BIOLOGICAL REVIEWS
Cambridge Philosophical Society
Biol. Rev. (2021), **96**, pp. 1951–1968.
doi: 10.1111/brv.12733

Who listens to mother? A whole-family perspective on the evolution of maternal hormone allocation

Kat Bebbington[1,2*] and Ton G. G. Groothuis[1]

[1]*Behavioural Biology, Groningen Institute for Evolutionary Life Sciences, University of Groningen, Nijenborgh 7, Groningen, 9747AG, The Netherlands*
[2]*Animal Sciences, Wageningen University and Research, de Elst 1, Wageningen, 6708WD, The Netherlands*

ABSTRACT

Maternal effects, or the influence of maternal environment and phenotype on offspring phenotype, may allow mothers to fine-tune their offspring's developmental trajectory and resulting phenotype sometimes long after the offspring has reached independence. However, maternal effects on offspring phenotype do not evolve in isolation, but rather within the context of a family unit, where the separate and often conflicting evolutionary interests of mothers, fathers and offspring are all at play. While intrafamilial conflicts are routinely invoked to explain other components of reproductive strategy, remarkably little is known about how intrafamilial conflicts influence maternal effects. We argue that much of the considerable variation in the relationship between maternally derived hormones, nutrients and other compounds and the resulting offspring phenotype might be explained by the presence of conflicting selection pressures on different family members. In this review, we examine the existing literature on maternal hormone allocation as a case study for maternal effects more broadly, and explore new hypotheses that arise when we consider current findings within a framework that explicitly incorporates the different evolutionary interests of the mother, her offspring and other family members. Specifically, we hypothesise that the relationship between maternal hormone allocation and offspring phenotype depends on a mother's ability to manipulate the signals she sends to offspring, the ability of family members to be plastic in their response to those signals and the capacity for the phenotypes and strategies of various family members to interact and influence one another on both behavioural and evolutionary timescales. We also provide suggestions for experimental, comparative and theoretical work that may be instrumental in testing these hypotheses. In particular, we highlight that manipulating the level of information available to different family members may reveal important insights into when and to what extent maternal hormones influence offspring development. We conclude that the evolution of maternal hormone allocation is likely to be shaped by the conflicting fitness optima of mothers, fathers and offspring, and that the outcome of this conflict depends on the relative balance of power between family members. Extending our hypotheses to incorporate interactions between family members, as well as more complex social groups and a wider range of taxa, may provide exciting new developments in the fields of endocrinology and maternal effects.

Key words: maternal effects, maternal hormones, intrafamilial conflict, parent–offspring conflict, sexual conflict, offspring competition, honest signalling, plasticity, interacting phenotypes

CONTENTS

* Address for correspondence (Tel: +0031624982876; E-mail: katlbebbington@gmail.com)

I. INTRODUCTION

Family members are united by the common goal of producing successful offspring in order to propagate their genes (Hamilton, 1964). However, families also consist of selfish individuals who each pursue their own optimal strategies and have a considerable degree of power to influence one another's physiology, behaviour and fitness (Royle, Hartley & Parker, 2002). The importance of incorporating the separate evolutionary interests of family members into our understanding of family life was highlighted by the work of Trivers (1972, 1974); the subsequent paradigm shift towards a focus on conflict between parents (Royle *et al.*, 2002; Lessells & McNamara, 2011; Bebbington & Hatchwell, 2016), between parents and their offspring (Godfray, 1995; Smiseth, Wright & Kölliker, 2008; Kölliker *et al.*, 2015), and between offspring themselves (O'Connor, 1978; Mock, 1984; Stockley & Parker, 2002), has substantially changed our view of family life. Researchers are increasingly recognising that the evolution of strategies in a family context is the result of both the cooperation that arises through shared fitness interests and the conflict that arises through selfish genes (Hamilton, 1964: Trivers, 1972; Royle *et al.*, 2002). Because of this complex interplay, family life is only understandable if the fitness interests of every family member, and their respective power to exert those interests, are taken into account.

With respect to many components of family life, such as offspring competition, parent–offspring conflict over resource allocation and parental conflict over reproductive investment, the assumption that outcomes are the result of an intra-familial tug-of-war between fitness interests that may or may not align is implicitly built into the hypotheses that researchers construct and test. When relatedness between family members is low, conflict and competition are expected to dominate (Godfray, 1995), but where family members benefit from each other, cooperation may have the upper hand (Rebar *et al.*, 2020). In other components of family life, however, the influence that family members have on each other is less well acknowledged. In this review, we highlight one prominent example of a neglected family conflict: maternal effects, or influences of the mother's genotype and phenotype on the phenotype of her offspring. One of the most extensively studied of these (Groothuis *et al.*, 2019), and the one which we therefore focus upon here, is the maternal prenatal provisioning of hormones to developing embryos. Across the animal kingdom, mothers expose their developing embryos to a variety of different hormones [e.g. birds (Schwabl, 1993; Eising *et al.*, 2001), reviewed by Groothuis *et al.*, 2005*b* and von Engelhardt & Groothuis, 2011; insects (Libbrecht *et al.*, 2013); mammals (Walsh, Stanczyk & Novy, 1984); reptiles (Elf, 2003); fish (Brown *et al.*, 1988)], the concentration of which varies both between mothers (Collier *et al.*, 1982; Tschirren *et al.*, 2009; Giesing *et al.*, 2010) and also between offspring of the same mother (Reed & Vleck, 2001; Groothuis & Schwabl, 2002; Lessells, Ruuskanen & Schwabl, 2016). Such maternally derived hormones have repeatedly been shown to affect offspring development trajectories. For example, androgens enhance embryonic development and post-hatching growth rate in black-headed gulls *Larus ridibundus* (Eising *et al.*, 2001), while oestradiol appears to mitigate the relationship between temperature and sex determination in several reptile species (reviewed in Elf, Lang & Fivizzani, 2002). In numerous insect taxa, maternal hormones are responsible for polyphenisms and caste determination (e.g. Rembold, Czoppelt & Rao, 1974; Simpson & Miller, 2007). Exposure to maternal hormones during development can also influence behavioural traits such as juvenile dispersal in lizards and birds (De Fraipont *et al.*, 2000; Tschirren, Fitze & Richner, 2006), gregarious behaviour in desert locusts *Schistocerca gregaria* (Tawfik & Sehnal, 2003) and offspring aggression in spotted hyenas *Crocuta crocuta* (Dloniak, French & Holekamp, 2006). In birds, variation in maternal hormone exposure can even be detected long after offspring reach maturity, influencing personality (Ruuskanen & Laaksonen, 2010), secondary sexual traits (Partecke & Schwabl, 2008) and parental care (Ruuskanen *et al.*, 2012). Clearly, maternal hormones can strongly influence a developing offspring's phenotype, going so far as to change its appearance and behaviour in many insect taxa. Given that hormones have such a potentially large impact on the fitness of offspring and their parents, it is surprising that they have received so little attention when compared to other sources of intrafamilial conflict, such as offspring begging or parental conflict over care.

As for any other family-based interaction, evolutionary conflicts over maternal hormone allocation arise because the maternal optimum weighs the mother's own survival and future reproduction more heavily than does the offspring optimum (Trivers, 1974), while the father's optimum is also driven by opportunities for reproduction outside of the

current breeding partnership (Michl *et al.*, 2004; Paquet & Smiseth, 2016). This creates an evolutionary battleground within which offspring and fathers evolve strategies to move maternal hormone allocation away from the mother's optimum and towards their own, while mothers evolve counter-adaptations to reassert their control (Müller *et al.*, 2007; Groothuis *et al.*, 2019; Fig. 1). Until relatively recently, researchers operated under the assumption that a mother is entirely in control of what quantity of hormones are provisioned to developing embryos and what phenotypic adjustments they induce; as such, mothers have 'won' any underlying conflict and offspring and fathers are helpless to oppose her decisions (Schwabl, 1998; Gil, 2003; Tobler & Smith, 2010). While the apparent prevalence and extensive influence of maternal hormones on offspring development across animal taxa supports this view to some extent, emerging research is demonstrating the potential for an active role for both offspring and fathers in determining the extent to which maternal hormones influence offspring phenotype. For example, avian embryos convert maternally derived yolk androgens into inactive forms that no longer influence offspring development (Paitz, Bowden & Casto, 2010); moreover, the rate at which they do so is dependent on their position in the laying order (Kumar *et al.*, 2018), indicating scope for offspring to employ context-dependent strategies to handle maternal hormone regimes. Intriguingly, the conversion of some maternal hormones from active to inactive forms is reversible, raising the possibility that offspring can effectively 'switch on' maternal hormone signals in certain ecological contexts (Kumar *et al.*, 2018), or even use them to synthesise other hormones (Paitz & Bowden, 2011). Avian embryos also appear to be capable of regulating the density of hormone receptors, providing another mechanism by which offspring can vary the extent of maternal influence on their emerging phenotype (Kumar *et al.*, 2019*b*). Fathers, too, may have considerable potential to move offspring phenotype towards that which best fits their own interests. Behavioural studies demonstrate that fathers show an enormous amount of plasticity in their parenting behaviour (Stamps *et al.*, 1987; Smiseth & Moore, 2004; Fresneau & Müller, 2019); just as offspring can alter the influence of maternal hormones on their phenotype before birth, fathers can alter it by varying their parental investment to influence not only offspring size and quality (e.g. Hunt & Simmons, 2000) but also behaviour (McGhee & Bell, 2014). Moreover, fathers may be able to adjust

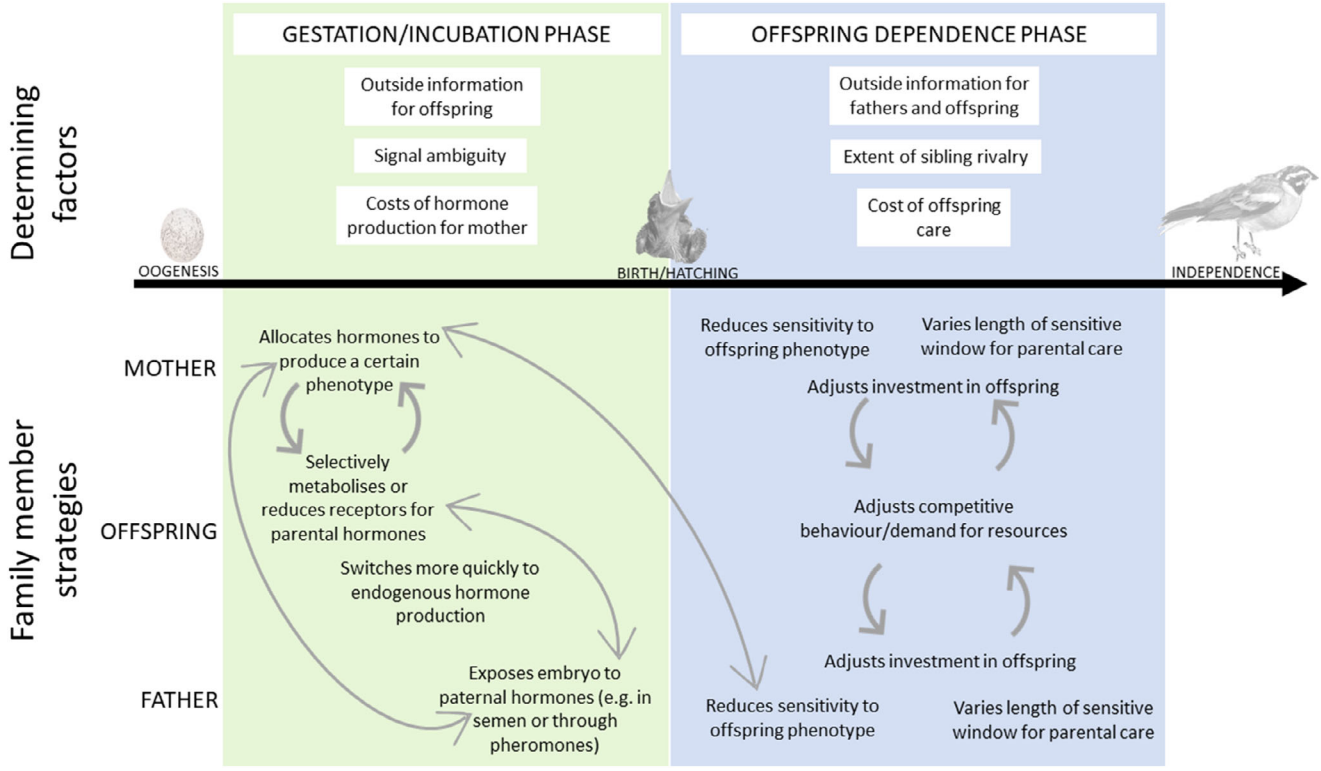

Fig 1. Overview of maternal hormone allocation under intrafamilial conflict. At each point along the timeline between oogenesis until offspring independence or dispersal, various family members have the opportunity to express strategies that aim to skew offspring phenotype towards their personal optimum. The success and evolutionary stability of such strategies is determined by various factors regarding costs, information or power asymmetries, and social context ('determining factors'), and also the actions of other family members (denoted by grey arrows). We argue that understanding the link between maternal hormone allocation and offspring phenotype requires understanding which family member strategies have evolved under intrafamilial conflict, which in turn depends on the ecological and social context.

embryonic development directly by exposing offspring to their own hormones, for example through semen in species with internal fertilisation (Lelono, Riedstra & Groothuis, 2019) or through urine in species with external fertilisation (Keller-Costa, Canàrio & Hubbard, 2015). Offspring and fathers, then, are limited in what maternal hormone signals they receive, but not necessarily in what they do with those signals. It is therefore important to consider how maternal hormone allocation affects the evolution of strategies employed by other family members.

