Ecophysiology of egg rejection in hosts of avian brood parasites: new insights and perspectives

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Abstract

Egg rejection is the most effective and widespread defence used by host species to counteract the extreme fitness costs frequently imposed by obligate avian brood parasites. Yet, the proximate mechanisms underlying between- and within-individual variation in host responses remain poorly explored. Emerging evidence suggests that egg rejection is dependent on individual physiological state, and draws attention to the role of hormones as mediators of flexible antiparasitic responses. In this perspective article, I outline recent advances in our understanding of the proximate factors that mediate egg rejection. I also point out some areas where knowledge remains still lacking, especially those related to the development and maintenance of effective cognitive functions, the potential role of oxidative stress, immunological state and developmental stressors. I propose new hypotheses that stimulate future research on behavioural host responses towards brood parasitism.

Keywords: cognitive performance, condition-dependence, corticosterone, flexible responses, prolactin

Introduction

Avian brood parasitism is an important selective force shaping a range of morphological and behavioural adaptations in some bird populations (Rothstein 1990). Obligate brood parasites lay their eggs in the nest of host species thereby exploiting the parental care that they provide to their offspring (Roldán and Soler 2011). The degree of virulence of brood parasitic species (i.e., the fitness costs that brood parasitism inflicts on hosts) is largely determined by the behaviour of the parasitic chick, which often eliminates or outcompetes their foster siblings (Soler 2014; Moskát, Hauber and Louder 2017). In response, hosts have evolved successive lines of antiparasitic defences that operate at all stages of the reproductive cycle (Feeney, Welbergen and Langmore 2014; Soler 2014, 2017a). The recognition and subsequent rejection of brood parasitic eggs is the most effective antiparasitic behaviour (Feeney, Welbergen and Langmore 2014; Soler 2014), and it can be achieved through the ejection of the parasitic egg or, alternatively, the
abandonment of the complete clutch (Davies and Brooke 1989; Peer and Sealy 2004). Natural selection has favoured the evolution of mimetic and polymorphic eggs in many brood parasitic species, which makes egg recognition harder for hosts (Brooke and Davies 1988; Yang, Liang and Møller 2017). Thus, egg-rejection decisions involves sensory stimuli associations within the nest, and flexible adjustments that result from updating available information on the parasitism context (Ruiz-Raya and Soler 2020).

There is remarkable variation in egg rejection at all levels: individual, population and species (Soler 2014). In some cases, this variation reflects differences in the extent of local adaptation across host populations as a result of dissimilar coevolutionary histories with brood parasites (Thompson 2005; Møller and Soler 2012); but also, absence of egg rejection may be the consequence of recent parasitism (evolutionary-lag hypothesis; Rothstein 1990). Sometimes, the acceptance of foreign eggs might be adaptive, presumably when rates (or costs) of brood parasitism are very low (evolutionary-equilibrium hypothesis; Rothstein 1990; Krüger 2011). Finally, spatial and temporal differences in egg rejection can result from flexible adjustments in host responses according to the current risk of parasitism, previous experience, parasite retaliation or social context (reviewed in Ruiz-Raya and Soler 2017). Egg rejection is the outcome of a decision-making process driven by two key components: host cognitive abilities and the influence of conditional factors (Stokke, Moksnes and Røskaft 2005; Soler et al. 2012; Ruiz-Raya and Soler 2017).

Co-evolutionary processes that shape antiparasitic defences have received considerable research attention over the last decades (reviewed in Feeney et al. 2014; Soler 2017). Egg-rejection experiments have revealed that hosts rely on discordance mechanisms (i.e., the rejection the most dissimilar egg) and/or innate or learned templates of their own eggs’ appearance (template-based mechanism) to recognize and reject foreign eggs (Manna, Moskát and Hauber 2017). Studies on avian vision have provided detailed information about the specific visual cues and cognitive rules leading to egg rejection (e.g., Cassey et al. 2008; Spottiswoode and Stevens 2010; Stevens, Troscianko and Spottiswoode 2013), and have shown the existence of marked sensory constraints in the cognitive decision rules used by some host species (Hanley et al. 2017).

