joshi P, Das SM, Mohan M, Davey Smith G, Prabhakaran D, Reddy KS, Indian Migration Study Group (2010). The effect of rural-to-urban migration on obesity and diabetes in India: a cross-sectional study. *PLoS Med* **7**, e1000268.

Ellenberg U., Setiawan AN., Cree A., Houston DM & Seddon PJ. (2009) Elevated hormonal stress response and reduced reproductive output in Yellow-eyed penguins exposed to unregulated tourism. *General and Comparative Endocrinology.* 152. 54-63.

Emack J, Kostaki A, Walker C-D, Matthews SG. (2008) Chronic maternal stress affects growth, behaviour and hypothalamo- pituitary adrenal function in juvenile offspring. *Horm. Behav*. **54**, 514–520

Emlen, S. T., Emlen, J. M., & Levin, S. A. (1986). Sex ratio selection in species with helpers-at-the-nest. The American Naturalist, 127, 1-8.

Engqvist, L. (2005). The mistreatment of covariate interaction terms in lin- ear model analyses of behavioural and evolutionary ecology studies. *Animal Behaviour*, *70*, 967–971.

English S. & Uller T (2016) Does early-life diet affect longevity? A meta-analysis across experimental studies. *Biology letters*. DOI: 10.1098/rsbl.2016.0291

Epel ES, Blackburn EH, Lin J, Dhabhar FS, Adler NE, Morrow JD, Cawthon RM. (2004) Accelerated telomere shortening in response to life stress. *Proc. Natl Acad. Sci. USA* **101**, 17 312–17 315.

Escribano-Avila G, Pettorelli N, Virgós E, Lara-Romero C, Lozano J, Barja I, Cuadra FS, Puerta M. (2013) Testing cort-fitness and cort-adaptation hypotheses in a habitat suitability gradient for roe deer, Acta Oecologica , vol. 53 (pg. 38-48)

Evans JP, Lymbery RA, Wiid KS, Rahman MM, Gasparini C. (2017). Sperm as moderators of environmentally induced paternal effects in a livebearing fish. *Biol Lett* 13:20170087.

Faulkes C.G, Bennett N.C (2001) Family values: group dynamics and social control of reproduction in African mole-rats. *Trends. Ecol. Evol*. **16**, 184–190.

Fay R., Barbraud C., Delord K., & Weimerskirch (2017) From early life to senescence: individual heterogeneity in a long-lived seabird. *Ecological Monographs*. DOI: 10.1002/ecm.1275

Feder A., Nestler EJ., Charney DS. (2009). Psychobiology and molecular genetics of resilience. *Nat Rev Neurosci* 10(6): 446–457.

Ferrari MCO, Gonzalo A, Messier F, Chivers DP. (2007). Generalization of learned predator recognition: an experimental test and framework for future studies. *Proc R Soc Lond B Biol Sci* 274:1853–9.

Fisher, R. The Genetical Theory of Natural Selection (Oxford University Press, 1930).

Fisher, P., Stoolmiller, M., Gunnar, M., Burraston, B., (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology* 32, 892–905.

Finch CE., & Kirkwood TBL (2000). *Chance, development and aging*. Oxford, UK: Oxford University Press

Finkel, T. & Holbrook, N. J. (2000) Oxidants, oxidative stress and the biology of ageing. Nature 408, 239–247.

Forstmeier, W., & Schielzeth, H. (2011). Cryptic multiple hypotheses test- ing in linear models: Overestimated effect sizes and the winner's curse. *Behavioral Ecology and Sociobiology*, *65*, 47–55.

Francis D, Diorio J, Liu D, Meaney MJ. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science* 286:1155–8.

Froy H, Phillips RA, Wood AG, Nussey DH, Lewis S. (2013) Age-related variation in reproductive traits in the wandering albatross: evidence for terminal improvement following senescence. *Ecol. Lett.* **16**, 642–649.

García-Herreros M, Bermejo-Álvarez P, Rizos D, Gutiérrez-Adán A, et al. (2010). Intrafollicular testosterone concentration and sex ratio in individually cultured bovine embryos. *Reprod Fertil Dev* **22**: 533–8.

Garratt, M., Lemaître, J. F., Douhard, M., Bonenfant, C., Capron, G., Warnant, C., ... Gaillard, J. M. (2015). High juvenile mortality is associated with sex-specific adult survival and lifespan in wild roe deer. *Current Biology*, *25*, 759–763.

Giesing ER, Suski CD, Warner RE, Bell AM. (2011). Female sticklebacks transfer information via eggs: effects of maternal experience with predators on offspring. *Proceedings of the Royal Society of London, Series B* 278:1753–9.

Gilchrist, J. S. 2004. Pup escorting in the communal breeding banded mongoose: behavior, benefits, and maintenance. *Behavioral Ecology*, **15**, 952–960.

Gilchrist, J. S. (2006). Female eviction, abortion, and infanticide in banded mongooses (*Mungos mungo*): implications for social control of reproduction and synchronized parturition. *Behav. Ecol.*, 17, 664–669.

Gilchrist, J. S., & Russell, A. F. 2007. Who cares? Individual contributions to pup care by breeders vs non-breeders in the cooperatively breeding banded mongoose (Mungos mungo). *Behavioral Ecology and Sociobiology*, **61**, 1053–1060.

Gilchrist, J. S., Otali, E., & Mwanguhya, F. (2008). Caregivers recognize and bias response towards individual young in a cooperative breeding mammal, the banded mongoose. *J. Zoo*., 275(1), 41-46.

Glazier, D. S. (2015) Is metabolic rate a universal 'pacemaker' for biological processes? *Biol. Rev*. 90, 377–407.

Gluckman, P. D., & Hanson, M. A. (2004). Living with the past: evolution, development, and patterns of disease. *Science*. 305(5691), 1733-1736.

Gluckman, P. D., Hanson, M. A., & Spencer, H. G. (2005a). Predictive adaptive responses and human evolution. *Trends in Ecology & Evolution*, *20*, 527–533.

Gluckman PD, Hanson MA, Spencer HG, Bateson P (2005b) Environmental influences during development and their later consequences for health and disease: implications for the interpretation on empirical studies. *Proceedings of the Royal Society of London B*, **272**, 671–677.

Godfrey KM (2006). The "developmental origins" hypothesis: epidemiology. In *Developmental Origins of Health and Disease*, eds Gluckman PD & Hanson MA, pp. 6–32. Cambridge University Press, Cambridge, UK.

Godfrey KM, Lillycrop KA, Burdge GC, Gluckman PD & Hanson MA (2007). Epigenetic mechanisms and the mismatch concept of the developmental origins of health and disease. *Pediatr Res* **61**, 5R–10R

Godfrey KM, Gluckman PD & Hanson MA (2010). Developmental origins of metabolic disease: life course and intergenerational perspectives. *Trends Endocrinol Metabol* **21**, 199–205

Goymann W, Wingfield JC (2004) Allostatic load, social status and stress hormones: the costs of social status matter. *Animal Behaviour*, **67**, 591–602.