Since conceptual studies first highlighted the possibility of intra-familial conflict over the allocation of maternal hormones within and between offspring (Groothuis *et al.*, 2005*b*; Müller *et al.*, 2007; Russell & Lummaa, 2009), a few models have been constructed to explore the theoretical outcomes of such conflict (Miller, Gavrilets & Rice, 2006; Uller & Pen, 2011; González-Forero, 2014; Kuijper & Johnstone, 2018). However, these models tend to focus on interactions between a mother and a set of offspring that are assumed to have identical fitness interests, rather than accounting for the unique and separate fitness interests of all family members, and empirical research that explicitly tests predictions about how family conflicts influence maternal effects is rare (but see Kumar *et al.*, 2018). This may seem a somewhat surprising statement, given that testing the influence of maternal hormones on offspring phenotype has been a popular topic of study for behavioural ecologists for several decades (Groothuis *et al.*, 2005*b*). Perhaps the tendency for maternal hormone allocation to fall within the realm of whole-organism behaviour, rather than also attracting the attention of mechanism-focussed endocrinologists, is one reason for the lack of concerted progress in this field. And while a recent meta-analysis suggested that, in general, maternal effects have adaptive value for offspring fitness (Yin *et al.*, 2019), the conclusions of this paper have since shown to be premature (Sánchez-Tójar *et al.*, 2020) and the relationship between maternal effects and offspring phenotype is far from ubiquitous (Uller, Nakagawa & English, 2013). So far, there has been little attempt to understand the substantial between- and within-species variation in the extent to which maternal hormones influence offspring phenotype (although see Podmokła, Drobniak & Rutkowska, 2018). We argue that, rather than being a nuisance factor in the field, the 'noise' found both within and between studies of maternal hormones offers the promise of a much larger conceptual advance. Why does the effect of maternal hormones on offspring development vary so much? If intrafamilial conflict is indeed at play, as evolutionary theory predicts, it is not at all surprising that this effect is so variable; mothers, fathers and offspring should all be under selection to exert their own personal optimum outcome for offspring development. As with so many aspects of family life, the social and environmental context is likely to be key, and will determine what strategies family members can evolve (Fig. 1). In order to progress further, we must predict when it might pay other family members to actively oppose the maternally induced offspring phenotype within a conceptual framework that explicitly assumes the presence of intrafamilial conflict, as is routinely done for other forms of family-based interactions (Parker, Royle & Hartley, 2002).

In this review, we highlight new themes and testable hypotheses that arise when approaching research on maternal hormones in the light of intrafamilial conflict (Table 1). The concepts we explore are relevant to all animal taxa; there is likely to be scope for mothers to manipulate the developmental environment, and for other family members to oppose her actions, regardless of whether embryos develop internally or in external eggs. Similarly, although largely applied to species exhibiting clear family-based social interactions, the concept of evolutionary conflicts between mothers and offspring, and between offspring themselves, is not dependent on a prolonged period of offspring dependence. While we therefore aim to draw on literature and discuss themes applicable on a broad taxonomic level, much of our understanding of maternal hormones is currently based on research in birds due to the relative ease of isolating and manipulating embryo environments in this class. Later in the review, we discuss how known mechanisms in birds are likely to compare to those in other animal taxa.

Specifically, we discuss three key themes that may be instrumental in moving the field of maternal hormone research towards a family-oriented conceptual framework: (*i*) the evolutionary implications of dishonest signalling by the mother; (*ii*) the impact of plasticity in the responses of other family members; and (*iii*) the role of interacting phenotypes in determining the various behaviours and strategies we observe in nature. Throughout and in Table 1, we provide suggestions for future research that may help align some of the current discrepancies in the extensive literature on maternal effects.

II. THE (DIS)HONESTY OF MATERNAL HORMONE SIGNALS

If maternal effects allow mothers to tailor offspring phenotypes to environmental conditions, what would be the benefit to the mother of producing dishonest signals? In the traditional, linear approach to studying maternal hormones, there is none. In the light of intrafamilial conflict, however, there are several possible scenarios under which mothers could be selected to be dishonest in their hormone signalling. Understanding when this might be the case is not only important for understanding variation in female allocation strategies, but is also the first step towards predicting how other family members should respond to maternal hormones.

(1) Maternal exaggeration of hormone signals

Maternal hormones can be thought of as signals sent by the mother and received by other family members (directly *via* hormone receptors by the developing embryo, indirectly *via* offspring phenotype by the father). Under intrafamilial

Table 1. Future directions for research into the evolution of maternal hormone allocation under intrafamilial conflict. For each of the three themes discussed in this review, we highlight important open questions and formulate hypotheses that can be tested. Full descriptions of these arguments can be found in the main text

Theme	Open questions	Hypotheses
The (dis)honesty of maternal hormone signals	(I) Maternal exaggeration: do mothers over-allocate hormones to compensate for reduced responses by other family members?	H1: Mothers allocate more dispersal-promoting hormones to embryos in habitats where mothers benefit from offspring dispersal and offspring do not H2: Offspring evolve more resistance to hormones that are cheap for mothers to produce than those that are costly to produce (e.g. thyroid hormones)
	(II) Maternal disguise: do mothers use hormones with multiple effects to ensure that other family members cannot afford to evolve resistance to their effect on offspring phenotype?	H1: The correlation between prenatal hormone concentration and postnatal trait expression in offspring or fathers is stronger for more pleiotropic hormones
	(III) Maternal bet-hedging: do family members respond differently to maternal hormones depending on the likely rate of maternal error?	H1: In predictable environments, maternal error rate is lower and family members respond more strongly to prenatal hormones H2: In predictable environments, family members are better informed about optimal offspring phenotype and respond less strongly to prenatal hormones
Adaptive plasticity in response to maternal hormone signals	(IV) Plasticity in offspring: can offspring use outside information to move their phenotypic trajectory away from pure maternal control?	H1: Offspring respond less strongly to maternal hormones when informative cues (vocalisations, incubation patterns) are available
	(V) Plasticity in fathers: do fathers tailor their response to offspring phenotype according to their (perceived) level of accurate information?	H1: Fathers respond more strongly to offspring phenotype when they are present before offspring are born than when they are only present after birth H2: Fathers only attempt to manipulate prenatal offspring phenotype if the contested hormone has a linear effect on offspring trait development
	(VI) Timing and mechanics of adaptive plastic responses: do fathers and offspring use variation in sensitive windows to optimise plastic responses to maternal hormones?	H1: Fathers and offspring have longer sensitive windows when maternal hormone signals are likely to be dishonest (exaggerated/erroneous) H2: Offspring switch to endogenous hormone production earlier in development when mother–offspring conflict is greater H3: Mothers and fathers adjust their parenting strategies in the postnatal period to counteract sub-optimal offspring phenotypes
Interacting phenotypes	(VII) Do family members tailor their response to maternal hormone signals according to the expected response of other family members?	H1: The correlation between prenatal hormone concentration and offspring phenotype is stronger when there is lower sexual conflict over offspring care H2: Fathers are more sensitive to maternally programmed offspring phenotype when sibling rivalry is weak

conflict, selection should favour family members who respond by either dampening or amplifying those signals, depending on whether the mother's optimal amount of hormone lies above or below that which is most beneficial for the receiver. The stage is set for an arms race similar to those in other family interactions, such as offspring begging for parental care (Müller *et al.*, 2007). For example, mothers might use testosterone to increase an offspring's growth rate and thus reduce the period of parental care. If that increased growth rate is not optimal for the offspring to express because of the associated oxidative stress (Råberg *et al.*, 1998), an arms race will ensue wherein offspring are selected to reduce their sensitivity to the effect of testosterone and mothers are selected to compensate by exaggerating the signal (i.e. increasing testosterone concentration; see Fig. 1). The same process will occur if mothers use hormones to create offspring that elicit what is, for the father, a super-optimal amount of parental investment: fathers are selected to dampen their response to that trait in order to avoid being over-exploited, and mothers counteract by amplifying the signal. To date, our understanding of intrafamilial arms races is largely restricted to studies of bi-directional nutrient and hormone transfer across mammalian placentas (e.g. Fowden, Comline & Silver, 1984; Chuong, Tong & Hoekstra, 2010), with very little known about dynamic adaptations and counter-adaptations in non-placental taxa [although evidence suggests a placenta-like role for the extra-embryonic membranes of oviparous species

(Albergotti *et al.*, 2009)]. This constitutes an important gap in our understanding; the absolute quantity of hormones provisioned to a given embryo might not tell researchers much about the adaptive value of maternal allocation strategies unless the potential for signal escalation is explicitly taken into account. For example, researchers might discover that mothers allocate a greater concentration of a given hormone to certain offspring or reproductive attempts than to others. If we assume that maternal hormone allocation is conflict-free, we may conclude that mothers are favouring those offspring with a greater hormonal signal. The alternative, conflict-centred interpretation would be that increased hormone allocation is a counter-adaptation by mothers to compensate for the fact that offspring are under selection to dampen the functional effect of that signal.

Given that maternal hormone signals exist across a wide range of taxa and appear in many cases to be at least somewhat linked to the development of offspring phenotype, it seems likely that certain mechanisms can prevent the escalation of this arms race. Firstly, manipulation of a signal by the mother can be maintained if the receiver of that manipulation (in this case, fathers and offspring) is selected to develop along the lines of the mother's intention as efficiently as possible, for example because the inclusive fitness benefits of doing so outweigh the direct cost (González-Forero, 2014). Secondly, conflict may be limited if the honesty of maternal hormones is kept in check by costs for the signaller (Zahavi, 1975; Kilner & Johnstone, 1997). Theoretical work by Uller & Pen (2011) supports this idea: where the action of hormone deposition is costly for the mother, the offspring phenotype approaches the offspring's optimum, whereas when selectively modifying maternal signals is costly for the embryo, it resembles the maternal optimum. It has been argued that costs associated with hormone deposition are likely minimal (Groothuis & Schwabl, 2007) [with the exception of thyroid hormones, which are synthesised using environmentally limited iodine (Fisher, 1996; Hsu *et al.*, 2016)]. However, there is some evidence that mothers may incur indirect metabolic (Tschirren *et al.*, 2016) and longevity (Tschirren *et al.*, 2014) costs of hormone transfer. Another intriguing possibility is that hormones themselves are cheap to produce but become costly if their intended function is tightly linked to the availability of some other resource that is costly for the mother to provide. For example, testosterone is known to increase offspring developmental growth rate (Schwabl, 1996; Helle, Laaksonen & Huitu, 2012) but suppress offspring immune function (Uller & Olsson, 2003; Sandell, Tobler & Hasselquist, 2012); in order to gain the intended benefit of allocating high testosterone to an embryo, mothers must also be able to afford the simultaneously increased allocation of energy and immune factors. This indirect form of cost might be incredibly important in determining the scope for maternal dishonesty in hormone signalling; a better understanding of the overall costs of maternal hormone production would greatly help in predicting the outcome of the intrafamilial arms race over their use to tailor offspring development.

Aside from production costs, whether or not it pays a mother to be dishonest in her hormone provisioning may also depend on the extent to which she can control the downstream effects of that hormone provisioning. If, for example, mothers produce offspring phenotypes that demand more parental care, this action would be costly for the mother if she, along with her partner, must increase her parental investment in these demanding offspring. If, in contrast, the mother can produce an offspring phenotype that only exerts extra care from the father (for example, by dampening her own response to offspring begging), there is no cost to her of exaggerating the signal to produce a highly demanding offspring phenotype as only her partner will bear the costs of increased care (as seems to be the case in yellow-legged gulls *Larus michaellis*; Noguera, Kim & Velando, 2013). In the latter case, selection should favour fathers who express a reduced response to offspring phenotypes to compensate for maternally induced exaggeration of offspring solicitation behaviour – and the parental arms race begins.