However, comparatively few studies have explored the proximate causes behind between- and within-individual variability in egg rejection. Cognitive functions depend on individual physiological state, so cognitive performance is expected to be largely determined by the organisms’ capacity to withstand environmental challenges (Buchanan, Grindstaff and Pravosudov 2013; Maille and Schradin 2016; CauchoiX, Chaine and Barragan-Jason 2020). It remains to be addressed whether the hosts’ capacity to identify and reject parasitic eggs is dependent on their physiological state (e.g., hormone levels, energetic and immunological states, oxidative status), or whether the development and maintenance of effective cognitive functions at adulthood can be constrained by adverse early-life conditions (Figure 1). Likewise, little is known about the physiological mechanisms that mediate the host propensity to egg rejection once the foreign egg has been identified. Hormones are prime candidates to mediate between- and within-individual variation in antiparasitic defences (Abolins-Abols and Hauber 2018). Until recently, a handful of pioneering studies had investigated the role of hormones in host-brood parasite systems (Briskie, Naugler and Leech 1994; Ibáñez-Álamo et al. 2012; Mark and Rubenstein 2013; Hahn et al. 2017; Ruiz-Raya et al. 2018; Anttonson et al. 2020; Scharf et al. 2021a, 2021b). Since, experimental evidence has confirmed that hormones indeed mediate host responses to brood parasitic eggs (Ruiz-Raya et al. submitted; Abolins-Abols and Hauber 2020a).

In this perspective article, I point out a number of physiological factors which could potentially impact host cognitive performance and explain individual variation in egg-rejection. I provide new hypotheses to stimulate future
research on condition-dependent responses to brood parasitic eggs. Finally, I discuss recent evidence on the endocrine mechanisms underlying egg rejection, with a special emphasis in prolactin, a pituitary hormone involved in the regulation of avian parental behaviour.

**Condition dependence and host cognitive performance**

As in many other ecological contexts, effective cognitive functions are crucial in avian brood parasitic systems. Parasitic cowbirds (*Molothrus sp.*), for example, show sex-specific hippocampal specialization that allow females to map the location of host nests and form dynamic memory libraries on available parasitism opportunities (Scardamaglia *et al.* 2017; Sherry and Guigueno 2019). On their part, hosts can assess the local parasitism risk by combining personal and social information about the presence of brood parasites, e.g., Gill and Sealy 2004; Welbergen and Davies 2012; Thorogood and Davies 2016; Tryjanowski *et al.* 2018. Rejecter hosts are able to acquire and integrate different informative signals that guide optimal rejection decisions, including the perceptual and cognitive process leading to egg recognition (Ruiz-Raya and Soler 2020). The host ability to identify and reject foreign eggs has evolved as the result of the strong selection pressures imposed by brood parasitism (Davies and Brooke 1989), and frequencies of potential rejecters and acceptors within host populations are known to have a strong genetic bases (Martin-Gálvez *et al.* 2006, 2007). Nonetheless, individual variation in cognitive performance can be caused by a number of non-cognitive factors, such as experience or current motivational state (Rowe and Healy 2014).

Environmentally induced changes in physiology can either enhance or impair cognitive functions and explain much of the individual cognitive variation within bird populations (Buchanan, Grindstaff and Pravosudov 2013; Maille and Schradin 2016). Beyond the genetic background, host cognitive abilities would therefore be expected to vary under different environmental context. The term *cognition* commonly refers to the acquisition, processing and use of valuable information from the environment, this being essential for biologically relevant processes such as learning, memory or decision making (Shettleworth 2010). Given the non-unitary nature of cognition, it is necessary a more precise delimitation of the specific cognitive ability that we are investigating (Rowe and Healy 2014). In egg-rejection studies, “host cognitive abilities” typically refers to the host capacity to recognize parasitic eggs so, accordingly, I will adopt this terminology throughout the present article to specifically refer to that cognitive task.

Despite the importance of effective egg-recognition abilities for rejecter species, the extent to which host cognitive performance depends on individual physiological state is unclear. There is evidence that some cognitive traits, such as problem-solving or learning, are condition dependent, with individuals in better physiological state showing more effective cognitive abilities (Bókony *et al.* 2014). In addition, the impact of early environmental conditions on the development of effective cognitive functions is another important factor that could explain individual differences in cognitive functions at adulthood (Buchanan, Grindstaff and Pravosudov 2013). We still do not know whether environmental challenges can induce shifts in host cognitive performance, or whether cognitive functions underlying antiparasitic defences are affected by developmental conditions in early life.