Grafen A. On the uses of data on lifetime reproductive success. In: Clutton-Brock

TH, editor. Reproductive success. University of Chicago Press; Chicago: 1988. pp. 454–47.

Grant VJ, Chamley LW. (2010). Can mammalian mothers influence the sex of their offspring peri-conceptually? *Reproduction* **140**: 425–33.

Grant VJ, Irwin RJ. (2005). Follicular fluid steroid levels and subsequent sex of bovine embryos. *J Exp Zool* **303**: 1120–25.

Griffin AS, Evans CS, Blumstein DT. (2001). Learning specificity in acquired predator recognition. *Anim Behav* 62:577–89.

Griffin, A. S., & West, S. A. 2003. Kin discrimination and the benefit of helping in cooperatively breeding vertebrates. *Science*, **302**, 634–636.

Griffin AS, Sheldon BC, West SA. (2005) Cooperative breeders adjust offspring sex ratios to produce helpful helpers. *Am. Nat.* **166**, 628–632

Groothuis T. G. G., Muller W., von Engelhardt N., Carere C., Eising C. (2005) Maternal hormones as a tool to adjust offspring phenotype in avian species. *Neurosci. Biobehav. Rev.* **29**, 329–352.

Guindre-Parker S, Rubenstein DR. (2018) Multiple benefits of alloparental care in a fluctuating environment. *R. Soc. open sci.* **5**: 172406.

Gunnar, M.R, Hostinar, C., Sanchez, M., Tottenham, N., Sullivan, R., (2015). Parental buffering of fear and stress neurobiolog reviewing parallels across rodent, NHP and human models. *Soc. Neurosci*. 1–5.

Haase C. G., Long A. K., & Gillooly J. F (2016) Energetics of stress: linking plasma cortisol levels to metabolic rate in mammals. *Biology letters*. 12 20150867.

Hackländer K, Möstl E, Arnold W. (2003) Reproductive suppression in female alpine marmots, *Marmota marmot*. *Anim. Behav.* **65**, 1133–1140.

Hadfield JD, Richardson DS, Burke T. (2006) Towards unbiased parentage

assignment: combining genetic, behavioural and spatial data in a Bayesian framework. *Mol. Ecol.* **15**, 3715–3730.

Hales CN (1997) Metabolic consequences of intrauterine growth retardation. *Acta Paediatrica*. 86, 184-187.

Hales CN, Barker DJ. The thrifty phenotype hypothesis. British Medical Bulletin. (2001). 60:5–20.

Hales C.N, Barker D.J.P (1992) Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia*. **35**, 595–601.

Hamilton WD. (1966) The moulding of senescence by natural selection. *J. Theor. Biol.* **12**, 12–45.

Hammers, M., Richardson, D. S., Burke, T., & Komdeur, J. (2013). The impact of reproductive investment and early-life environmental conditions on senescence: Support for the disposable soma hypothesis. *Journal of Evolutionary Biology*, *26*, 1999–2007.

Hanson MA, & Gluckman PD (2014) Early developmental conditioning of later health and disease: Physiology or pathophysiology? *Physiol Rev* 94:1027–1076.

Harris A., & Seckl J., (2011) Glucocorticoids, prenatal stress and the programming of disease. *Horm. Behav.* **59**, 279.

Hatchwell BJ. (1999) Investment strategies of breeders in avian cooperative breeding systems. *Am. Nat.* **154**, 205–219

Hatchwell BJ. (2009). The evolution of cooperative breeding in birds: kinship, dispersal and life history*. Phil. Trans. R. Soc. B* **364**, 3217–3227

Hayward LS, & Wingfield JC (2004) Maternal corticosterone is transferred to avian yolk and may alter offspring growth and adult phenotype. *General and Comparative Endocrinology*, **135**, 365–371.

Hayward, A. D., Wilson, A. J., Pilkington, J. G., Clutton-brock, T. H., Pemberton,

J. M. & Kruuk, L. E. B. (2013). Reproductive senescence in female Soay sheep: variation across traits and contributions of individual ageing and selective disappearance. *Functional Ecology* **27**, 184–195.

Heim C.M., Nemeroff C. B (2001) The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies *Biol. Psychiatry*, 49 pp. 1023-1039

Helle S, Laaksonen T, Adamsson A, Paranko J, et al. (2008). Female field voles with high testosterone and glucose levels produce male-biased litters. *Anim Behav* **75**: 1031–9.

Helle S, Laaksonen T, Huitu O. (2013). Sex-specific offspring growth according to maternal testosterone, corticosterone, and glucose levels. *Behav Ecol* **24**: 205–12.

Hewison, A. J. M. & Gaillard, J.-M. (1999) Successful sons or advantaged daughters? The Trivers–Willard model and sex-biased maternal investment in ungulates. *Trends Ecol. Evol*. 14, 229–234.

Hinde CA. (2006). Negotiation over offspring care? A positive response to partner-provisioning rate in great tits. *Behav Ecol* 17:6–12.

Hinde K, & Milligan LA. (2011) Primate milk: proximate mechanisms and ultimate perspectives. *Evol. Anthropol*. **20**, 9–23

Hodge, S. J. (2005). Helpers benefit offspring in both the short and long-term in the cooperatively breeding banded mongoose. *Proceedings of The Royal Society B,* **272**, 2479–84.

Hodge, S. J. (2007). Counting the costs: the evolution of male-biased care in the cooperatively breeding banded mongoose. *Animal Behaviour*, **74**, 911–919.

Hodge, S. J., Bell, M. B., Mwanguhya, F., Kyabulima, S., Waldick, R. C., & Russell, A. F. (2009). Maternal weight, offspring competitive ability, and the evolution of communal breeding. Behav. Ecol. 20, 729-735.

Hodge, S. J., Bell, M. B. V, & Cant, M. A. 2011. Reproductive competition and the

evolution of extreme birth synchrony in a cooperative mammal. *Biology Letters*, 7, 54–6.

Hoffman JI, Amos W (2005) Microsatellite genotyping errors: detection approaches, common sources and consequences for paternal exclusion. *Molecular Ecology*, 14(2), 599–612.

Hostinar, C.E., Sullivan, R.M., Gunnar, M.R., (2014). Psychobiological mechanisms underlying the social buffering of the HPA axis: a review of animal models and human studies across development. Psychol. Bull. 140, 256–282.

Huchard E, English S, Bell MBV, Thavarajah N, Clutton-Brock T. (2016) Competitive growth in a cooperative mammal. *Nature* **533**, 532–534

Ideta A, Hayama K, Kawashima C, Urakawa M, et al. (2009). Subjecting Holstein heifers to stress during the follicular phase following super-ovulatory treatment may increase the female sex ratio of embryos. *J Reprod Dev* **55**: 529–33.