The first step in testing whether intrafamilial conflict selects for mothers who exaggerate their hormone signals would be to measure within-population variation in hormone allocation across environmental contexts. One particularly interesting case study that has already received theoretical attention (Uller & Pen, 2011) is juvenile dispersal. Several different hormones are known to play a role in dispersal decisions; corticosterone reduces dispersal in common lizards *Lacerta vivipara* (De Fraipont *et al.*, 2000), while testosterone is positively related to dispersal distance in great tits *Parus major* (Tschirren *et al.*, 2006). Importantly, there is often intrafamilial conflict over juvenile dispersal, which usually relates to the problem of sharing limited local resources (Ekman, Eggers & Griesser, 2002; Stephens *et al.*, 2004; Kingma *et al.*, 2016). In areas where mothers benefit from offspring dispersal but offspring have higher fitness if they stay, we would expect mothers to deposit a higher concentration of hormones that promote exploratory, dispersal-prone offspring phenotypes, and a concurrent reduction in the strength of offspring responses to those hormones. Testing this hypothesis requires systems where optimal offspring dispersal is known to vary between mothers; a great deal of basic ecological information is necessary to determine when this is the case. We argue that the necessary combination of detailed behavioural data and lifetime fitness measures is already available for numerous existing wild population studies, some of which have already explored variation in endocrinological profiles in birds (e.g. Paquet *et al.*, 2013), mammals (e.g. Dantzer *et al.*, 2013) and fish (e.g. Bender *et al.*, 2006). With such invaluable long-term study systems, it will hopefully be possible to reveal whether, and when, selection can indeed lead to a hormonal arms race between mothers and other family members.

(2) Maternal disguising of contested signals

In the event that signaller costs are not sufficient to keep maternal hormones honest, how then can we explain their

persistence in the light of intrafamilial conflict? One possibility is that females are selected to use signals with a high level of ambiguity. Given that hormone receptors respond similarly regardless of a hormone's origin and intended function (Haig, 1996), a mother is safe to be dishonest if a hormone, alongside producing a signal of debated value or reliability to fathers and offspring, also signals something that is always of vital importance for all family members. Maternal hormones often exert pleiotropic effects on offspring development (Groothuis *et al.*, 2005*b*), and family members who dampen or oppose a dishonest hormone signal might simultaneously forego any pleiotropic advantages that the signal brings. While hormones that exert multiple effects on offspring development can certainly make it more difficult for fathers and offspring to oppose the maternal hormone regime, it is not yet clear whether such multiple effects have inescapable linkages, or whether mechanisms exist that allow selective uptake of a hormone signal in, for example, certain embryonic tissues (see Section III). Interestingly, if hormones indeed have multiple but inseparable effects, it may also be more difficult for mothers to use them to tailor offspring phenotype in the first place (Groothuis *et al.*, 2005*b*). Exactly how signal ambiguity affects the balance of competing interests under intrafamilial conflict is not yet known, but is likely to be important in understanding the role of maternal hormones.

To test the evolutionary stability of contested hormone signals that operate within a suite of other, pleiotropic, functions, we suggest that comparative studies might be highly informative. To date, a large number of experimental studies in birds, reptiles, fish and insects have manipulated embryo exposure to maternal hormones and tested the effect on offspring development (e.g. Schwabl, 1996; Uller & Olsson, 2003; Crook, Flatt & Smiseth, 2008; Gagliano & McCormick, 2009), and these studies can be used to explore patterns across different hormones. We predict that maternal hormones that influence traits across a range of systems (such as steroid hormones; Groothuis *et al.*, 2005*b*) will have a stronger influence on offspring phenotype, and on fathers' response to that phenotype, than those hormones that influence a small number of associated traits (such as glucagon; Braun & Sweazea, 2008). If the opposite result is found, this suggests that mothers themselves are limited in their ability to use pleiotropic hormones as a means of manipulating offspring phenotype, simply because such hormones change multiple offspring traits in concert and thus offer less opportunity for fine-tuning of the offspring phenotype.

(3) Maternal bet-hedging

A third way in which mothers might be selected to be dishonest is with regard to their own level of information. In this sense, we consider the degree of information, and hence the evolvability of a given maternal effect, in terms of the strength of autocorrelation between the environment experienced by the mother, and that later experienced by offspring (Burgess & Marshall, 2014; Kuijper, Johnstone & Townley, 2014). Put simply, maternal effects rely on some degree of environmental predictability, such that the resulting offspring phenotype matches its expected surroundings either after it is born or when it becomes independent (Reed *et al.*, 2010; Burgess & Marshall, 2014). However, no environment is entirely predictable and so a certain error rate on the part of the mother is largely unavoidable. One possible reason for the observed inter- and intra-brood variation in maternal hormone allocation is that mothers hedge their bets with regard to optimal offspring phenotype (Marshall & Uller, 2007; Kuijper & Johnstone, 2018). Assuming that there is no 'one-size-fits-all' offspring phenotype that can be employed when mothers cannot predict future conditions, the likely scenario is that mothers instead aim to produce a mix of offspring phenotypes in the hope that at least a certain percentage will match their environments. Although such a mother is not under selection to provide deliberately unreliable cues, other family members might be under selection to operate as if she is, since the cues she is using are nonetheless untrustworthy. Indeed, recent theoretical work by Kuijper & Johnstone (2018) suggests that maternal bet-hedging can have varying effects on the outcome of family conflict: when mothers benefit from producing a mix of phenotypes, they tend to gain an advantage in the underlying conflict, whereas when offspring benefit from the mother producing only one phenotype, the whole maternal hormone signal breaks down. Given that, under intrafamilial conflict, informative signals are more likely to evolve (Kuijper & Johnstone, 2018), it is important to consider the maternal error threshold above which family members might be selected to start opposing the hormone signal. For mothers, a mismatching risk of one offspring in ten may be acceptable; the fitness loss of one mismatched offspring may be outweighed by benefits accrued from nine well-matched ones. Assuming equal relatedness to all offspring, a one-in-ten error would also be acceptable to the father (although how the father's acceptable error rate might change in the presence of extra-pair paternity is an intriguing question). For the offspring, however, the same error rate may be unacceptable because the relative fitness impact of being mismatched is higher (as an offspring weighs its own success much higher than the success of each of the other nine offspring).

First and foremost, testing how intrafamilial conflict over maternal hormones varies according to maternal error rates requires a good understanding of the level of information available to mothers; in other words, researchers must determine the extent of autocorrelation between maternal and offspring environments (Groothuis & Taborsky, 2015; Groothuis *et al.*, 2019). Studies might thereafter follow two routes: researchers could either measure species-level variation in maternal error rates *per se*, or instead consider finer-scale variation in the factors that determine what level of maternal error other family members are likely to accept. In the case of the former, broad-scale comparative studies that measure differences in maternal hormone allocation and the response of other family members in highly

predictable *versus* highly unpredictable environments would be very useful. In the case of the latter, individual-based population studies are particularly suitable. For example, while a father might be selected to accept a one-in-ten maternal error rate if he can be sure that he sired the nine well-matched offspring, we might predict that the same error rate would generate between-parent conflict under high levels of extra-pair paternity because the male does not gain fitness benefits from well-matched offspring that are extra-pair. In both cases, theoretical modelling could elucidate predictions about how populations and individuals should respond to maternal error, which might then be verified in the next generation of empirical studies.

Investigating how variation in acceptable error rate might affect disagreements between family members could help to form predictions about when fathers and offspring should oppose maternal hormone signals. However, as is the case more generally, whether or not selection will ever favour family members who do not utilise the information cues in maternal signals depends on the risk of doing so in the event that the mother is correct. In the following section, we discuss how fathers and offspring can minimise that risk.

III. ADAPTIVE PLASTICITY IN RESPONSE TO MATERNAL HORMONE SIGNALS

Family members face a major problem: the potential cost of not listening to good advice. Information disparity, or the propensity for mothers to have more reliable cues than other family members with regards to offspring quality or need, affects the outcome of conflict in all sorts of family interactions (Kilner & Hinde, 2008) and has been proposed as a mechanism by which mothers might retain the upper hand in the conflict over maternal hormone allocation (Tobler & Smith, 2010). While fathers and offspring may have to accept this imbalance of power to some extent, it is important to explore scenarios in which family members can reclaim some control over the outcome of maternal hormone provisioning. Specifically, the balance of power between mothers, fathers and offspring depends on the ability of each party to obtain information about optimal offspring phenotype from sources other than the mother (English *et al.*, 2015).

(1) Plasticity in offspring: potential cues of developmental context

If offspring have no sources of environmental information other than what the mother provides with hormonal signals, there is little opportunity for them to exert their own fitness optima and mothers are able to selectively withhold information and temporarily win the conflict (Kuijper & Johnstone, 2018). However, if offspring are able to utilise other sources of environmental information, there is no reason for them to depend on maternally provided cues. So far, little empirical work has been conducted to determine how much offspring know about the outside world, but it has recently been suggested that the rest of the egg's contents may inform an embryo about its context (Groothuis *et al.*, 2019). While hormones themselves might not generally be costly for the mother to produce, other substances in the egg certainly are; mothers are probably limited in the amount of nutrition, carotenoids and other important micronutrients they can deposit depending on an egg's position in the laying sequence. Other aspects of resource availability in the mother's environment, or even her own physical condition or parasite load, might all affect prenatal allocation and thus provide unintended cues to embryos (Groothuis, Kumar & Hsu, 2020). In this 'information war' between mothers under selection to withhold cues from offspring and embryos under selection to find out about their environment, the arms race is brought to an end by escalating costs for the mother; she cannot afford to hide nutrition-based cues, and the offspring has the advantage.

If selection is to favour offspring who obtain outside information in this way, any variation in egg contents must be consistent such that it gives a reliable environmental cue across different nests within a population. Whether egg contents can indeed act in this way depends on the environmental factor in question. For example, mothers are often limited in resource availability later in the breeding season, leading to poorer nutrient provisioning to eggs (Arnold, Alisauskas & Ankney, 1991; Baur & Baur, 1996; Castro *et al.*, 2009). Whereas late-breeding mothers might benefit from increasing their deposition of hormones that produce fast-growing, rapidly independent offspring, offspring might benefit from growing at a slower pace and maximising the period of parental care to help compensate for poor environmental conditions. Under this conflict over the duration of parental care, offspring might evolve a simple response rule whereby their response to maternal hormones depends on egg nutrient content. However, in other contexts it is more difficult to see how offspring could win the information war. In many species, offspring are born into an age and/or size hierarchy and into broods of varying sex ratio; it should benefit a developing embryo to find out such social information because the effect of maternal hormones can vary across the laying sequence (Müller, Dijkstra & Groothuis, 2003) or be sex dependent (Rubolini *et al.*, 2005). However, it has been shown in birds that inter-brood variation in egg contents is as large, or larger, than that within broods (Ricklefs, 1984; Birkhead, 1985; Mentesana *et al.*, 2019); essentially, a given concentration of micronutrient may signal that an embryo is first-born in one brood, and last-born in another. At least in the case of egg nutritional contents, potential cues about social rank are probably too ambiguous for an offspring to interpret. For now, the mother wins the information war.

Egg contents are not the only type of cue that an offspring might use to gain outside information about its environment. For example, it has been demonstrated that crocodilian embryos produce vocalisations and also respond to those of their nest-mates (Vergne & Mathevon, 2008). Such communication can facilitate the sharing of information about

predation risk, such as demonstrated in yellow-legged gull *Larus michahellis* embryos (Noguera & Velando, 2019), or optimal hatching conditions, as is the case in pig-nosed turtles *Carettochelys insculpta* (Doody *et al.*, 2012). In taxa where parents brood or incubate the eggs [chiefly birds, but also pythons (Aubret *et al.*, 2005), monotreme mammals (Beard & Grigg, 2000), and several amphibian species (Stebbins & Cohen, 1995)], embryos may also gain considerable information from parental behaviours. For example, parental vocalisations produced while on the nest have been suggested to influence nestling phenotype in zebra finches *Taeniopygia guttata* (Mariette & Buchanan, 2016), although whether or not such vocalisations are indeed adaptive is still debated (McDiarmid, Naguib & Griffith, 2018, 2019; Mariette & Buchanan, 2019). Eggs experience different temperatures when a parent is incubating the clutch compared to when they are unattended (Boulton & Cassey, 2012), and presumably also variation in light and relative humidity. Such patterns during incubation can tell an offspring a lot about its social context: in species where incubation only begins after partial or full clutch completion, whether or not an egg experiences temperature changes straight away after oviposition can inform the embryo about whether it was early or late in the laying sequence. The information gathered through incubation patterns might make it possible for embryos to learn about their social context even when other cues, like egg nutrient concentration, are harder to interpret. It also seems likely that the timing and duration of incubation bouts hold an enormous wealth of other information that developing offspring might use to learn about the outside world. Parental incubation behaviours are highly flexible within populations and are known to vary in response to any number of environmental and social factors including predation risk (Fontaine & Martin, 2006), partner cooperation (Bulla *et al.*, 2013), local food availability (Eikenaar, Berg & Komdeur, 2003) and temperature (Conway & Martin, 2000; Cooper & Voss, 2013). Moreover, parents might be limited in the extent to which they can 'mask' the cues that incubation provides because selection already operates strongly to optimise the trade-off between the costs and benefits of parental care behaviours (Alonso-Alvarez & Velando, 2012). To our knowledge, no studies have yet tested whether parental incubation patterns might provide cues that guide offspring development and whether such cues might result in offspring gaining outside information that allows them to be plastic in their response to maternal hormones.