**Environmental stress, physiological state and cognition**
External (environmental) factors such as the social context or abiotic stressors, and the internal physiological state, are known to impact cognitive performance in vertebrates (Cauchoix et al. 2020). Brain tissues and neural processing are energetically expensive and require comparatively more energy than several other somatic tissues at rest (Laughlin 2001). Indeed, variations in body mass, or blood glucose levels, are associated with impaired cognitive abilities in birds, although the degree of impairment may depend on the intensity of food restriction and the energy status (Maille and Schradin 2016; Shaw 2017). Seasonal changes in food availability, body condition or glucose levels might determine the outcome of the egg-rejection process, although this link may not be straightforward. The presence of non-mimetic eggs in the nest is known to impact the energy stores of adult hosts, presumably as a consequence of energy mobilization to cope with brood parasitism (Ruiz-Raya et al. 2018). In some species, egg rejection probability appears to be negatively related to body mass at an intraspecific level (Abolins-Abols and Hauber 2020b), yet the effects of seasonal changes in body condition on host cognitive performance are still unclear.

Hormones could mediate the effects of environmental challenges on cognition and lead to reversible changes in host performance. Catecholamine and glucocorticoid responses to stress are known to influence cognitive functions, although their effects depend on the intensity and duration of stressors (McEwen and Sapolsky 1995; Maille and Schradin 2016). Moderate and short-term elevations in corticosteroid levels enhance cognition, while greater and sustained increases in circulating corticosteroids can lead to cognitive impairment (Pravosudov 2003; Lupien et al. 2009). Individual differences in baseline glucocorticoids, or changes in glucocorticoid levels in response to environmental challenges, has been hypothesized to impact the host ability to recognize and reject parasitic eggs. In American robins Turdus migratorius, an occasional host of the brown-headed cowbird Molothrus ater, the probability of egg rejection has been shown to be negatively related to plasma corticosterone (CORT) levels (Abolins-Abols and Hauber 2020b). In other experimental study, the authors found that the suppression of CORT synthesis through mitotane injections increased the probability of egg acceptance by female robins (Abolins-Abols and Hauber 2020a). These results strongly suggest that glucocorticoids mediate behavioural responses to parasitic eggs and bring new insights into the endocrine regulation of egg rejection. Future studies should determine whether variations in CORT levels impact host cognitive performance or, instead, modify maternal behaviours in the nest (e.g., maternal attachment to eggs; Figure 1).

Other aspects of host physiology, such as oxidative status, could mediate life-history trade-offs linked to individual cognitive performance. The relative abundance of pro-oxidant and antioxidant agents occurring in cells determines the individual reproductive potential, gradual deterioration of bodyline functions and lifespan (Monaghan, Metcalfe and Torres 2009). Specifically, prolonged exposure to oxidative stress is expected to lead to brain damage and cognitive impairment (Liu et al. 2003; Dröge and Schipper 2007). In birds, plasma levels of enzymatic antioxidant are positively associated with enhanced cognitive functions (Bókony et al. 2014), while the increased production of plasma lipid peroxidation markers (MDA), and the simultaneous decrease in antioxidants (SOD, GPX), are linked to cognitive impairment (Padurariu et al. 2010). One might simplistically predict that those individuals showing evidence of accumulated oxidative damage (e.g., increased plasma MDA levels), or lower antioxidant levels, will show poorer sensory-cognitive abilities to recognize and reject parasitic eggs. Likely, the study of different components of oxidative stress will allow to obtain a broader view of individual oxidative status. Among the most feasible measurements are enzymatic and non-enzymatic antioxidant defences, total antioxidant capacity (TAC), and measurements of oxidative damage to proteins, lipids and DNA (Monaghan, Metcalfe and Torres 2009).
Oxidative stress has been suggested to underly life-history trade-offs associated with reproductive costs (Monaghan, Metcalfe and Torres 2009; Metcalfe and Monaghan 2013). Further experimental investigation is needed to establish whether there is any link between parental investment and host cognitive performance. Evolutionary cost-benefit theory predicts that hosts investing more in reproduction will be more likely to reject brood parasitic eggs (the “maternal investment” hypothesis; (Hauber et al. 2020)). However, if greater reproductive effort does lead to increased oxidative damage to the soma (and impaired brain functions), then hosts investing more in their clutch (e.g., larger eggs and broods, earlier onset of laying, post-hatching investment) might exhibit impaired cognitive abilities. Correlational evidence suggests that hosts with smaller clutches are more likely to reject brood parasitic eggs (Abolins-Abols and Hauber 2020b), although further studies are needed to establish a causal link between parental effort and host cognitive performance.