Inzani, E.L. (2016) Female reproductive conflict and prenatal investment. Masters by Research Thesis. University of Exeter.

I**nzani, E. L., Marshall, H. H., Sanderson, J. L., Nichols, H. J., Thompson, F. J., Kalema-Zikusoka, G., Hodge, S.J., Cant M.A. & Vitikainen, E. I**. (2016). Female reproductive competition explains variation in prenatal investment in wild banded mongooses. *Scientific reports*. 6. 20013

Isaac J (2005) Potential causes and life-history consequences of sexual size dimorphism in mammals. *Mammal Review*. 35, 101-115.

Jones O. R & Wang J. (2010) COLONY: a program for parentage and sibship inference from multilocus genotype data. *Mol. Ecol. Resour.* **10**, 551–555.

Jablonka E, Oborny B, Molnar I, Kisdi E, Hofbauer J, Czaran T. (1995). The adaptive advantage of phenotypic memory in changing environments. *Philos Trans R Soc Lond B Biol Sci* 350:133–41.

Jablonka E & Raz G (2009). Transgenerational epigenetic inheritance: prevalence,

mechanisms, and implications for the study of heredity and evolution. *Q Rev Biol* **84**, 131–176

Jenkins, B. R., Vitousek, M. N., Hubbard J. K., Safran R. J (2014) An experimental analysis of the heritability of variation in glucocorticoid concentrations in a wild avian population. *Proc. R. Soc. B Biol. Sci*., 281 (2014), p. 2014130

Jones AP & Friedman MI (1982). Obesity and adipocyte abnormalities in offspring of rats undernourished during pregnancy. *Science* **215**, 1518–1519.

Johnstone RA, Hinde CA. (2006). Negotiation over offspring care—how should parents respond to each other's efforts? *Behav Ecol* 17:818–27.

Jordan, N. R., Manser, M. B., Mwanguhya, F., Kyabulima, S., Rüedi, P., & Cant, M. A. 2011. Scent marking in wild banded mongooses: 1. Sex-specific scents and overmarking. *Animal Behaviour*, 81, 31–42.

Jordan, N. R., Mwanguhya, F., Kyabulima, S., Ruedi, P., & Cant, M. A. 2010. Scent marking within and between groups of wild banded mongooses. *Journal of Zoology*, 280, 72–83.

Kalinowski ST, Taper ML, Marshall TC (2007) Revising how the computer program CERVUS accommodates genotyping error increases success in paternity assignment. *Molecular Ecology,* 16, 1099–1106.

Kapoor A, Matthews SG. (2005). Short periods of prenatal stress affect growth, behaviour and HPA axis activity in male guinea pig offspring. *J Physiol* 566:967–77.

Kapoor A, Kostaki A, Janus C, Matthews SG. (2009). The effects of prenatal stress on learning in adult offspring is dependent on the timing of the stressor. *Behav Brain Res* 197:144–9.

Kirkwood T. (1977). Evolution of ageing. *Nature* **270**, 301–304.

Keane B, Waser PM, Creel SR, Creel NM, Elliott LF, Minchella DJ. (1994). Subordinate reproduction in dwarf mongooses. *Anim Behav*. 47:65–75.

Keller L & Genoud M (1997) Extraordinary lifespans in ants: a test of evolutionary

theories of ageing. *Nature*. **389**, 958–960.

Kitaysky, A.S., Wingfield, J.C. & Piatt, J.F. (1999). Dynamics of food availability, body condition and physiological stress response in breeding black-legged kittiwakes. *Funct. Ecol*., 13, 577–584

Klaus T, Schoepper H, Huber S. (2013) Effects of chronic stress during pregnancy on maternal performance in the guinea pig (*Cavia aperea f. porcellus*). *Behav. Process*. **94**, 83–88.

Koenig A (1995) Group size, composition, and reproductive success in wild common marmosets (*Callithrix jacchus*). *Am. J. Primatol*. **35**, 311–317

Koenig WD, Dickinson JL, Emlen ST. (2016) Synthesis: cooperative breeding in the twenty-first century. In *Cooperative breeding in vertebrates* (eds WD Koenig, JL Dickinson). Cambridge, UK: Cambridge University Press.

Knapp, R., & Moore, M. C. 1997. Male morphs in tree lizards have different testosterone responses to elevated levels of corticosterone. *General and Comparative Endocrinology*, **107**, 273–9.

Komdeur, J., Daan, S., Tinbergen, J., & Mateman, C. (1997). Extreme adaptive modification in sex ratio of the Seychelles warbler's eggs. Nature, 385.

Krackow S. (1995). Potential mechanisms for sex ratio adjustment in mammals and birds. *Biol Rev* **70**: 225–41.

Kruuk, L. E., Clutton-Brock, T. H., Rose, K. E., & Guinness, F. E. (1999). Early determinants of lifetime reproductive success differ between the sexes in red deer. *Proceedings of the Royal Society of London B: Biological Sciences*, *266*, 1655–1661.

Kuijper, B., & Hoyle, R. B. (2015). When to rely on maternal effects and when on phenotypic plasticity? *Evolution*, *69*, 950–968.

Lack D (1947). The significance of clutch size. *Ibis*. **89**, 302–352.

Ladd, C.O., Huot, R.L., Thrivikraman, K.V., Nemeroff, C.B., Plotsky, P.M., (2004).

Long-term adaptations in glucocorticoid receptor and mineralocorticoid receptor mRNA and negative feedback on the HPA axis following neonatal maternal separation. *Biol. Psychiatry* 55, 367–375.

Landys, M. M., Ramenofsky, M., & Wingfield, J. C. 2006. Actions of glucocorticoids at a seasonal baseline as compared to stress-related levels in the regulation of periodic life processes. *General and Comparative Endocrinology*, **148**, 132–49.

Laforsch C, Beccara L & Tollrian R (2006). Inducible defenses: the relevance of chemical alarm cues in *Daphnia*. *Limnol Oceanogr* **51**, 1466–1472.

Lea AJ, Altmann J, Alberts SC, Tung J (2015) Developmental constraints in a wild primate. *The American Naturalist*, **185**, 809–821.

Lee TM & Zucker I (1988). Vole infant development is influenced perinatally by maternal photoperiodic history. *Am J Physiol Regul Integr Comp Physiol* **255**, R831–R838.

Leimar,O. (1996) Life-history analysis of the Trivers and Willard sex-ratio problem. *Behav. Ecol*. 7, 316–325.

Leimar O, McNamara JM. (2015). The evolution of transgenerational integration of information in heterogeneous environments. *Am Nat* 185:E55–69.

Lightman S.L (2008) The neuroendocrinology of stress: a never ending story. *J. Neuroendocrinol*., 20, pp. 880-884

Lindström J (1999) Early development and fitness in birds and mammals. *Trends Ecol. Evol*. **14**, 343–348.

Linklater WL. (2007). Translocation reverses birth sex ratio bias depending on its timing during gestation: evidence for the action of two sex-allocation mechanisms. *Reprod Fertil Dev* **19**: 831–39.

Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Perason, D., Plotsky, P., & Meaney, M. 1997. Maternal care,

hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science*, **277**, 1659–1662.

Liu D, Caldji C, Sharma S, Plotsky PM, Meaney MJ (2000). Influence of neonatal rearing conditions on stress-induced adrenocorticotropin responses and norepinepherine release in the hypothalamic paraventricular nucleus. J Neuroendocrinoly;12: 5–12.

Lesage J, Blondeau B, Grino M, Bréant B, Dupouy JP. (2001). Maternal undernutrition during late gestation induces fetal overexposure to glucocorticoids and intrauterine growth retardation, and disturbs the hypothalamo-pituitary adrenal axis in the newborn rat. *Endocrinology* 142:1692–702.

Levins R. (1968). *Evolution in changing environments: some theoretical explorations*. Princeton (NJ): Princeton University Press.

Lillycrop, KA. Phillips, ES. Jackson, AA. Hanson, MA. Burdge GC (2005) Dietary protein restriction of pregnant rats induces and folic acid supplementation prevents epigenetic modification of hepatic gene expression in the offspring *The Journal of Nutrition*, 135 (6), p. 1382

Love OP, Chin EH, Wynne-Edwards KE, Williams TD. (2005). Stress hormones: a link between maternal condition and sex-biased reproductive investment. *Am Nat* 166:751–66.

Love OP, McGowan PO, Sheriff MJ. (2013). Maternal adversity and ecological stressors in natural populations: the role of stress axis programming in individuals, with implications for populations and communities. *Funct Ecol* 27:81–92.

Love, O. P., Madliger, C. L., Bourgeon, S., Semeniuk, C. A. D. and Williams, T. D. (2014). Evidence for baseline glucocorticoids as mediators of reproductive investment in a wild bird. *Gen. Comp. Endocrinol*. 199, 65-69.

Love OP & Williams T (2008) The Adaptive Value of Stress-Induced Phenotypes: Effects of Maternally Derived Corticosterone on Sex- Biased Investment, Cost of Reproduction, and Maternal Fitness. *The American Naturalist.* 172, 4

Lucassen PJ, Bosch OJ, Jousma E, Kramer SA, Andrew R, Seckl JR, Neumann ID. (2009). Prenatal stress reduces postnatal neurogenesis in rats selectively bred for high, but not low, anxiety: possible key role of placental 11b-hydroxysteroid dehydrogenase type 2. *Eur J Neurosci* 29:97–103.

Lummaa V, & Clutton-Brock T (2002) Early development, survival and reproduction in humans. *Trends Ecol Evol* 17:141–147.

MacColl A. D. C (2011) The ecological causes of evolution. *Trends Ecol. Evol.* **26**, 514.

Mahy M (2003) Childhood Mortality in the Developing World. A Review of Evidence from the Demographic and Health Surveys (ORC Macro, Calverton, MD).

Marshall D.J, & Uller T (2007) When is a maternal effect adaptive?. *Oikos*. **116**, 1957–1963.

Marshall, H. H., Sanderson, J. L., Mwanghuya, F., Businge, R., Kyabulima, S., Hares, M. C., ... Cant, M. A. (2016). Variable ecological conditions promote male helping by changing banded mongoose group composition. *Behavioral Ecology*, *27*, 978–987.

Marshall HH, Vitikainen EIK, Mwanguhya F, Businge R, Kyabulima S, Hares MC, et al. (2017) Lifetime fitness consequences of early-life ecological hardship in a wild mammal population. *Ecol Evol*; 7: 1712– 1724.

Masoro, E. J. (2006). Dietary restriction-induced life extension: A broadly based biological phenomenon. *Biogerontology*, *7*, 153–155.

Mateo JM (2007). Ecological and hormonal correlates of antipredator behavior in adult Belding's ground squirrels (*Spermophilus beldingi*). *Behav Ecol Sociobiol* **62**, 37–49

Matthews, S.G & Phillips, D. I (2012) Transgenerational inheritance of stress pathology *Exp. Neurol*., 233, pp. 95-101

Mathis A, Ferrari MC, Windel N, Messier F & Chivers DP (2008). Learning by embryos and the ghost of predation future. *Proc R Soc B* **275**, 2603–2607.

Matthews SG. (2002). Early programming of the hypothalamo–pituitary–adrenal axis. *Trends Endocrinol Metab* 13:373–80.

Mayr, E. 1974. Teleological and teleonomic: A new analysis. *Boston Studies in the Philosophy of Science*, **14**, 91–117.

McAdam A. G., & Boutin. S., (2003) Variation in viability selection among cohorts of juvenile red squirrels (*Tamiasciurus hudsonicus*). *Evolution* **57**, 1689.

McCleery, R., C. Perrins, B. Sheldon, and A. Charmantier. (2008). Age-specific reproduction in a long-lived species: the combined effects of senescence and individual quality. *Proceedings of the Royal Society B* **275**:963–970.

McCormick C. M & Green M. R (2013) From the stressed adolescent to the anxious and depressed adult: investigations in rodent models Neuroscience, 249, pp. 242-257

McEwen B. S. (1998) Stress, adaptation, and disease: allostasis and allostatic load. In *Neuroimmunomodulation: molecular aspects, integrative systems, and clinical advances*, **vol. 840** (eds McCann S. M., Lipton J. M., Sternberg E. M., Chrousos G. P., Gold P. W., Smith C. C.), pp. 33–44. New York, NY: New York Academy of Sciences

McEwen, B. S., & Wingfield, J. C. 2003. The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, **43**, 2–15.

McEwen, B. S. and Wingfield, J. C. (2010). What's in a name? Integrating homeostasis, allostasis and stress. *Horm. Behav*. 57, 105-111.

McGhee KE, Pintor LM, Suhr EL, Bell AM. (2012). Maternal exposure to predation risk decreases offspring antipredator behaviour and survival in three-spined stickleback. *Funct Ecol* 26:932–40.

MacLeod, K. J., & Clutton-Brock, T. H. (2013). No evidence for adaptive sex ratio variation in the cooperatively breeding meerkat, Suricata suricatta. *Animal Behaviour*, 85, 645-653.

Mealey L (2000). Sex differences: developmental and evolutionary strategies. In Academic Press New York, NY: Academic Press

M.J. Meaney, B.M. Tannenbaum, D.D. Francis, S. Bhatnagar, N. Shanks, V. Viau, D. O'Donnell, P.M. Plotsky (1994) Early environmental programming HPA responses to stress *Semin. Neurosci.*, 6, pp. 247-259

Meaney MJ (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annu Rev Neurosci* 24:1161–92.

Meaney MJ, Szyf M, Seckl JR. (2007). Epigenetic mechanisms of perinatal programming of HPA function and health. *Trends Mol Med* 13:269–77.