The most pressing question with regard to offspring ability to be plastic in their response to maternal hormones remains broad: how much do offspring 'know' about the outside world? Simple tests in controlled laboratory populations could begin to address this question. For example, does altering specific aspects of the pre- and post-oviposition environment affect offspring responses to hormones? As a further step, we recommend studies that explore the mechanisms by which offspring obtain this information; experiments that manipulate egg micronutrients or vary the incubation regime and subsequently test for differences in offspring responses to maternal hormones, both in terms of hormone metabolism and uptake and on the morphological or behavioural phenotype, would help to determine whether and how environmental cues can promote offspring plasticity in this regard.

As a last note on offspring plasticity, it is interesting to consider cases in which the balance of information, and thus power, is reversed; are offspring ever better informed about their context than their mother? In later stages of development, offspring may be able to access and share information with one another through vibrations and vocalisations, but there is one example of offspring power that might be consistent across the developmental period: offspring genotype. Let us take a gene for heat tolerance, where one allele is better equipped for dealing with heat stress and a second allele is less well equipped but outperforms the other in ambient temperatures. Mothers have no way of knowing which of their offspring will inherit which allele and so her hormone allocation will follow a bet-hedging strategy such that she hopes to optimise at least some of her offspring's phenotypes with their genetic condition and the environment they are likely to encounter. The offspring, on the other hand, may be able to infer which allele they are in possession of based on physiological responses to heat, and as such may be better off following a developmental trajectory different to that prescribed by the maternal hormone allocation. Whether or not such corrections would instigate a parent–offspring conflict, and which party would gain the upper hand, in turn depends on whether the optimum development trajectory differs for mothers and offspring (Kuijper & Johnstone, 2018).

(2) Plasticity in fathers: the role of information asymmetry

Unlike their offspring, who can already detect maternal hormones and potentially adjust their effect on its phenotype soon after conception (e.g. Kumar *et al.*, 2019*b*), a father has little information about emerging offspring phenotype until shortly before its hatching or birth, when embryos' movement and vocalisations are detectable from the outside (Vergne & Mathevon, 2008; Noguera & Velando, 2019). Fathers may have a direct effect on hormone allocation to embryos: there is evidence that seminal hormones can influence offspring phenotype (Lelono *et al.*, 2019), while fathers in species with external development may have direct opportunities to expose embryos to various substances of paternal origin (Keller-Costa *et al.*, 2015). However, fathers have no way of estimating emerging offspring phenotype, and hence whether this differs from their own optimum, until the offspring is born or hatches. The extent to which fathers may be selected to be plastic in their strategies before the offspring emerges would then depend on the dose–response curve of the contested hormone signal. Specifically, if the relationship between hormone allocation and the father's optimum for a given offspring trait is non-linear (for example, following a U-shaped curve), fathers cannot act before the offspring is born because they have no way of knowing where on the dose–

response curve the embryo already lies. If the relationship is linear, however, fathers can always be selected either to enhance or reduce a maternal hormonal signal because such actions will never push the offspring phenotype past a father's hypothetical optimum.

Given the general consensus that hormonal effects on offspring are often non-linear (Muriel *et al.*, 2015; Podmokła *et al.*, 2018; Groothuis *et al.*, 2019), plastic responses in fathers are likely to be largely postnatal. As a result, mechanistic weaponry with which fathers might be able to oppose maternal hormone signals are likely to be indirect, temporally limited and mainly under selection in species where fathers routinely interact with, and can influence the development of, offspring after their birth. However, fathers have one advantage over offspring in that they have much more information about the external environment, meaning that fathers in family-living species have the potential to respond plastically to maternal hormone signals and skew offspring phenotype away from the maternal optimum, should that optimum be different from his own. To determine whether this is the case, we must measure paternal response to maternally varied offspring phenotypes.

Happily, tests of how fathers respond to experimentally manipulated maternal hormone allocation are already available, albeit largely restricted to avian species (reviewed in Paquet & Smiseth, 2016). Such experiments are designed to test the 'differential allocation' (females invest more in offspring of desirable males; Sheldon, 2000) and 'manipulating androgens' (females use hormones to elicit extra parental care from the father; Moreno-Rueda, 2007) hypotheses. The results are mixed; in one study, fathers seem to blindly follow the instructions provided by maternal hormones (providing more care to more demanding offspring, for example; Noguera *et al.*, 2013) whereas in others, paternal strategy appears to be completely independent of maternal hormone allocation (Ruuskanen *et al.*, 2009; Barnett *et al.*, 2011). In the context of intrafamilial conflict, we can make clear predictions about this variation: fathers should respond positively to maternal hormone allocation if they lack sufficient information about the environment to reliably exert their own optimum offspring phenotype. While fathers arguably have greater access to outside information than offspring, they may still be less well informed than the mother (who can gain information at the very beginning of offspring development). Even under the manipulating androgens hypothesis, where males might otherwise be selected to disregard maternal hormone signals in the offspring (as the female uses these to manipulate male care), fathers who have no environmental information may still follow maternal hormone cues, whereas those with at least some outside information should not. Alternatively, fathers might adopt a strategy similar to offspring: initially follow the directions of maternal hormones and subsequently 'update' his strategy if and when more information about the environment becomes available.

A relatively simple way to test how the level of information available to fathers affects their response to maternal hormone allocation would be to artificially manipulate embryo hormones to create two different offspring phenotypes, and then experimentally vary paternal exposure to environmental context during crucial periods (such as just before conception, during the gestation/incubation periods, or shortly after birth/hatching). If outside information facilitates paternal plasticity in response to maternal hormones, we would expect that fathers who were separated from the rearing environment and subsequently returned once the offspring are past a given developmental stage to respond differently to the two offspring phenotypes. Fathers who were present in the rearing environment before conception and during embryo development have as much information about the environment as the mother; they should therefore base their response to offspring phenotype on their own perceived optimum for offspring development rather than simply following the instructions provided by the maternally programmed phenotype.

(3) Timing and mechanics of adaptive plastic responses to maternal hormones

It seems likely that both offspring and fathers have access to a certain degree of outside information that allows them to judge the use of, and plastically respond to, the phenotypic effects of a maternal hormone signal. But what might such a plastic response look like? One interesting concept to consider is variation in sensitive windows: periods in an organism's life where the phenotype is particularly sensitive to environmental conditions. Research has demonstrated that such windows extend well beyond early life and into adulthood (reviewed in Groothuis & Taborsky, 2015) and the timing and length of sensitive windows could themselves be plastic and variable between and within individuals (reviewed in Fawcett & Frankenhuis, 2015). Could family members who are more in conflict with the mother be selected to prolong certain sensitive windows to allow the possibility of updating sub-optimal information? Theoretically at least, there is evidence that offspring are under selection to balance the effects of early-life influences by constantly updating their phenotype (English *et al.*, 2016). Although yet to be tested, offspring might lengthen or bring forward a postnatal sensitive window if there is a strong possibility that their optimal phenotype is different from that programmed by the mother, thus allowing them to update their phenotype accordingly. There is also indirect evidence that arguably demonstrates such a tactic in fathers. In the parental care literature, it has been shown that parental investment decisions are ongoing rather than being fixed at the start of the reproductive attempt (Lendvai, Barta & Chastel, 2009; Lessells & McNamara, 2011); we could argue that such flexibility allows fathers to prolong the sensitive window for their parenting phenotype in order to supplement information they obtain through offspring phenotype (which is maternally controlled) with cues from maternal parenting behaviour and local environmental conditions.

Conversely, offspring might actively shorten their prenatal sensitive window in cases of conflict, essentially reducing the

timescale over which maternal hormones can affect development. A similar process might occur in uterine mammals, where it has been suggested that embryos filter out maternal stress hormones in order to reduce the sensitive window for postnatal maternal influences (Del Giudice, 2012). In the case of prenatal hormonal influences, offspring might bring forward the timing of switching to endogenous hormone production; the sooner an embryo is no longer reliant on maternal hormones for development, the sooner it can begin exerting its own fitness optimum. Such plasticity in the length of exposure to maternal hormones would be similar in its effect to emerging research suggesting that offspring metabolise maternal hormones, converting them into inactive or alternative forms to suit their personal optima better (Kumar *et al.*, 2018, 2019*b*). In both cases, the offspring is under selection to minimise maternal influence in the presence of parent–offspring conflict. Of course, there is also scope for mothers to counter-evolve strategies that bring hormonal effects back in line with the maternal interest. Like fathers, mothers could alter the duration of the sensitive window within which their parenting strategy is determined in order to respond flexibly to offspring-induced deviations from the mother's optimum offspring phenotype. Evolutionary arms races, such as those discussed above with respect to signal honesty, may therefore also be relevant with respect to plasticity.

Very little is known about variation in sensitive windows with regard to maternal hormones; we argue that this is a very promising area for future research. In offspring, testing for variation in the timing of the switch to endogenous hormone production or in the metabolism of maternal hormones would provide important information about whether or not offspring can indeed use such mechanisms to update their development trajectory, should it seem that their own fitness optimum lies on another trajectory than that of the mother. If this variation indeed exists, we suggest that researchers could test whether, within one population, differing metabolism and endogenous switch regimes are related to the factors suggestive of intrafamilial conflict that we have discussed above: arms races (Section II.1), ambiguity in hormone signalling (Section II.2) or environmental unpredictability (Section II.3). Such a within-population approach may also be useful with respect to paternal plasticity: we can expect fathers who benefit from the maternal hormone signal, either because they do not have access to outside information or because their optimal offspring phenotype is in line with that of the mother, to employ a more fixed parenting strategy than those who are under selection to disregard the maternal signal and update their behaviour as outside information becomes available. Lastly, it is important to note that studying variation in plasticity comes with several potential pitfalls: parental quality may confound the relationship between maternal effects and the offspring's environment (Engqvist & Reinhold, 2016, 2017), and selective mortality on certain plasticity-inducing alleles can create an upward bias in estimates of plasticity (Santos *et al.*, 2019). Whether and how fathers and offspring vary their response to maternal hormones is also likely to be dependent on the environmental and social context, which must be taken into account if within- and between-species variation is to be understood (Groothuis *et al.*, 2020). Accounting for such caveats is crucial when testing how plasticity of fathers and offspring evolves under intrafamilial conflict.

IV. INTERACTING PHENOTYPES

As stressed above, the honesty and reliability of mothers, along with the availability of outside information and the ability to be plastic, should predict how offspring and fathers respond to maternal hormones. However, it is important to acknowledge that such responses do not occur in isolation, but rather co-evolve. Understanding the influence that interacting individuals have on one another's optimal strategies is critical to the study of family life (Wolf, Brodie III & Moore, 1999; Alonzo & Klug, 2012), but is rarely taken into account in hormonal studies.

In the absence of conflict with the mother, embryos may still be selected to adjust their response to maternal hormones if there is strong sexual conflict between the parents. For example, let us assume that a mother has reliable information about the environment such that there is a relatively low error rate for a given embryo, and that this embryo benefits from listening to maternal hormones because they encourage a strongly competitive, demanding phenotype designed to maximise food acquisition [such as one that expresses intense begging behaviour in birds (Eising & Groothuis, 2003), or results in a more robust, immune-competent morph in locusts (Wilson *et al.*, 2002)]. In the first instance, we would predict a strong relationship between maternal hormones and emerging offspring phenotype (since it pays the offspring to listen to the mother). However, if the father gains little beneficial information from the maternal hormone signal, for example because it creates a demanding offspring that forces him into providing overly high levels of parental care, such a competitive offspring phenotype might not benefit the offspring at all. In fact, if the father does not reward the competitive phenotype with more food, the offspring gains little yet still bears the numerous costs that may be associated with heighted competitiveness, such as a trade-off with immune function (Groothuis *et al.*, 2005*a*) or increased exposure to oxidative stress (Haussmann *et al.*, 2011). As such, whether or not an offspring is selected to utilise maternal hormone signals depends on whether its father does the same (Fig. 1). This opens up the possibility that offspring are not employing a fixed strategy with regards to their handling of maternal hormone signals, but rather engage in a dynamic learning process after birth to determine the benefits of expressing hormone-induced traits. Evidence of learned behaviours has been shown with respect to offspring begging (e.g. Kedar *et al.*, 2000); we suggest that dynamic expression of strategies is likely to be more common within family interactions.