Similarly, given the link between aging, oxidative stress and cognitive decline (Golden, Hinerfeld and Melov 2002; Dröge and Schipper 2007), it could be predicted the existence of age-dependent responses to brood parasitism, with old individuals showing impaired cognitive performance. Current evidence, however, suggests that egg-rejection abilities do not decrease over the life. In the great-spotted cuckoo *Clamator glandarius* - magpie *Pica pica* system, longer-lived magpies are more likely to reject cuckoo eggs, and brood parasites are expected to benefit from exploiting young and naïve individuals (Martínez et al. 2020). Importantly, long-lived hosts might show reduced cumulative oxidative damage, or physiological mechanisms to resist oxidative stress. As the development and maintenance of cognitive skills is costly, it should be only favoured when individuals obtain fitness benefits from effective cognitive abilities (Morand-Ferron, Cole and Quinn 2016). Natural selection should select for physiological mechanisms protecting essential cognitive functions against the detrimental effects of seasonal environmental challenges (cognitive resilience; Buchanan, Grindstaff and Pravosudov 2013). Given the high fitness costs linked to brood parasitism, resilience mechanisms would reduce the brain susceptibility to stress and allow rejecter species to maintain an effective cognitive performance. These mechanisms might involve the modulation of glucocorticoid receptor densities, or sensitivity, protecting essential cognitive abilities against changes in glucocorticoids levels. Also, the detrimental effects of oxidative stress could be buffered through the modulation of circulating antioxidants (Buchanan, Grindstaff and Pravosudov 2013).

Future studies should explore potential trade-offs between cognitive performance and the immune system in the context of avian brood parasitism. Pathogens have a negative impact on cognitive functions in vertebrates, including humans (Jukes et al. 2002; Binning et al. 2018), and parasite infection is known to impact cognitive performance in birds (Ducatez et al. 2020). Brood parasitism increases bacterial load in parasitized nests, (Soler et al. 2011); however, whether parasite infection in adult hosts impact their cognitive performance is unknown. Equally, recent research has highlighted the role of the gut microbiome as a potential driver of individual cognitive variation in natural populations. Avian gut microbiota is affected by a number of intrinsic (e.g., age, sex, diet, behaviour) and extrinsic factors (e.g., environment, resources) (Grond et al. 2018), and the gut-brain axis seems to play a key role in brain functions and the maintenance of effective cognitive abilities (Davidson et al. 2018). Gut microbiota has been characterized in different brood parasite-hosts systems (Ruiz-Rodríguez et al. 2009; Lee et al. 2020; Schmiedova et al. 2020), but the link between gut microbiome and host cognitive performance at adulthood has not yet been explored. Experimental manipulations of gut microbial communities at both perinatal environments and adulthood would likely help to disentangle the potential link between hosts performance and gut microbiota (Davidson et al. 2018).
Developmental conditions and cognitive performance

Early-life events induce anatomical, behavioural and physiological changes in developmental trajectories (West-Eberhard 2003). Environmental conditions experienced during early development shape individual phenotypes and have long-lasting effects on cognitive functions (Buchanan, Grindstaff and Pravosudov 2013). Early-life stress has deleterious effects on a number of cognition-dependent traits in birds, such as spatial memory or vocal learning (Buchanan et al. 2003; Spencer et al. 2003; Pravosudov 2009). Nutritional deficits and chronic exposure to glucocorticoids during postnatal development impair cognitive performance at adulthood (Kitaysky et al. 2003; Pravosudov, Lavenex and Omanska 2005; Lucassen et al. 2013). In some brood parasitic systems, host nestlings are exposed to important nutritional and physiological stressors. Non-evictor parasitic young are preferentially fed by their foster parents (Soler et al. 1995; Lorenzana and Sealy 1999; Soler 2017b) and outcompete host nestlings (Soler 2017b), not only for quantity of food, but also for high-quality food, thereby imposing nutritional restrictions on host offspring (Ladin et al. 2015). Host nestlings may respond by increasing the intensity of begging signals to compete with their parasitic nestmates (Rivers, Loughin and Rothstein 2010), which could lead to immunological (Moreno-Rueda 2010) and oxidative costs (Noguera et al. 2010). Interestingly, Ibáñez-Álamo et al. (2012) found that magpie nestlings reared together with great-spotted cuckoos had higher CORT levels than magpies from unparasitized nests, which indicates that sharing the nest with parasitic chicks is an important physiological stressor for developing hosts. (but see (Scharf et al. 2021b)).