Meikle, D.B., Tilford, B.L. & Vessey, S.H. (1984). Dominance rank, secondary sex-ratio, and reproduction of offspring in polygynous primates. *American Naturalist*, **124**, 173–188.

Metcalfe NB, Monaghan P (2001) Compensation for a bad start: Grow now, pay later? *Trends Ecol Evol* 16:254–260.

Metcalfe NB, Monaghan P (2003) Growth versus lifespan: Perspectives from evolutionary ecology. Exp Gerontol 38:935–940.

Meylan, S. & Clobert, J. (2004). Maternal effects on offspring locomotion: influence of density and corticosterone elevation in the lizard Lacerta vivipara. *Physiol. Biochem. Zool*. 77, 450-458.

Meylan, S., Clobert, J., (2005). Is corticosterone-mediated phenotype development adaptive? Maternal corticosterone treatment enhances survival in male lizards. Hormones and Behavior 48, 44–52.

Miles JL, Landon J, Davison M, Krageloh CU, Thompson NM, Triggs CM & Breier BH (2009). Prenatally undernourished rats show increased preference for wheel running *v*. lever pressing for food in a choice task. *Br J Nutr* **101**, 902–908.

Moisiadis VG, Matthews SG (2014)a Glucocorticoids and fetal programming part 1: Outcomes. *Nat Rev Endocrinol* 10:391–402.

Moisiadis VG, Matthews SG (2014)b Glucocorticoids and fetal programming part 2: Mechanisms. *Nat Rev Endocrinol* 10:403–411.

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

Monaghan, P. & Haussmann, M. F. (2006). Do telomere dynamics link lifestyle and lifespan? *Trends Ecol. Evol.* 21, 47–53.

Monaghan P, Spencer KA. (2014). Stress and life history. *Curr Biol* 10:408–12

Moorad, J. A. & Nussey, D. H. (2016). Evolution of maternal effect senescence. *Proceedings of the National Academy of Sciences of the United States of America* **113**, 362–367

Moritz KM & Cullen-McEwen LA (2006). Kidney development and fetal programming. In *Early Life Origins of Health and Disease*, eds Wintour-Coghlan EM & Owens J, pp. 130–144. Springer.

Mousseau, T. A., & Fox, C. W. (1998). The adaptive significance of maternal effects. *Trends in Ecology & Evolution.* 13(10), 403-407.

Mousseau, T. A., Uller, T., Wapstra, E. and Badyaev, A. V. (2009). Evolution of maternal effects: past and present. *Philos. Trans. R. Soc. Lond. B Biol. Sci*. 364, 1035-1038.

Mueller BR, & Bale TL. (2008). Sex-specific programming of offspring emotionality after stress early in pregnancy. *J Neurosci* 28:9055–65.

Muller, C. A., & Manser, M. B. (2008). Scent-marking and intrasexual competition in a cooperative carnivore with low reproductive skew. *Ethology*, **114**, 174–185.

Müller W, Lessells CM, Korsten P, von Engelhardt N. (2007). Manipulative signals in family conflict? On the function of maternal yolk hormones in birds. *Am Nat* 169:E84.

Mumby HS, Mar KU, Hayward AD, Htut W, et al. (2015). Elephants born in the

high stress season have faster reproductive ageing. *Sci Rep* **5**: 13946.

Munro, C., & Stabenfeldt, G. (1984). Development of a microtitre plate enzyme immunoassay for the determination of progesterone. *Journal of Endocrinology*, **101**, 41–49.

Navara KJ. (2010). Programming of offspring sex ratios by maternal stress in humans: assessment of physiological mechanisms using a comparative approach. *J Comp Physiol B* **180**: 785–96.

Nelson-Flower, M. J., Hockey, P. A. R., O'Ryan, C., Raihani, N. J., Du Plessis, M. A., & Ridley, A. R. (2011). Monogamous dominant pairs monopolize reproduction in the cooperatively breeding pied babbler. *Behavioral Ecology*, **22**, 559–565.

Nettle D. (2014) What the future held: childhood psychosocial adversity is associated with health deterioration through adulthood in a cohort of British women. *Evol. Hum. Behav*. **35**, 519–525

Nettle D & Bateson M (2015) Adaptive developmental plasticity: What is it, how can we recognize it and when can it evolve? *Proc Biol Sci* 282:20151005.

Nettle D, Monaghan P, Gillespie R, Brilot B, Bedford T, Bateson M. (2015) An experimental demonstration that early-life competitive disadvantage accelerates telomere loss. *Proc. R. Soc. B* **282**, 20141610.

Nichols, H. J., Amos, W., Cant, M. A., Bell, M. B. V, & Hodge, S. J. (2010). Top males gain high reproductive success by guarding more successful females in a cooperatively breeding mongoose. *Animal Behaviour*, 80, 649–657.

Nichols, H. J., Bell, M. B. V, Hodge, S. J., & Cant, M. A. (2012). Resource limitation moderates the adaptive suppression of subordinate breeding in a cooperatively breeding mongoose. *Behavioral Ecology*, 23, 635–642.

Nichols, H. J., Fullard,K., & Amos, W (2014). Costly sons do not lead to adaptive sex ratio adjustment in pilot whales *Globicephala melas*. *Animal Behaviour*, 88, 203-209.

Nichols, H.J., Cant, M.A. & Sanderson, J.L. (2015). Adjustment of costly extra-group paternity according to inbreeding risk in a cooperative mammal. *Behav. Ecol*. **26**, 1486.

Nussey DH, Kruuk LE, Donald A, Fowlie M, Clutton-Brock TH. (2006) The rate of senescence in maternal performance increases with early-life fecundity in red deer. *Ecol. Lett.* **9**, 1342–1350

Nussey, D. H., Kruuk, L. E. B., Morris, A., Clements, M. N., Pemberton, J. M. & Clutton-Brock, T. H. (2009). Inter and intra-sexual variation in aging patterns across reproductive traits in a wild red deer population. *American Naturalist* **174**, 342–357.

O'Riain, M. J., Bennett, N. C., Brotherton, P. N. M., McIlrath, G., & Clutton-Brock, T. H. (2000). Reproductive suppression and inbreeding avoidance in wild populations of co-operatively breeding meerkats (Suricata suricatta). *Behavioural Ecology Sociobiology*. 48(6), 471-477.

Packer C, Pusey A.E, Eberly L.E (2001) Egalitarianism in female African lions. *Science*. **293**, 690–693.

Paitz RT, Bowden RM. (2013). Sulfonation of maternal steroids is a conserved metabolic pathway in vertebrates. *Integr Comp Biol* 53:895–901.

Paitz RT, Bukhari SA, Bell AM. (2016). Stickleback embryos use ATP-binding cassette transporters as a buffer against exposure to maternally derived cortisol. *Proc R Soc Lond B Biol Sci* 283 published online (doi:10.1098/rspb.2015.2838).

Palmer AC. (2011) Nutritionally mediated programming of the developing immune system. *Adv. Nutr*. **2**, 377–395.