Similarly, whether fathers should respond to maternal hormones (in the form of offspring phenotype) depends on the level of conflict between the offspring and its mother. As explained above, we might predict that fathers benefit from maternal hormones if they have little outside information to make investment decisions (as seems to be the case in great tits *Parus major*; Hinde & Kilner, 2007). However, if offspring are under selection to dampen the effect of those hormones, the emerging offspring phenotype may not align with that intended by the mother. In such cases, the maternal signal becomes unreliable for the father because his indirect source, offspring phenotype, has been moved away from what the mother intended. In this scenario, any attempt by the mother to use hormones to manipulate her partner's parental investment [manipulating androgens hypothesis (Michl *et al.*, 2004; Paquet & Smiseth, 2016)] is therefore unlikely to succeed.

Lastly, how offspring respond to maternal hormones also depends on how an offspring's siblings, and hence its inclusive fitness, are affected as a result. One particularly well-studied example of where such sibling effects might impact intrafamilial conflict over offspring phenotype comes from eusocial insects, where conflicts over hormonally controlled caste differentiation are mediated by relatedness between sisters (Wenseleers, Ratnieks & Billen, 2003). In avian species, it is hypothesised that mothers vary hormone deposition within a brood in order either to consolidate or mitigate the effects of hatching asynchrony on competitive asymmetry within the brood (Müller *et al.*, 2004; Müller & Groothuis, 2012). If mothers use hormones to enhance competitive asymmetry and even facilitate brood reduction, as has been suggested (Müller & Groothuis, 2012), the question is whether there is indeed an evolutionary threshold for self-sacrifice; does it ever pay a weak offspring to follow the developmental trajectory as programmed by maternal hormones and accept its own demise for the sake of its siblings' fitness? Such a threshold does, theoretically, exist (O'Connor, 1978) and has been invoked to explain why last-born bird nestlings appear such willing victims of siblicide (Mock & Parker, 1998). By examining variation in sibling relatedness and expected inclusive fitness, researchers should be able to apply similar principles and techniques found in the more traditional sibling rivalry literature to determine whether, and when, ill-fated offspring should use maternal hormone signals for the sake of their kin.

Given the multi-directional nature of sibling interactions, the case is complicated further by the fact that potential costs and benefits of different offspring strategies will simultaneously depend on the strategies of the other, concurrent siblings. For example, first-hatched nestlings might benefit from obeying the instructions of maternal hormones to develop a less-competitive phenotype, if the inclusive fitness benefit of allowing their younger siblings to survive is sufficiently high. However, last-hatched siblings might employ strategies to lessen the influence of maternal hormones if, due to their small size or poor condition, the costs of expressing a competitive phenotype are excessive. In this scenario, both first- and last-hatched nestlings compete at a relatively low level (the first-hatched according to the mother's will, the last-hatched against it), and the result is that the entire brood receives less food from parents (Fig. 1). If last-hatched offspring are under selection to oppose maternal hormones, it will therefore pay first-hatched nestlings to do the same. As with many components of offspring competition, the best strategy with regard to maternal hormones is likely to be largely socially mediated (Smiseth, Scott & Andrews, 2011).

Performing studies that aim to test the above ideas about interacting phenotypes within the family will be highly complicated, as the expected fitness outcomes of all family members must be simultaneously taken into account. What this essentially requires is the manipulation of one level of conflict, such as changing the costs and benefits of care for mothers and fathers such that their optimum for offspring phenotype differs, and testing how this affects both the father's response to maternal hormones, and the offspring's response to the change in the father's strategy. This type of work is perhaps best suited to captive populations, but may also lend itself to theoretical work. For example, models might be constructed to test whether selection on offspring response to hormones produces a weaker association between maternal programming and offspring phenotype when sexual selection is very strong. Equally, simulations could reveal whether reduced paternal sensitivity to offspring phenotype spreads more easily through populations if sibling rivalry is acting strongly on offspring traits. Analyses that consider interactions between levels of conflict within the family might provide exciting insights into the paths that evolution takes under such complex, intrafamilial interactions.

V. SUGGESTIONS FOR FUTURE STUDIES

In this review, we have argued that the effect of maternal hormones on offspring phenotype, and the way that fathers respond to that phenotype, depends on the degree of intrafamilial conflict over offspring development. As shown in Fig. 1, when family members have conflicting interests, variation in maternal honesty, father and offspring plasticity, and interactions between the strategies of individual family members will determine the eventual outcome. Experimental, comparative and theoretical work is now needed to test whether these factors indeed influence the evolution and function of maternal hormones; we hope that the suggestions we have provided throughout this review and summarised in Table 1 will serve as inspiration for behavioural ecologists and endocrinologists alike.

One important additional avenue for research concerns the balance between cooperation and conflict between family members. While individual family members have unique fitness interests, these often overlap, leading to cooperation and even co-adaptation of traits (Smiseth *et al.*, 2008; Hinde, Johnstone & Kilner, 2010). The very fact that embryos have the necessary receptors for responding to maternal hormones before they begin to produce their own, endogenous

hormones, as well as the apparent developmental plasticity in offspring response to maternal hormones, is strong evidence for such co-adaptation. Several factors may determine the balance between co-adaptation and conflict, but these two processes are not mutually exclusive and one may even facilitate the other. For example, the evolution of adequate hormone receptors in embryos can be seen as cooperative, but variation in the presence and efficiency of these receptors is also the required first step towards the evolution of strategies in the offspring to modulate maternal effects (e.g. Kumar *et al.*, 2019*a*). While we advocate for a stronger emphasis on the role of intrafamilial conflict in driving the evolution of maternal effects, it is important to consider that cooperative and conflicting processes act concurrently (e.g. Patten *et al.*, 2014).

In this review we have only discussed the role of core family members (parents and their offspring). However, it is important to consider that not all families are so simple; across the majority of animal taxa, there are examples of social species in which the core unit also includes mature or partially mature offspring, brothers and sisters of the breeding pair, or even unrelated immigrants. The phenomenon of intrafamilial conflict over maternal hormone provisioning becomes even more complicated, and certainly more intriguing, when we account for the separate fitness interests of these extended family members. One very exciting avenue for future research would be to investigate how the presence of alloparents or 'helpers' that contribute towards the parental care of the offspring affects maternal hormone provisioning strategies. This is particularly interesting in the eusocial insects; here, there may be conflict over the worker phenotype that is prenatally programmed by the mother (Bourke & Ratnieks, 1999) but the presence of these workers may in turn influence how mothers allocate hormones to future offspring (as highlighted by Russell & Lummaa, 2009). Equally intriguing are cases of reproductive sharing or 'co-breeding', in which multiple females in a family group simultaneously produce young. Reproductive competition between mothers in the same generation (e.g. smooth-billed anis *Crotophaga sulcirostris*; Vehrencamp, 1977) or across generations (e.g. killer whales *Orcinus orca*; Croft *et al.*, 2017) may cause conflict over prenatal provisioning not only between mothers and offspring, but also between different mothers. In our discussion of interacting phenotypes, we highlight that social context is hugely important; this is likely to be even more so in species living in extended social groups.

The extensive evidence for maternal hormone effects across taxa suggests that they are likely to be an important selective force in all animals. The potential for offspring to influence this process is well accepted in mammals, where the placental transfer of hormones from mother to offspring and from offspring to mother offers a clear mechanism through which mother–offspring conflict can arise (Del Giudice, 2012). However, viviparity is not limited to mammals and maternal transfer of corticosterone has also been shown to occur across the placenta of grass skinks *Pseudemoia entrecasteauxii* (Itonaga, Wapstra & Jones, 2011). More broadly still, it is becoming increasingly clear that similar processes can occur across the extraembryonic membranes common to all amniotes (mammals, birds and reptiles), including oviparous species. So far, research into embryonic uptake and manipulation of maternal hormones across extraembryonic membranes in egg-laying species is restricted to birds (e.g. Albergotti *et al.*, 2009; Paitz *et al.*, 2010; Kumar *et al.*, 2019*b*), and we strongly encourage herpatologists in particular to explore these mechanisms in egg-laying reptiles. Mothers are also known to influence offspring phenotype *via* hormone allocation in the eggs of many fish species [e.g. antipredation behaviour (Giesing *et al.*, 2010); social competition (Burton *et al.*, 2011)] and similarly, hormonal control of the developmental trajectory of insects is well established [e.g. caste differentiation (Bortolotti, Duchateau & Sbrenna, 2001), offspring begging (Crook *et al.*, 2008) and secondary sexual traits (Emlen & Nijhout, 2001)], but to our knowledge there seems very little understanding of variation in offspring response to these maternal signals, especially outside of the better studied taxa. Insects lend themselves easily to experimental study and arguably exhibit the most diverse and extreme cases of morphological development in the animal kingdom; as such, this taxon may be particularly promising for future research. (e.g. Kapheim, 2017).

In addition to a broader taxonomic scope, further conceptual advances may be stimulated by expanding the concepts discussed here to other types of maternal effects. While this review focusses on hormones (a popular and well-understood maternal effect; Groothuis *et al.*, 2019), we see no reason to assume that intra-familial conflict would not manifest over all varieties of maternal effect. However, three properties are likely to determine what strategies can evolve in different family members. Firstly, there may be differences between signal-based maternal effects [e.g. epigenetic factors such as DNA methylation in the germline (Pickard *et al.*, 2001; Cooney, Dave & Wolff, 2002) and maternally derived microRNAs (e.g. McJunkin, 2018)] and resource-based maternal effects [-e.g. immune factors (Grindstaff, Brodie & Ketterson, 2003), yolk carotenoids (Surai, Speake & Sparks, 2001), maternally inherited symbionts (Chamberland *et al.*, 2017) and antimicrobial proteins (Saino *et al.*, 2002)]. Maternal production and transfer costs are likely to be much higher in resource-based effects, which may restrict the extent to which mothers can exaggerate their control over family members and limit the escalation of arms races between family members (as discussed in Section II). Second, prenatal and postnatal maternal effects place different temporal restrictions on the counter-strategies that fathers and offspring can evolve (Fig. 1). Postnatal maternal effects, such as *post-partum* transfer of hormones in mammalian milk, can have strong effects on offspring phenotype (Maestripieri & Mateo, 2009), but may be more susceptible to resistance by fathers and offspring because information about offspring phenotype and environmental conditions is more readily available after the offspring is born (see Section III). Third, it is important to distinguish between maternal effects that exert systemic influences on offspring phenotype, and those that affect single pathways or a narrow, related group of traits. Propagule size is a prominent

and widespread maternal effect that affects numerous components of offspring phenotype and success (Krist, 2011; Rollinson & Rowe, 2016); both mothers and other family members might be more restricted in how they can manipulate their respective allocation of and response to such pleiotropic maternal effects (see Section II). On the other hand, maternal effects on offspring mate preference [e.g. through maternal behaviour (Cameron, Fish & Meaney, 2008) or sexual imprinting (Kendrick *et al.*, 1998)] may present a more easily manipulated maternal effect because they concern one specific component of behaviour. Exploring the hypotheses we present here across different types of maternal effects might be the key to understanding how social selection pressures shape their form and function. Although we believe that empirical research is needed across all taxa to fill the gaps in our understanding of how maternal hormones operate under intrafamilial conflict, there is certainly scope for more theoretical work. Indeed, all of the hypotheses considered in this review could potentially be tested by modelling the responses of family members when factors like outside information, hormone signal ambiguity or social environments vary, as has already been done with respect to environmental unpredictability and maternal bet-hedging (Uller & Pen, 2011; Kuijper & Johnstone, 2018). Incorporating the field of maternal prenatal hormones, and indeed the field of maternal (and paternal) effects more generally, into a framework of thinking that explicitly considers intrafamilial conflict will help to explain the considerable variation in the apparent role of maternal hormones and, perhaps more importantly, facilitate cross-fertilisation of ideas between endocrinologists and researchers studying parental care, offspring competition and other components of family life.

VI. CONCLUSIONS

(1) Maternal effects on offspring phenotype, such as those transmitted through the allocation of maternally derived hormones to developing embryos, constitute a component of family life that is subject to the separate evolutionary interests of all family members. As such, we expect both the hormone-allocation strategies of mothers and the responses of other family members to hormone exposure to vary according to the extent of intrafamilial conflict.