Brood parasitism is associated with faster growth rate and shorter nestling periods in hosts, these effects being more pronounced in host species showing higher sibling competition (Remes 2006). Faster growth rates may cause the loss of cognitive abilities in adulthood (Fisher, Nager and Monaghan 2006), so the accelerated nestling growth might impact the host ability to respond against brood parasitism in adulthood, especially in small-sized hosts in which sibling competition is more intense. Alternatively, it could be predicted the existence of resilience mechanisms protecting hosts from the negative effects of early-life stressors. Brood parasitism is known to result in carry-over effects on future reproductive effort in adult hosts (Mark and Rubenstein 2013); however, whether sharing the nest with parasitic chicks has long-term effects on host cognitive performance is still unknown.

Beyond egg recognition: endocrine regulation of flexible host responses

The outcome of the egg-rejection process is largely determined by intrinsic sensory and perceptual abilities, but egg rejection is likewise conditioned by a number of environmental and life-history factors, such as the risk of parasitism or parasite retaliation, the moment of the breeding season, or previous experience (reviewed in Ruiz-Raya and Soler 2017). This means that egg rejection does not only depend on the host ability to recognize foreign eggs, but also on its propensity to reject them. Several hypotheses have been proposed to explain how hormones mediate the host propensity to either reject or accept foreign eggs, paving the way for clear predictions about proximate factors underlying flexible changes in rejection responses. Abolins-Abols & Hauber (2018) provided a comprehensive overview on the endocrine mechanisms potentially underlying antiparasitic behaviours at different stages of the breeding cycle. The authors argue that the propensity to accept or reject any egg in the nest is mediated by major hormones involved in the regulation of avian parental behaviour. For instance, endocrine pathways involved in the suppression of maternal behaviour might regulate the ejection of parasitic eggs or nest desertion decisions. While empirical evidence on the endocrine regulation
of egg rejection is still scarce, recent experimental studies have provided strong support for the role of certain hormones (e.g., prolactin, corticosterone) as mediators of host responses to brood parasitism. Below, I outline recent advances and future avenues in understanding the hormonal bases of egg rejection.

Prolactin is a pituitary hormone that mediates major aspects of avian parental care, including ultimate decisions such as the abandonment of eggs and chicks (Angelier and Chastel 2009). Plasma prolactin levels are positively related to the initiation and maintenance of associative parental behaviour in birds (e.g., egg incubation, post-hatching parental care), whereas decreasing prolactin concentrations are often linked to breeding failure (Angelier and Chastel 2009; Angelier et al. 2016; Smiley 2019). Prolactin is thought to mediate those antiparasitic defences that entail the suppression of affiliative parental behaviours, such as the ejection of foreign eggs and the complete abandonment of parasitized clutches (Abolins-Abols and Hauber 2018; Ruiz-Raya and Soler 2020). Recent studies have revealed that the experimental reduction of plasma prolactin concentrations through bromocriptine implants increases the probabilities of egg rejection in Eurasian blackbirds (Turdus merula) (Ruiz-Raya et al. submitted). Notably, bromocriptine treated blackbirds showed higher ejection rates and faster rejection decisions (without incurring in recognition errors) in response to mimetic model eggs. Experimental evidence confirms that decreasing prolactin levels facilitate egg-ejection decisions, yet future research should investigate whether prolactin can somehow impact cognitive performance in other host species.

Circulating prolactin levels often decrease in response to environmental stressors (Angelier and Chastel 2009; Angelier et al. 2016). This prolactin response to stress may help to disentangle the proximate mechanisms mediating flexible responses to parasitic eggs. Cues of brood parasitism, such as the presence of adult parasites or social information on parasitism risk, might lead to changes in plasma prolactin levels and thus facilitate egg-rejection decisions. While the presence of non-mimetic eggs in the nest does not seem to modify prolactin levels in adult hosts, experimentally parasitized individuals exhibit a more robust prolactin responses to standardized stressors (Ruiz-Raya et al. 2018). This suggests that prolactin response may be more complex than expected and require multiple cues of parasitism (e.g., a parasitic egg and the presence of the adult parasite). Experimental studies assessing the host’s prolactin response to variable risk of parasitism will be essential to understand the role of endocrine flexibility in mediating antiparasitic defences.