Panchanathan K, Frankenhuis WE. (2016). The evolution of sensitive periods in a model of incremental development. *Proc R Soc Lond B Biol Sci* doi: 10.1098/ rspb. 2015.2439.

Patel JV, Vyas A, Cruickshank JK, Prabhakaran D, Hughes E, Reddy KS, Mackness MI, Bhatnagar D & Durrington PN (2006). Impact of migration on coronary heart disease risk factors: comparison of Gujaratis in Britain and their

contemporaries in villages of origin in India. *Atherosclerosis* **185**, 297–306

Pizzari, T., Dean, R., Pacey, A., Moore, H. & Bonsall, M. (2007). The evolutionary ecology of pre- and post-meiotic sperm senescence. *Trends in Ecology and Evolution* **23**, 131–140

Pontzer, H. (2015). Energy expenditure in humans and other primates: a new synthesis. *Annual Review of Anthropology*, **44**, 169–187.

Pontzer, H. (2017). The crown joules: energetics, ecology, and evolution in humans and other primates. *Evolutionary anthropology, Issues, news and reviews.* 26, 12-24.

R Core Team. 2016 *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.

Rando OJ. (2012). Daddy issues: paternal effects on phenotype. *Cell* 151:702–8.

Raulo A & Dantzer B. (2018). Associations between glucocorticoids and sociality across a continuum of vertebrate social behavior. *Ecology and Evolution.* 1-20.

Raynaud J & Schradin C. (2015) Corticosterone levels correlate with alloparental care in a sex-dependent manner in African striped mice, *Rhabdomys pumilio*. *Ethology* **121**, 57–67.

Rich EL & Romero LM (2005). Exposure to chronic stress downregulates corticosterone response to acute stressors. *American Journal of Physiology.* 288, 6. 1628-1636.

Rodgers AB, Morgan CP, Bronson SL, Revello S, Bale TL. (2013). Paternal stress exposure alters sperm microRNA content and reprograms offspring HPA stress axis regulation. *J Neurosci* 33:9003–12.

Romero, L.M., & Wikelski, M., (2001). Corticosterone levels predict survival probabilities of Galapagos marine iguanas during El Nino events. *Proc. Natl. Acad. Sci. USA* 98, 7366–7370.

Romero, L.M. (2002). Seasonal changes in plasma glucocorticoid concentrations in

free-living vertebrates. *Gen. Comp. Endocrinol*., 128, 1–24.

Romero, L. M. 2004. Physiological stress in ecology: lessons from biomedical research. *Trends in Ecology & Evolution*, **19**, 249–55.

Romero, L. M., Dickens, M. J. & Cyr, N. E. (2009). The reactive scope model - a new model integrating homeostasis, allostasis, and stress. *Horm. Behav*. 55, 375-389.

Rowell C (1972). The variable coloration of the acridoid grasshoppers. *Adv In Insect Phys* **8**, 145–198

Ruckstuhl, K. E., Colijn, G. P., Amiot, V. & Vinish, E. (2010) Mother's occupation and sex ratio at birth. BMC Public Health 10, 269.

Rubenstein DR (2007) Stress hormones and sociality: integrating social and environmental stressors. *Proceedings of the Royal Society of London B*, **274**, 967–975.

Rubenstein DR, Shen S-F (2009) Reproductive conflict and the costs of social status in cooperatively breeding vertebrates. *The American Naturalist*, **173**, 650–661.

Rubenstein, D. R., Skolnik, H., Berrio, A., Champagne, F. A., Phelps, S., & Solomon, J. (2016). Sex-specific fitness effects of unpredictable early life conditions are associated with DNA methylation in the avian gluco- corticoid receptor. *Molecular Ecology*, *8*, 1714–1728.

Rukstalis M, French JA. (2005) Vocal buffering of the stress response: exposure to conspecific vocalizations moderates urinary cortisol excretion in isolated marmosets. *Hormones and behavior.* 47:1–7.

Russell A. F. (2004) Mammals: comparisons and contrasts. In *Ecology and evolution of cooperative breeding in birds* (eds Koenig W. D., Dickinson J. L.), pp. 210–227. New York, NY: Cambridge University Press.

Russell, A. F., Young, A. J., Spong, G., Jordan, N., & Clutton-Brock, T. H. (2007). Helpers increase the reproductive potential of offspring in cooperative meerkats. *Proceedings of the Royal Society B*, **274**, 513–520.

Russell AF, Langmore NE, Cockburn A, Astheimer LB, Kilner RM. (2007). Reduced egg investment can conceal helper effects in cooperatively breeding birds. *Science* 317:941–4.

Russell AF, Langmore NE, Gardner JL, Kilner RM. (2008). Maternal investment tactics in superb fairy-wrens. *Proc R Soc B Biol Sci* 275:29–36.

Russell AF, & Lummaa V (2009) Maternal effects in cooperative breeders: from hymenopterans to humans. *Philosophical Transactions of the Royal Society B*, **364**, 1143–1167.

Saltz D. (2001). Sex ratio variation in ungulates: adaptation meets environmental perturbation of demography. *Oikos* **94**: 377–84.

Sanchez, M., (2006). The impact of early adverse care on HPA axis development: nonhuman primate models. *Horm. Behav*. 50, 623–631.

Sanchez, M., McCormack, K., Howell, B., (2015). Social Buffering of Stress Responses in Non-human Primates: Maternal Regulation of Development of Emotional Egulatory Brain Circuits. *Soc. Neurosci* 10 (5), 474–478.

Sanderson JL. (2012) Conflict and cooperation in vertebrate societies. PhD Thesis. University of Exeter.

Sanderson JL, Young AJ, Hodge SJ, Kyabulima S, Walker SL, Cant MA. (2014) Hormonal mediation of a carry-over effect in a wild cooperative mammal. *Functional Ecology* 28, 1377-1386.

Sanderson JL, Nichols HJ, Marshall HH, Vitikainen EIK, Thompson FJ, Walker SL, Cant MA, Young AJ. (2015a) Elevated glucocorticoid concentrations during gestation predict reduced reproductive success in subordinate female banded mongooses. *Biol. Lett.* **11**, 20150620.

Sanderson, J., Wang, J., Vitikainen, E.I.K., Cant, M.A. & Nichols, H.J. (2015b). Banded mongooses avoid inbreeding when mating with members of the same natal group. *Molecular. Ecology*.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, **21**, 55–89.

Savage, J. L., Russell, A. F & Johnstone, R. L (2015). Maternal allocation in cooperative breeders: should mothers match or compensate for expected helper contributions? *Animal behavior* 102, 189-197.

Schindler S, Gaillard J-M, Grüning A, Neuhaus P, et al. (2015). Sex-specific demography and generalization of the Trivers-Willard theory. *Nature* **526**: 249–52.