(2) Under intrafamilial conflict, mothers may be selected to be dishonest in their hormone signalling. This can occur due to an arms race between family members, if offspring and fathers are selected to amplify/dampen a hormone signal to better fit their own developmental optima. Mothers might also be selected to disguise contested hormone signals within a suite of other signals that other family members benefit from receiving; this is one way in which the mother might maintain an advantage in the intrafamilial conflict. Alternatively, the maternal allocation of hormones may follow a simple bet-hedging strategy that maximises the mother's success in cases where the offspring's future environment is unpredictable; in this case, whether other family members should contest these hormones depends on the relative personal fitness cost of potential maternal errors. As highlighted in theoretical models, such situations may result in the breakdown of the maternal signal.

(3) As the first family member to influence maternal hormones, mothers have an advantage over other family members (who can only react to maternal hormones after they are allocated). However, plasticity in fathers and offspring may allow them to update their strategy at various points along the developmental trajectory, should it appear that the original hormone signal does not facilitate an optimal offspring phenotype. Such plasticity is not necessarily a source of conflict if it serves to correct maternal errors, and could be facilitated by the availability of alternative sources of information about the expected environment. Potential selection on the frequency and duration of sensitive windows within which offspring and their fathers can alter their strategies in response to maternal hormones is likely to influence the degree to which family members can contest maternal effects during offspring development.

(4) Intrafamilial conflicts are inherently complex because conflicts at one level of the family (for example, between the parents) can influence the extent and outcome of conflicts at another level (e.g. between offspring). The impact of interacting phenotypes on the evolution of maternal effects is currently unknown, but we argue that simultaneously considering multiple levels of conflict will provide a much clearer picture about how different family members are expected to respond to maternal hormones.

(5) Understanding how maternal effects operate within a framework of intrafamilial conflict requires a combination of experimental, comparative and theoretical work to test both existing hypotheses and new hypotheses presented here. In addition, it would be worthwhile to extend this treatment to broader forms of social interactions, such as those including extended family (for example, in group-living and cooperatively breeding species). Lastly, our knowledge of the mechanisms by which offspring can alter the influence of maternal hormones is largely limited to bird and mammal species; further work in other taxa is much needed to broaden our understanding of this likely ubiquitous component of reproductive biology.

VII. ACKNOWLEDGEMENTS

We would like to thank Sjouke Anne Kingma for useful discussions about the topics covered in this review, and three

anonymous referees for providing insightful suggestions. This work was supported by a NWO (Netherlands Organisation for Scientific Research) Veni Fellowship awarded to K.B. (grant number 016.veni.192.067).

VIII. REFERENCES

Albergotti, L. C., Hamlin, H. J., McCoy, M. W. & Guillette, L. J. Jr. (2009). Endocrine activity of extraembryonic membranes extends beyond placental amniotes. *PLoS One* **4**, e5452.

Alonso-Alvarez, C. & Velando, A. (2012). Benefits and costs of parental care. In *The Evolution of Parental Care* (eds M. Kölliker, P. T. Smiseth and N. J. Royle), pp. 40–60.Oxford University Press, Oxford.

Alonzo, S. H. & Klug, H. (2012). Paternity, maternity and parental care. In *The Evolution of Parental Care* (eds M. Kölliker, P. T. Smiseth and N. J. Royle), pp. 189–205.Oxford University Press, Oxford.

Arnold, T. W., Alisauskas, R. T. & Ankney, C. D. (1991). Egg composition of American coots in relation to habitat, year, laying date, clutch size, and supplemental feeding. *The Auk* **108**, 532–547.

Aubret, F., Bonnet, X., Shine, R. & Maumelat, S. (2005). Why do female ball pythons (*Python regius*) coil so tightly around their eggs? *Evolutionary Ecology Research* **7**, 743–758.

Barnett, C. A., Clairardin, S. G., Thompson, C. F. & Sakaluk, S. K. (2011). Turning a deaf ear: a test of the manipulating androgens hypothesis in house wrens. *Animal Behaviour* **81**, 113–120.

Baur, A. & Baur, B. (1996). Seasonal variation in size and nutrient content of eggs of the land snail *Arianta arbustorum*. *Invertebrate Reproduction and Development* **32**, 55–62.

Beard, L. & Grigg, G. C. (2000). Reproduction in the short-beaked echidna. *Proceedings of the Linnean Society of New South Wales* **122**, 89–99.

Bebbington, K. & Hatchwell, B. J. (2016). Coordinated parental provisioning is related to feeding rate and reproductive success in a songbird. *Behavioral Ecology* **27**, 652–659.

Bender, N., Heg, D., Hamilton, I. M., Bachar, Z., Taborsky, M. & Oliveira, R. F. (2006). The relationship between social status, behaviour, growth and steroids in male helpers and breeders of a cooperatively breeding cichlid. *Hormones and Behavior* **50**, 173–182.

Birkhead, M. (1985). Variation in egg quality and composition in the mallard *Anas platyrhynchos*. *Ibis* **127**, 467–475.

Bortolotti, L., Duchateau, M. J. & Sbrenna, G. (2001). Effect of juvenile hormone on caste determination and colony processes in the bumblebee *Bombus terrestris*. *Entomologia Experimentalis et Applicata* **101**, 143–158.

Boulton, R. L. & Cassey, P. (2012). How avian incubation behaviour influences egg surface temperatures: relationships with egg position, development and clutch size. *Journal of Avian Biology* **43**, 289–296.

Bourke, A. & Ratnieks, F. (1999). Kin conflict over caste determination in social hymenoptera. *Behavioral Ecology and Sociobiology* **46**, 287–297.

Braun, E. J. & Sweazea, K. L. (2008). Glucose regulation in birds. *Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology* **151**, 1–9.

Brown, S. L., Doroshov, S. I., Nunex, J. M., Hadley, C., Vaneenennaam, J., Nishioka, R. S. & Bern, H. A. (1988). Maternal triiodothyronine injections cause increases in swimbladder inflation and survival rates in larval striped bass, *Morone saxatilis*. *Journal of Experimental Zoology* **248**, 168–176.

Bulla, M., Valcu, M., Rutten, A. L. & Kempenaers, B. (2013). Biparental incubation patterns in a high-Arctic breeding shorebird: how do pairs divide their duties? *Behavioral Ecology* **25**, 152–164.

Burgess, S. C. & Marshall, D. J. (2014). Adaptive parental effects: the importance of estimating environmental predictability and offspring fitness appropriately. *Oikos* **123**, 769–776.

Burton, T., Hoogenboom, M. O., Armstrong, J. D., Groothuis, T. G. & Metcalfe, N. B. (2011). Egg hormones in a highly fecund vertebrate: do they influence offspring social structure in competitive conditions? *Functional Ecology* **25**, 1379–1388.

Cameron, N. M., Fish, E. W. & Meaney, M. J. (2008). Maternal influences on the sexual behavior and reproductive success of the female rat. *Hormones and Behavior* **54**, 178–184.

Castro, R. L., Claramunt, G., Krautz, M. C., Llanos-Rivera, A. & Moreno, P. (2009). Egg trait variation in anchoveta *Engraulis ringens*: a maternal response to changing environmental conditions in contrasting spawning habitats. *Marine Ecology Progress Series* **381**, 237–248.

Chamberland, V. F., Latijnhouwers, K. R., Huisman, J., Hartmann, A. C. & Vermeij, M. J. (2017). Costs and benefits of maternally inherited algal symbionts in coral larvae. *Proceedings of the Royal Society B: Biological Sciences* **284**, 20170852.

Chuong, E. B., Tong, W. & Hoekstra, H. E. (2010). Maternal–fetal conflict: rapidly evolving proteins in the rodent placenta. *Molecular Biology and Evolution* **27**, 1221–1225.

Collier, R. J., Doelger, S. G., Head, H. H., Thatcher, W. W. & Wilcox, C. J. (1982). Effects of heat stress during pregnancy on maternal hormone concentrations, calf birth weight and postpartum milk yield of Holstein cows. *Journal of Animal Science* **54**, 309–319.

Conway, C. J. & Martin, T. E. (2000). Effects of ambient temperature on avian incubation behavior. *Behavioral Ecology* **11**, 178–188.

Cooney, C. A., Dave, A. A. & Wolff, G. L. (2002). Maternal methyl supplements in mice affect epigenetic variation and DNA methylation of offspring. *Journal of Nutrition* **132**, 2393S–2400S.

Cooper, C. B. & Voss, M. A. (2013). Avian incubation patterns reflect temporal changes in developing clutches. *PLoS One* **8**, e65521.

Croft, D. P., Johnstone, R. A., Ellis, S., Nattrass, S., Franks, D. W., Brent, L. J. N., Mazzi, S., Balcomb, K. C., Ford, J. K. B. & Cant, M. A. (2017). Reproductive conflict and the evolution of menopause in killer whales. *Current Biology* **27**, 298–304.

Crook, T. C., Flatt, T. & Smiseth, P. T. (2008). Hormonal modulation of larval begging and growth in the burying beetle *Nicrophorus vespilloides*. *Animal Behaviour* **75**, 71–77.

Dantzer, B., Newman, A. E., Boonstra, R., Palme, R., Boutin, S., Humphries, M. M. & McAdam, A. G. (2013). Density triggers maternal hormones that increase adaptive offspring growth in a wild mammal. *Science* **340**, 1215–1217.

De Fraipont, M., Clobert, J., John-Alder, H. & Meylan, S. (2000). Increased pre-natal maternal corticosterone promotes philopatry of offspring in common lizards *Lacerta vivipara*. *Journal of Animal Ecology* **69**, 404–413.

Del Giudice, M. (2012). Fetal programming by maternal stress: insights from a conflict perspective. *Psychoneuroendocrinology* **37**, 1614–1629.

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

Doody, J. S., Stewart, B., Camacho, C. & Christian, K. (2012). Good vibrations? Sibling embryos expedite hatching in a turtle. *Animal Behaviour* **83**, 645–651.

Eikenaar, C., Berg, M. L. & Komdeur, J. (2003). Experimental evidence for the influence of food availability on incubation attendance and hatching asynchrony in the Australian reed warbler *Acrocephalus australis*. *Journal of Avian Biology* **34**, 419–427.

Eising, C. M., Eikenaar, C., Schwabl, H. & Groothuis, T. G. (2001). Maternal androgens in black-headed gull (*Larus ridibundus*) eggs: consequences for chick development. *Proceedings of the Royal Society of London. Series B: Biological Sciences* **268**, 839–846.

Eising, C. M. & Groothuis, T. G. (2003). Yolk androgens and begging behaviour in black-headed gull chicks: an experimental field study. *Animal Behaviour* **66**, 1027–1034.

Ekman, J., Eggers, S. & Griesser, M. (2002). Fighting to stay: the role of sibling rivalry for delayed dispersal. *Animal Behaviour* **64**, 453–459.

Elf, P. K. (2003). Yolk steroid hormones and sex determination in reptiles with TSD. *General and Comparative Endocrinology* **132**, 349–355.

Elf, P. K., Lang, J. W. & Fivizzani, A. J. (2002). Dynamics of yolk steroid hormones during development in a reptile with temperature-dependent sex determination. *General and Comparative Endocrinology* **127**, 34–39.

Emlen, D. J. & Nijhout, H. F. (2001). Hormonal control of male horn length dimorphism in *Onthophagus taurus* (Coleoptera: Scarabaeidae): a second critical period of sensitivity to juvenile hormone. *Journal of Insect Physiology* **47**, 1045–1054.

English, S., Fawcett, T. W., Higginson, A. D., Trimmer, P. C. & Uller, T. (2016). Adaptive use of information during growth can explain long-term effects of early life experiences. *The American Naturalist* **187**, 620–632.

English, S., Pen, I., Shea, N. & Uller, T. (2015). The information value of non-genetic inheritance in plants and animals. *PLoS One* **10**, e0116996.

Engqvist, L. & Reinhold, K. (2016). Adaptive trans-generational phenotypic plasticity and the lack of an experimental control in reciprocal match/mismatch experiments. *Methods in Ecology and Evolution* **7**, 1482–1488.

Engqvist, L. & Reinhold, K. (2017). Adaptive parental effects and how to estimate them: a comment to Bonduriansky and Crean. *Methods in Ecology and Evolution* **9**, 457–459.

Fawcett, T. W. & Frankenhuis, W. E. (2015). Adaptive explanations for sensitive windows in development. *Frontiers in Zoology* **12**, S3.

Fisher, D. A. (1996). Physiological variations in thyroid hormones: physiological and pathophysiological considerations. *Clinical Chemistry* **42**, 135–139.

Fontaine, J. J. & Martin, T. E. (2006). Parent birds assess nest predation risk and adjust their reproductive strategies. *Ecology Letters* **9**, 428–434.

Fowden, A. L., Comline, R. S. & Silver, M. (1984). Insulin secretion and carbohydrate metabolism during pregnancy in the mare. *Equine Veterinary Journal* **16**, 239–246.