Schmidt KL, MacDougall-Shackleton EA, MacDougall-Shackleton SA. (2012). Developmental stress has sex-specific effects on nestling growth and adult metabolic rates but no effect on adult body size or body composition in song sparrows. *J Exp Biol* 215:3207–17.

Schlaepfer MA, Runge MC, Sherman PW. (2002). Ecological and evolutionary traps. *Trends Ecol Evol* 17:474–80.

Schöpper H, Klaus T, Palme R, Ruf T, Huber S. (2012) Sex-specific impact of prenatal stress on growth and reproductive parameters of guinea pigs. *J. Comp. Physiol. B* **182**, 1117–1127

Sharp, S. P. & Clutton-Brock, T. H. (2010). Reproductive senescence in a cooperatively breeding mammal. *Journal of Animal Ecology* **79**, 176–183

Sheldon, B. C. & West, S. A. (2004) Maternal dominance, maternal condition, and offspring sex ratio in ungulate mammals. *Am. Nat*. 163, 40–54.

Sheriff MJ, Krebs CJ, Boonstra R. (2009). The sensitive hare: sub-lethal effects of predator stress on reproduction in snowshoe hares. *J Anim Ecol* 78:1249–58.

Sheriff MJ, Krebs CJ, Boonstra R. (2010). The ghosts of predators past: population cycles and the role of maternal programming under fluctuating predation risk. *Ecology* 91:2983–94.

Sheriff MJ, Krebs CJ, Boonstra R. (2011). From process to pattern: how fluctuating

predation risk impacts the stress axis of snowshoe hares during the 10-year cycle. *Oecologia* 166:593–605.

Sheriff MJ, & Love OP (2013) Determining the adaptive potential of maternal stress. *Ecol Lett* 16:271–280.

Sheriff MJ, McMahon EK, Krebs CJ, Boonstra R. (2015). Predator-induced maternal stress and population demography in snowshoe hares: the more severe the risk, the longer the generational effect. *J Zool* 296:305–10.

Sheriff MJ, Dantzer B, Love OP and Orrock JL. (2018). Error management theory and the adaptive significance of transgenerational maternal-stress effects on offspring phenotype. *Ecology and Evolution.* 1-10.

Sheppard CE., Inger R., McDonald RA., Barker S., Jackson AL., Thompson FJ., Vitikainen EIK., Cant MA., & Marshall HH (in press). Intragroup competition predicts individual foraging specialisation in a group-living mammal. *Ecology Letters.*

Sih A, Bolnick DI, Luttbeg B, Orrock JL, Peacor SD, Pinto LM, Preisser E, Rehage JS, Vonesh JR. (2010). Predator-prey naïveté, antipredator behavior, and the ecology of predator invasions. *Oikos* 119:610–21.

Silk, J.B. (1993). The evolution of social conflict among female primates. In: *Primate Social Conflict* (Ed. by W. A. Mason & S. Mendoza), pp. 49-83. Albany, New York: State University of New York Press.

Silk JB & Brown GR. (2008) Local resource competition and local resource enhancement shape primate birth sex ratios. *Proceedings of the Royal Society of London, Series B* 275. 1761-1765

Sloboda DM, Hart R, Doherty DA, Pennell CE & Hickey M (2007). Age at menarche: Influences of prenatal and postnatal growth. *J Clin Endocrinol Metab* **92**, 46–50.

Sloboda DM, Howie GJ, Pleasants A, Gluckman PD, Vickers MH (2009) Pre- and postnatal nutritional histories influence reproductive maturation and ovarian function in the rat. *PLoS One* 46744.

Smith CC & Fretwell SD (1974) The Optimal balance between size and number of offspring. *The American Naturalist.* 108, 962

Smith TE, French JA. (1997) Psychosocial stress and urinary cortisol excretion in marmoset monkeys. *Physiology & behavior*. 62: 225–232.

Snell-Rood EC. (2012). Selective processes in development: implications for the costs and benefits of phenotypic plasticity. *Integr Comp Biol* 52:31–42.

Solomon N.G, French J.A Cooperative breeding in mammals. In Cambridge University Press 1997 Cambridge, UK. Cambridge University Press

St-Cyr S, Abuaish S, Sivanathan S, McGowan PO (2017). Maternal programming of sex-specific responses to predator odor stress in adult rats. *Horm Behav* 94:1–12.

Stearns SC & Koella JC. (1986). The evolution of phenotypic plasticity in life-history traits: predictions of reaction norms for age and size at maturity. *Evolution.* 40, 5, 893-913.

Stearns, S. C. (1992). The evolution of life histories. 249. Oxford: Oxford University Press.

Sterling P & Eyer J. (1988) Handbook of life stress. Cognition and Health. J. Wiley Ltd

Stier, A., Reichert, S., Massemin, S., Bize, P. and Criscuolo, F. (2012). Constraint and cost of oxidative stress on reproduction: correlative evidence in laboratory mice and review of the literature. *Front. Zool*. 9, 37-46.

Storm J. J & Lima S. L (2010) Mothers forewarn offspring about predators: a transgenerational maternal effect on behavior *Am. Nat.,* 175, pp. 382-390

Strange MS, Bowden RM, Thompson CF, Sakaluk SK. (2016). Pre and postnatal effects of corticosterone on fitness-related traits and the timing of endogenous corticosterone production in a songbird. *J Exp Zool A Ecol Genet Physiol* 325:347–59.

Sullivan EC, Hinde K, Mendoza SP, Capitanio JP. (2011). Cortisol concentrations

in the milk of rhesus monkey mothers are associated with confident temperament in sons, but not daughters. *Dev Psychobiol* 53:96–104.

Taborsky, B. (2006). The influence of juvenile and adult environments on life-history trajectories. *Proceedings of the Royal Society of London B: Biological Sciences*, *273*, 741–750.

Tammen SA., Friso S., & Choi S. (2013). Epigenetics: The link between nature and nurture. *Molecular Aspects of Medecine.* 34, 4, 753-764.

Trimmer PC, Ehlman SM, Sih A. (2017). Predicting behavioural responses to novel organisms: state-dependent detection theory. *Proc R Soc Lond B Biol Sci* 284 published online (doi:10.1098/rspb.2016.2108).

Thompson, F. J., Marshall, H. H., Sanderson, J. L., Vitikainen, E. I., Nichols, H. J., Gilchrist, J. S., Young A.J., Hodge, S.J. & Cant, M. A. (2016). Reproductive competition triggers mass eviction in cooperative banded mongooses. *Proceedings of The Royal Society B*, 283, 1826, 20152607.

Tinbergen, N. 1963. On aims and methods of ethology. *Zeitschrift für Tierpsychologie*, **20**, 410–433.

Török, J., R. Hargitai, G. Hegyi, Z. Matus, G. Michl, P. Péczely, B. Rosivall, and G. Tóth. (2007). Carotenoids in egg yolks of collared flycatchers (*Ficedula albicollis*) in relation to parental quality, environmental factors and laying order. *Behavioral Ecology and Sociobiology* 61:541–550.

Trivers, R. L. 1972. Parental Investment and Sexual Selection. In: *Sexual Selection and the Descent of Man*, (Ed. by B. Campbell), pp. 136–179. Aldine-Atherton, Chicago.

Trivers RL, & Willard DE (1973) Natural selection of parental ability to vary the sex ratio of offspring. *Science* **179**, 90–92.

Trivers RL (1974). Parent-offspring conflict. *Am Zool* **14**, 249–264.

Tung J, Archie EA, Altmann J, Alberts SC. (2016). Cumulative early life adversity

predicts longevity in wild baboons. *Nat Commun* **7**: 11181.

Van de Pol, M. & Wright, J. A. (2009). Simple method for distinguishing within-versus between-subject effects using mixed models. *Animal Behaviour*. 77(3), 753-758.

Veru F, Laplante DP, Luheshi G, King S. (2014) Prenatal maternal stress exposure and immune function in the offspring. *Stress* **17**, 133–148.

Vickers MH, Breier BH, Cutfield WS, Hofman PL & Gluckman PD (2000). Fetal origins of hyper-phagia, obesity, and hypertension and postnatal amplification by hyper-caloric nutrition. *Am J Physiol Endocrinol Metab* **279**, E83–E87.

Vickers MH, Breier BH, McCarthy D & Gluckman PD (2003). Sedentary behavior during postnatal life is determined by the prenatal environment and exacerbated by postnatal hypercaloric nutrition. *Am J Physiol Regul Integr Comp Physio* **285**, R271–R273.

Vitikainen EIK, Cant MA, Sanderson JL, Mitchell C, Nichols HJ, Marshall HH, et al. (2016) Evidence of oxidative shielding of offspring in a wild mammal. *Front Ecol Evol*.; 4: 58.

Vitikainen EIK, Marshall HH, Thompson FJ, Sanderson JL, Bell MBV, Gilchrist JS, et al. (2017) Biased escorts: offspring sex, not relatedness explains alloparental care patterns in a cooperative breeder. *Proc R Soc B Biol Sci;* 284: 20162384.

Uller, T., Pen, I., Wapstra, E., Beukeboom, L. W., & Komdeur, J. (2007). The evolution of sex ratios and sex-determining systems. Trends in Ecology & Evolution, 22, 292-297.

Uller T (2008) Developmental plasticity and the evolution of parental effects. *Trends Ecol. Evol*. **23**, 432–438.

Uller T, Pen I. (2011). A theoretical model for the evolution of maternal effects under parent–offspring conflict. *Evolution* 65:2075–84.

Uller T, Nakagawa S, English S. (2013). Weak evidence for anticipatory parental

effects in plants and animals. *J Evol Biol* 26:2161–70.

Wade M. J., & Kalisz S. (1990) The causes of natural selection. *Evolution* **44**, 1947

Wang J (2010) Do marker-based paternity assignments favour heterozygous and unrelated males? *Molecular Ecology*, 19, 1898–1913.

Wasser, S.K., Keim, J.L., Taper, M.L. & Lele, S.R. (2011). The influences of wolf predation, habitat loss, and human activity on caribou and moose in the Alberta oil sands. *Front. Ecol. Environ*., 9, 546–551.

West-Eberhard MJ. (2003). *Developmental plasticity and evolution*. New York (NY): Oxford University Press.

Weaver ICG, Cervoni N, Champagne FA, D'Alessio AC, Sharma S, Seckl JR, Dymov S, et al. (2004) Epigenetic programming by maternal behavior. *Nature Neuroscience*. 7 :847–854.

Weladji R.B, Loison A, Gaillard J.-M, Holand O, Mysterud A, Yoccoz N.G, Nieminen M, Stenseth N.C (2008) Heterogeneity in individual quality overrides costs of reproduction in female reindeer. *Oecologia*. **156**, 237–247.

Wells JCK (2007) The Thrifty Phenotype as an adaptive maternal effect. *Biological Reviews.* 82, 1, 143-172

Wells JCK., Yao P., Williams JE., & Gaynor R. (2016). Maternal investment, life-history strategy of the offspring and chronic adult disease risk in South Asian women in the UK. *Evolution, medicine and public health.* 1. 133-145.

West S.A, Sheldon B.C (2002) Constraints in the evolution of sex ratio adjustment. *Science*. **295**, 1685–1688

West SA. (2009). *Sex Allocation*. Princeton: Princeton University Press.

Wiebe, K. L. & Bortolotti, G. R.(1992) Facultative sex ratio manipulation in American kestrels. *Behav. Ecol. Sociobiol*. 30, 379–386.

Wilkin, T. A., & Sheldon, B. C. (2009). Sex differences in the persistence of natal environmental effects on life histories. *Current Biology*, *19*, 1998–2002.

Williams GC. (1957) Pleiotropy, natural selection, and the evolution of senescence. *Evolution* **11**, 398–411

Williams GC. (1966) Natural selection, the costs of reproduction, and a refinement of Lack's principle. *Am. Nat.* **100**, 687–690.

Williams, G. C. (1979) Question of adaptive sex-ratio in outcrossed vertebrates. Proc. Biol. Sci. 205, 567–580.

Wingfield, J. C., Maney, D., Breuner, C., Jacobs, J., Lynn, S., Ramenofsky, M., & Richardson, R. (1998.) Ecological bases of hormone-behavior interactions: The "emergency life history stage". *American Zoologist*, **38**, 191–206.

Wingfield, J. C. (2005). A continuing saga: the role of testosterone in aggression. *Hormones and Behavior*, **48**, 253–5.

Wolf J.B, Brodie E.D, Cheverud J.M, Moore A.J, Wade M.J (1998) Evolutionary consequences of indirect genetic effects. *Trends Ecol. Evol*. **13**, 64–69.

Wong, J. W. Y., & Kölliker, M. (2014). Effects of food restriction across stages of juvenile and early adult development on body weight, survival and adult life history. *Journal of Evolutionary Biology*, *27*, 2420–2430.

Young A.J, Carlson A.A, Montfort S.L, Russell A.F, Bennett N.C, Clutton-Brock T.H (2006) Stress and the suppression of subordinate reproduction in cooperatively breeding meerkats. *Proc. Natl Acad. Sci. USA*. **103**, 12 005–12 010.

Young, K. M., Walker, S. L., Lanthier, C., Waddell, W. T., Monforts, S. L., & Brown, J. L. (2004). Non- invasive monitoring of adrenocortical activity in carnivores by fecal glucocorticoid analyses. *General and Comparative Endocrinology*, **137**, 148–65.

Zanette LY, White AF, Allen MC, Clinchy M. (2011). Perceived predation risk reduces the number of offspring songbirds produce per year. *Science* 334:1398–

401.

Zera, A. J., & Harshman, L. G. (2001). The physiology of life history trade- offs in animals. *Annual Review of Ecology and Systematics*, *32*, 95–126.