Fresneau, N. & Müller, W. (2019). Flexible communication within bird families – the consequences of behavioral plasticity for parent-offspring co-adaptation. *Ecology and Evolution* **9**, 693–702.

Gagliano, M. & McCormick, M. I. (2009). Hormonally mediated maternal effects shape offspring survival potential in stressful environments. *Oecologia* **160**, 657–665.
Giesing, E. R., Suski, C. D., Warner, R. E. & Bell, A. M. (2010). Female sticklebacks transfer information via eggs: effects of maternal experience with predators on offspring. *Proceedings of the Royal Society B: Biological Sciences* **278**, 1753–1759.
Gil, D. (2003). Golden eggs: maternal manipulation of offspring phenotype by egg androgen in birds. *Ardeola* **50**, 281–294.
Godfray, H. C. (1995). Evolutionary theory of parent–offspring conflict. *Nature* **376**, 133–138.
González-Forero, M. (2014). An evolutionary resolution of manipulation conflict. *Evolution* **68**, 2038–2051.
Grindstaff, J. L., Brodie, E. D. & Ketterson, E. D. (2003). Immune function across generations: integrating mechanism and evolutionary process in maternal antibody transmission. *Proceedings of the Royal Society B: Biological Sciences* **270**, 2309–2319.
Groothuis, T. G., Eising, C. M., Dijkstra, C. & Müller, W. (2005a). Balancing between costs and benefits of maternal hormone deposition in avian eggs. *Biology Letters* **1**, 78–81.
Groothuis, T. G., Hsu, B. Y., Kumar, N. & Tschirren, B. (2019). Revisiting mechanisms and functions of prenatal hormone-mediated maternal effects using avian species as a model. *Philosophical Transactions of the Royal Society B: Biological Sciences* **374**, 20180115.
Groothuis, T. G., Kumar, N. & Hsu, B. Y. (2020). Explaining discrepancies in the study of maternal effects: the role of context and embryo. *Current Opinion in Behavioral Sciences* **36**, 185–192.
Groothuis, T. G., Müller, W., von Engelhardt, N., Carere, C. & Eising, C. (2005b). Maternal hormones as a tool to adjust offspring phenotype in avian species. *Neuroscience & Biobehavioral Reviews* **29**, 329–352.
Groothuis, T. G. & Schwabl, H. (2002). Determinants of within-and among-clutch variation in levels of maternal hormones in black-headed gull eggs. *Functional Ecology* **16**, 281–289.
Groothuis, T. G. & Schwabl, H. (2007). Hormone-mediated maternal effects in birds: mechanisms matter but what do we know of them? *Philosophical Transactions of the Royal Society B: Biological Sciences* **363**, 1647–1661.
Groothuis, T. G. & Taborsky, B. (2015). Introducing biological realism into the study of developmental plasticity in behaviour. *Frontiers in Zoology* **12**, S6.
Haig, D. (1996). Placental hormones, genomic imprinting, and maternal-fetal communication. *Journal of Evolutionary Biology* **9**, 357–380.
Hamilton, W. D. (1964). The genetical evolution of social behaviour II. *Journal of Theoretical Biology* **7**, 17–52.
Haussmann, M. F., Longenecker, A. S., Marchetto, N. M., Juliano, S. A. & Bowden, R. M. (2011). Embryonic exposure to corticosterone modifies the juvenile stress response, oxidative stress and telomere length. *Proceedings of the Royal Society B: Biological Sciences* **279**, 1447–1456.
Helle, S., Laaksonen, T. & Huitu, O. (2012). Sex-specific offspring growth according to maternal testosterone, corticosterone, and glucose levels. *Behavioral Ecology* **24**, 205–212.
Hinde, C. A. & Kilner, R. M. (2007). Negotiations within the family over the supply of parental care. *Proceedings of the Royal Society B: Biological Sciences* **274**, 53–60.
Hinde, C. A., Johnstone, R. A. & Kilner, R. M. (2010). Parent-offspring conflict and coadaptation. *Science* **327**, 1373–1376.
Hunt, J. & Simmons, L. W. (2000). Maternal and paternal effects on offspring phenotype in the dung beetle *Onthophagus taurus*. *Evolution* **54**, 936–941.
Hsu, B. Y., Dijkstra, C., Darras, V. M., de Vries, B. & Groothuis, T. G. (2016). Maternal adjustment or constraint: differential effects of food availability on maternal deposition of macro-nutrients, steroids and thyroid hormones in rock pigeon eggs. *Ecology and Evolution* **6**, 397–411.
Itonaga, K., Wapstra, E. & Jones, S. M. (2011). Evidence for placental transfer of maternal corticosterone in a viviparous lizard. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology* **160**, 184–189.
Kapheim, K. M. (2017). Nutritional, endocrine, and social influences on reproductive physiology at the origins of social behavior. *Current Opinion in Insect Science* **22**, 62–70.
Kedar, H., Rodriguez–Gironés, M. A., Yedvab, S., Winkler, D. W. & Lotem, A. (2000). Experimental evidence for offspring learning in parent–offspring communication. *Proceedings of the Royal Society of London. Series B: Biological Sciences* **267**, 1723–1727.
Keller-Costa, T., Canàrio, A. V. & Hubbard, P. C. (2015). Chemical communication in cichlids: a mini-review. *General and Comparative Endocrinology* **221**, 64–74.
Kendrick, K. M., Hinton, M. R., Atkins, K., Haupt, M. A. & Skinner, J. D. (1998). Mothers determine sexual preferences. *Nature* **395**, 229–230.
Kilner, R. & Johnstone, R. A. (1997). Begging the question: are offspring solicitation behaviours signals of need? *Trends in Ecology & Evolution* **12**, 11–15.
Kilner, R. M. & Hinde, C. A. (2008). Information warfare and parent–offspring conflict. *Advances in the Study of Behavior* **38**, 283–336.
Kingma, S. A., Bebbington, K., Hammers, M., Richardson, D. S. & Komdeur, J. (2016). Delayed dispersal and the costs and benefits of different routes to independent breeding in a cooperatively breeding bird. *Evolution* **70**, 2595–2610.
Kölliker, M., Boos, S., Wong, J. W., Röllin, L., Stucki, D., Raveh, S., Wu, M. & Meunier, J. (2015). Parent–offspring conflict and the genetic trade-offs shaping parental investment. *Nature Communications* **6**, 6850.
Krist, M. (2011). Egg size and offspring quality: a meta-analysis in birds. *Biological Reviews* **86**, 692–716.
Kuijper, B. & Johnstone, R. A. (2018). Maternal effects and parent–offspring conflict. *Evolution* **72**, 220–233.
Kuijper, B., Johnstone, R. A. & Townley, S. (2014). The evolution of multivariate maternal effects. *PLoS Computational Biology* **10**, e1003550.
Kumar, N., Lohrentz, A., Gahr, M. & Groothuis, T. G. (2019a). Steroid receptors and their regulation in avian extraembryonic membranes provide a novel substrate for hormone mediated maternal effects. *Scientific Reports* **9**, 11501.
Kumar, N., van Dam, A., Permentier, H., van Faassen, M., Kema, I., Gahr, M. & Groothuis, T. G. (2019b). Avian yolk androgens are metabolized rather than taken up by the embryo during the first days of incubation. *Journal of Experimental Biology* **222**, 193961.
Kumar, N., van Faassen, M., Kema, I., Gahr, M. & Groothuis, T. G. (2018). Early embryonic modification of maternal hormones differs systematically among embryos of different laying order: a study in birds. *General and Comparative Endocrinology* **269**, 53–59.
Lelono, A., Riedstra, B. & Groothuis, T. G. (2019). The relationship between male social status, ejaculate and circulating testosterone concentration and female yolk androgen transfer in red junglefowl (*Gallus gallus*). *Hormones and Behavior* **116**, 104580.
Lendvai, A. Z., Barta, Z. & Chastel, O. (2009). Conflict over parental care in house sparrows: do females use a negotiation rule? *Behavioral Ecology* **20**, 651–656.
Lessells, C. M. & McNamara, J. M. (2011). Sexual conflict over parental investment in repeated bouts: negotiation reduces overall care. *Proceedings of the Royal Society B: Biological Sciences* **279**, 1506–1514.
Lessells, C. M., Ruuskanen, S. & Schwabl, H. (2016). Yolk steroids in great tit *Parus major* eggs: variation and covariation between hormones and with environmental and parental factors. *Behavioral Ecology and Sociobiology* **70**, 843–856.
Libbrecht, R., Corona, M., Wende, F., Azevedo, D. O., Serrão, J. E. & Keller, L. (2013). Interplay between insulin signaling, juvenile hormone, and vitellogenin regulates maternal effects on polyphenism in ants. *Proceedings of the National Academy of Sciences* **110**, 11050–11055.
Maestripieri, D. & Mateo, J. M. (2009). The role of maternal effects in mammalian evolution and adaptation. In *Maternal Effects in Mammals* (eds D. Maestripieri and J. M. Mateo), pp. 1–10.The University of Chicago Press, Chicago.
Mariette, M. M. & Buchanan, K. L. (2016). Prenatal acoustic communication programs offspring for high posthatching temperatures in a songbird. *Science* **353**, 812–814.
Mariette, M. M. & Buchanan, K. L. (2019). Calling in the heat: the zebra finch incubation call depends on heat AND reproductive stage—a comment on McDiarmid et al. 2018. *Behavioral Ecology* **30**, e1–e3.
Marshall, D. J. & Uller, T. (2007). When is a maternal effect adaptive? *Oikos* **116**, 1957–1963.
McDiarmid, C. S., Naguib, M. & Griffith, S. C. (2018). Calling in the heat: the zebra finch "incubation call" depends on heat but not reproductive stage. *Behavioral Ecology* **29**, 1245–1254.
McDiarmid, C. S., Naguib, M. & Griffith, S. C. (2019). Zebra finch v-calls and the evidence for a signal: a response to comments on McDiarmid et al. *Behavioral Ecology* **30**, e4–e5.
McGhee, K. E. & Bell, A. M. (2014). Paternal care in a fish: epigenetics and fitness enhancing effects on offspring anxiety. *Proceedings of the Royal Society B: Biological Sciences* **281**, 20141146.
McJunkin, K. (2018). Maternal effects of microRNAs in early embryogenesis. *RNA Biology* **15**, 165–169.
Mentesana, L., Isaksson, C., Goymann, W., Andersson, M. N., Trappschuh, M. & Hau, M. (2019). Female variation in allocation of steroid hormones, antioxidants and fatty acids: a multilevel analysis in a wild passerine bird. *Journal of Avian Biology* **50**, e01859.
Michl, G., Török, J., Péczely, P., Garamszegi, L. Z. & Schwabl, H. (2004). Female collared flycatchers adjust yolk testosterone to male age, but not to attractiveness. *Behavioral Ecology* **16**, 383–388.
Miller, P. M., Gavrilets, S. & Rice, W. R. (2006). Sexual conflict via maternal-effect genes in ZW species. *Science* **312**, 73.
Mock, D. W. (1984). Siblicidal aggression and resource monopolization in birds. *Science* **225**, 731–733.
Mock, D. W. & Parker, G. A. (1998). Siblicide, family conflict and the evolutionary limits of selfishness. *Animal Behaviour* **56**, 1–10.

Moreno-Rueda, G. (2007). Yolk androgen deposition as a female tactic to manipulate paternal contribution. *Behavioral Ecology* **18**, 496–498.
Müller, M. & Groothuis, T. G. (2012). Within-clutch variation in yolk testosterone as an adaptive maternal effect to modulate avian sibling competition: evidence from a comparative study. *The American Naturalist* **181**, 125–136.
Müller, W., Dijkstra, C. & Groothuis, T. G. (2003). Inter-sexual differences in T-cell-mediated immunity of black-headed gull chicks (*Larus ridibundus*) depend on the hatching order. *Behavioral Ecology and Sociobiology* **55**, 80–86.
Müller, W., Eising, C. M., Dijkstra, C. & Groothuis, T. G. (2004). Within-clutch patterns of yolk testosterone vary with the onset of incubation in black-headed gulls. *Behavioral Ecology* **15**, 893–397.
Müller, W., Lessells, C. K., Korsten, P. & von Engelhardt, N. (2007). Manipulative signals in family conflict? On the function of maternal yolk hormones in birds. *The American Naturalist* **169**, E84–E96.
Muriel, J., Pérez-Rodríguez, L., Puerta, M. & Gil, D. (2015). Diverse dose–response effects of yolk androgens on embryo development and nestling growth in a wild passerine. *Journal of Experimental Biology* **218**, 2241–2249.
Noguera, J. C., Kim, S. Y. & Velando, A. (2013). Maternal testosterone influences a begging component that makes fathers work harder in chick provisioning. *Hormones and Behavior* **64**, 19–25.
Noguera, J. C. & Velando, A. (2019). Bird embryos perceive vibratory cues of predation risk from clutch mates. *Nature Ecology and Evolution* **3**, 1225–1232.
O'Connor, R. J. (1978). Brood reduction in birds: selection for fratricide, infanticide and suicide? *Animal Behaviour* **26**, 79–96.
Paitz, R. T. & Bowden, R. M. (2011). Biological activity of oestradiol sulphate in an oviparous amniote: implications for maternal steroid effects. *Proceedings of the Royal Society B: Biological Sciences* **278**, 2005–2010.
Paitz, R. T., Bowden, R. M. & Casto, J. M. (2010). Embryonic modulation of maternal steroids in European starlings (*Sturnus vulgaris*). *Proceedings of the Royal Society B: Biological Sciences* **278**, 99–106.
Paquet, M., Covas, R., Chastel, O., Parenteau, C. & Doutrelant, C. (2013). Maternal effects in relation to helper presence in the cooperatively breeding sociable weaver. *PLoS One* **8**, e59336.
Paquet, M. & Smiseth, P. T. (2016). Maternal effects as a mechanism for manipulating male care and resolving sexual conflict over care. *Behavioral Ecology* **27**, 685–694.
Parker, G. A., Royle, N. J. & Hartley, I. R. (2002). Intrafamilial conflict and parental investment: a synthesis. *Philosophical Transactions of the Royal Society B: Biological Sciences* **357**, 295–307.
Partecke, J. & Schwabl, H. (2008). Organizational effects of maternal testosterone on reproductive behavior of adult house sparrows. *Developmental Neurobiology* **68**, 1538–1548.
Patten, M. M., Ross, L., Curley, J. P., Queller, D. C., Bonduriansky, R. & Wolf, J. B. (2014). The evolution of genomic imprinting: theories, predictions and empirical tests. *Heredity* **113**, 119–128.
Pickard, B., Dean, W., Engemann, S., Bergmann, K., Fuermann, M., Jung, M., Reis, A., Allen, N., Reik, W. & Walter, J. (2001). Epigenetic targeting in the mouse zygote marks DNA for later methylation: a mechanism for maternal effects in development. *Mechanisms of Development* **103**, 35–47.
Podmokła, E., Drobniak, S. M. & Rutkowska, J. (2018). Chicken or egg? Outcomes of experimental manipulations of maternally transmitted hormones depend on administration method – a meta-analysis. *Biological Reviews* **93**, 1499–1517.
Råberg, L., Grahn, M., Hasselquist, D. & Svensson, E. (1998). On the adaptive significance of stress-induced immunosuppression. *Proceedings of the Royal Society B: Biological Sciences* **265**, 1637–1641.
Rebar, D., Bailey, N. W., Jarrett, B. J. & Kilner, R. M. (2020). An evolutionary switch from sibling rivalry to sibling cooperation, caused by a sustained loss of parental care. *Proceedings of the National Academy of Sciences* **117**, 2544–2550.
Reed, T. E., Waples, R. S., Schindler, D. E., Hard, J. J. & Kinnison, M. T. (2010). Phenotypic plasticity and population viability: the importance of environmental predictability. *Proceedings of the Royal Society B: Biological Sciences* **277**, 3391–3400.
Reed, W. L. & Vleck, C. M. (2001). Functional significance of variation in egg-yolk androgens in the American coot. *Oecologia* **128**, 164–171.
Rembold, H., Czoppelt, C. H. & Rao, P. J. (1974). Effect of juvenile hormone treatment on caste differentiation in the honeybee, *Apis mellifera*. *Journal of Insect Physiology* **20**, 1193–1202.
Ricklefs, R. E. (1984). Variation in the size and composition of eggs of the European starling. *The Condor* **86**, 1–6.
Rollinson, N. & Rowe, L. (2016). The positive correlation between maternal size and offspring size: fitting pieces of a life-history puzzle. *Biological Reviews* **91**, 1134–1148.
Royle, N. J., Hartley, I. R. & Parker, G. A. (2002). Sexual conflict reduces offspring fitness in zebra finches. *Nature* **416**, 733–736.
Rubolini, D., Romano, M., Martinelli, R. & Saino, N. (2005). Effects of elevated yolk testosterone levels on survival, growth and immunity of male and female yellow-legged gull chicks. *Behavioral Ecology and Sociobiology* **59**, 344–352.
Russell, A. F. & Lummaa, V. (2009). Maternal effects in cooperative breeders: from hymenopterans to humans. *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**, 1143–1167.
Ruuskanen, S., Doligez, B., Gustafsson, L. & Laaksonen, T. (2012). Long-term effects of yolk androgens on phenotype and parental feeding behavior in a wild passerine. *Behavioral Ecology and Sociobiology* **66**, 1201–1211.
Ruuskanen, S., Doligez, B., Tschirren, B., Pitala, N., Gustafsson, L., Groothuis, T. G. G. & Laaksonen, T. (2009). Yolk androgens do not appear to mediate sexual conflict over parental investment in the collared flycatcher *Ficedula albicollis*. *Hormones and Behavior* **55**, 514–519.
Ruuskanen, S. & Laaksonen, T. (2010). Yolk hormones have sex-specific long-term effects on behavior in the pied flycatcher (*Ficedula hypoleuca*). *Hormones and Behavior* **57**, 119–127.
Saino, N., Dall'ara, P., Martinelli, R. & Møller, A. P. (2002). Early maternal effects and antibacterial immune factors in the eggs, nestlings and adults of the barn swallow. *Journal of Evolutionary Biology* **15**, 735–743.
Sánchez-Tójar, A., Lagisz, M., Moran, N. P., Nakagawa, S., Noble, D. W. & Reinhold, K. (2020). The jury is still out regarding the generality of adaptive 'transgenerational' effects. *Ecology Letters* **23**, 1715–1718.
Sandell, M. I., Tobler, M. & Hasselquist, D. (2012). Yolk androgens and the development of avian immunity: an experiment in jackdaws (*Corvus monedula*). *Journal of Experimental Biology* **212**, 815–822.
Santos, M., Matos, M., Wang, S. P. & Althoff, D. M. (2019). Selection on structural allelic variation biases plasticity estimates. *Evolution* **73**, 1057–1062.
Schwabl, H. (1993). Yolk is a source of maternal testosterone for developing birds. *Proceedings of the National Academy of Sciences* **90**, 11446–11450.
Schwabl, H. (1996). Maternal testosterone in the avian egg enhances postnatal growth. *Comparative Biochemistry and Physiology Part A: Physiology* **114**, 271–276.
Schwabl, H. (1998). Mother knows best. *Natural History* **107**, 24–25.
Sheldon, B. C. (2000). Differential allocation: tests, mechanisms and implications. *Trends in Ecology and Evolution* **15**, 397–402.
Simpson, S. J. & Miller, G. A. (2007). Maternal effects on phase characteristics in the desert locust, *Schistocerca gregaria*: a review of current understanding. *Journal of Insect Physiology* **53**, 869–876.
Smiseth, P. T. & Moore, A. J. (2004). Signalling of hunger when offspring forage by both begging and self-feeding. *Animal Behaviour* **67**, 1083–1088.
Smiseth, P. T., Scott, M. P. & Andrews, C. (2011). Hormonal regulation of offspring begging and mediation of parent–offspring conflict. *Animal Behaviour* **81**, 507–517.
Smiseth, P. T., Wright, J. & Kölliker, M. (2008). Parent–offspring conflict and co-adaptation: behavioural ecology meets quantitative genetics. *Proceedings of the Royal Society B: Biological Sciences* **275**, 1823–1830.
Stamps, J., Clark, A., Kus, B. & Arrowood, P. (1987). The effects of parent and offspring gender on food allocation in budgerigars. *Behaviour* **101**, 177–199.
Stebbins, R. C. & Cohen, N. W. (1995). *A Natural History of Amphibians*. Princeton University Press, Princeton.
Stephens, P. A., Russell, A. F., Young, A. J., Sutherland, W. J. & Clutton-Brock, T. H. (2004). Dispersal, eviction, and conflict in meerkats (*Suricata suricatta*): an evolutionarily stable strategy model. *The American Naturalist* **165**, 120–135.
Stockley, P. & Parker, G. A. (2002). Life history consequences of mammal sibling rivalry. *Proceedings of the National Academy of Sciences* **99**, 12932–12937.
Surai, P. F., Speake, B. K. & Sparks, N. H. C. (2001). Carotenoids in avian nutrition and embryonic development. 2. Antioxidant properties and discrimination in embryonic tissues. *Journal of Poultry Science* **38**, 117–145.
Tawfik, A. I. & Sehnal, F. (2003). A role for ecdysteroids in the phase polymorphism of the desert locust. *Physiological Entomology* **28**, 19–24.
Tobler, M. & Smith, H. G. (2010). Mother–offspring conflicts, hormone signaling, and asymmetric ownership of information. *Behavioral Ecology* **21**, 893–897.
Trivers, R. L. (1972). Parental investment and sexual selection. In *Sexual Selection & The Descent of Man* (ed. B. Campbell), pp. 136–179.Aldine Publishing, Chicago.
Trivers, R. L. (1974). Parent-offspring conflict. *Integrative and Comparative Biology* **14**, 249–264.
Tschirren, B., Fitze, P. S. & Richner, H. (2006). Maternal modulation of natal dispersal in a passerine bird: an adaptive strategy to cope with parasitism? *The American Naturalist* **169**, 87–93.
Tschirren, B., Postma, E., Gustafsson, L., Groothuis, T. G. & Doligez, B. (2014). Natural selection acts in opposite ways on correlated hormonal mediators of prenatal maternal effects in a wild bird population. *Ecology Letters* **17**, 1310–1315.
Tschirren, B., Sendecka, J., Groothuis, T. G., Gustafsson, L. & Doligez, B. (2009). Heritable variation in maternal yolk hormone transfer in a wild bird population. *The American Naturalist* **174**, 557–564.

Tschirren, B., Ziegler, A. K., Canale, C. I., Okuliarová, M., Zeman, M. & Giraudeau, M. (2016). High yolk testosterone transfer is associated with an increased female metabolic rate. *Physiological and Biochemical Zoology* **89**, 448–452.

Uller, T., Nakagawa, S. & English, S. (2013). Weak evidence for anticipatory parental effects in plants and animals. *Journal of Evolutionary Biology* **26**, 2161–2170.

Uller, T. & Olsson, M. (2003). Prenatal exposure to testosterone increases ectoparasite susceptibility in the common lizard (*Lacerta vivipara*). *Proceedings of the Royal Society B: Biological Sciences* **270**, 1867–1870.

Uller, T. & Pen, I. (2011). A theoretical model of the evolution of maternal effects under parent–offspring conflict. *Evolution* **65**, 2075–2084.

Vehrencamp, S. L. (1977). Relative fecundity and parental effort in communally nesting anis, *Crotophaga sulcirostris*. *Science* **197**, 403–405.

Vergne, A. L. & Mathevon, N. (2008). Crocodile egg sounds signal hatching time. *Current Biology* **18**, R513–R514.

von Engelhardt, N. & Groothuis, T. G. (2011). Maternal hormones in avian eggs. In *Hormones and Reproduction of Vertebrates* (eds D. O. Norris and K. H. Lopez), pp. 91–127.Academic Press, Cambridge.

Walsh, S. W., Stanczyk, F. Z. & Novy, M. J. (1984). Daily hormonal changes in the maternal, fetal, and amniotic fluid compartments before parturition in a primate species. *The Journal of Clinical Endocrinology & Metabolism* **58**, 629–639.

Wenseleers, T., Ratnieks, F. L. & Billen, J. (2003). Caste fate conflict in swarm-founding social hymenoptera: an inclusive fitness analysis. *Journal of Evolutionary Biology* **16**, 647–658.

Wilson, K., Thomas, M. B., Blanford, S., Doggett, M., Simpson, S. J. & Moore, S. L. (2002). Coping with crowds: density-dependent disease resistance in desert locusts. *Proceedings of the National Academy of Sciences* **99**, 5471–5475.

Wolf, J. B., Brodie, E. D. III & Moore, A. J. (1999). Interacting phenotypes and the evolutionary process. II. Selection resulting from social interactions. *The American Naturalist* **153**, 254–266.

Yin, J., Zhou, M., Lin, Z., Li, Q. Q. & Zhang, Y. Y. (2019). Transgenerational effects benefit offspring across diverse environments: a meta-analysis in plants and animals. *Ecology Letters* **22**, 1976–1986.

Zahavi, A. (1975). Mate selection - a selection for a handicap. *Journal of Theoretical Biology* **53**, 205–214.

(*Received 31 March 2020; revised 30 April 2021; accepted 4 May 2021; published online 14 May 2021*)