Prolactin regulation of antiparasitic defences may be especially important in host species that use nest abandonment as main rejection mechanism, as is the cases of many brown-headed cowbird hosts (Hosoi and Rothstein 2000). In such cases, individual differences in circulating prolactin could determine the host propensity to abandon the nests. In medium-sized hosts, the experimental reduction of plasma prolactin concentrations does not affect the probability of nest abandonment in response to experimental parasitism (Ruiz-Raya et al. submitted). However, it is noteworthy that that medium-sized ejectors might not use nest desertion as a genuine response to brood parasitism (Soler et al. 2015; but see Hosoi and Rothstein 2000). The role of prolactin in mediating nest desertion responses should be further explored in small-sized hosts which are not able to reject parasitic eggs by grasping or puncturing them. In addition, it should be noted that the adaptive value of nest desertion is likely determined by life-history traits. As nest abandonment would be adaptive only when the value of current reproduction is low (e.g., lower clutch sizes) (Servedio and Hauber 2006), prolactin might mediate optimal rejection decisions according to the fitness payoffs of alternative host responses. Prolactin decrease in response to environmental stressors can be down-regulated to maintain parental care when current reproduction has important fitness value (Angelier and Chastel 2009; Angelier et al. 2016).
Other hormones such as testosterone and CORT, which both play key roles as mediators of parental behaviour in bird species (Schoech et al. 1998; Bókony et al. 2009), could likewise impact the host propensity to egg rejection (Figure 1). CORT might directly affect host cognitive performance (see above), but also impact the host behaviour once the parasitic egg has been recognized. CORT responsiveness to environmental factors has been suggested to be an important mechanism mediating flexible host responses to brood parasitism (Abolins-Abols and Hauber 2018). For example, raising CORT levels in response to brood parasitism (or high risk of brood parasitism) could suppress affiliative parental behaviour and facilitate rejection decisions. As seen above, decreasing CORT levels are known to reduce egg-rejection responses in American robins (Abolins-Abols and Hauber 2020a). Experimental parasitism causes an increase in CORT levels in host species with fine-tuned recognition abilities (Ruiz-Raya et al. 2018); however, accepter species show unaffected plasma CORT levels in response to foreign eggs (Scharf et al. 2021). These results indicate that changes in plasma CORT elicited by parasitic eggs orchestrate effective responses to brood parasitism, a process that will likely depend on egg mimicry and host recognition abilities. Finally, given that prolactin can act sequentially or interact with other hormones such as CORT (Angelier et al. 2016), further studies should consider that the effect of CORT might depend on the circulating levels of prolactin.

Concluding remarks

The host abilities to recognize and reject brood parasitic eggs have been widely explored in different brood parasitic systems, which has brought us closer to understanding the co-evolutionary forces that shape avian brood parasite-host interactions. Despite this, we still lack knowledge about the proximate mechanisms underlying individual variation in egg rejection. In this article, I propose that host cognitive performance and rejection decisions are dependent on individual physiological condition (e.g., hormone levels, oxidative status, immunological and energetic state). Individual differences in cognitive performance would be determined not only by genetic inheritance, but other factors, such as developmental history (cognitive plasticity), social context, or the effects of environmental stressors, should also be considered (Boogert et al. 2018; Cauchoix, Chaine and Barragan-Jason 2020). Future studies should explore how changes in host physiology across early-life and adult environments affect cognitive performance in egg-rejection experiments. Understanding how physiology determines individual variation in host performance will likely be challenging since sensory, perception and cognitive performance cannot be directly observed. Egg-rejection rates alone will not allow to separate the perceptual and conditional components of egg rejection, so future research will benefit from the use of both behavioural (e.g., egg-touching behaviour) and physiological (e.g., hormone variation) proxies of egg recognition to assess cognitive performance separately from host responses (Ruiz-Raya and Soler 2020).

Even though individual differences in cognition are well documented among vertebrate taxa (Thornton and Lukas 2012), the extent to which these variations determine the reproductive fitness in wild populations remains challenging (Morand-Ferron, Cole and Quinn 2016). In avian brood parasitic systems, fine-tuned cognitive abilities (e.g., strong egg-recognition abilities) are essential to avoid the frequently high fitness costs inflicted by brood parasitism, so physiological mechanisms favouring cognitive resilience would be expected to be found in host species. These resilience mechanisms might operate by modulating tissue sensitivity to stress (e.g., glucocorticoids receptor density), or protecting brain from the detrimental effects of oxidative damage (e.g., antioxidant defences).
The evidence reviewed here strongly suggests that egg rejection is mediated by endocrine pathways involved in the regulation of parental behaviour, yet additional research is needed to unravel whether the endocrine regulation of host responses is associated with variations in cognitive performance or changes in associative maternal behaviours, as seems to be the case with prolactin. Future studies should explore whether different hormones (e.g., prolactin, corticosterone, testosterone) interact to mediate egg-rejection behaviours. Finally, besides changes in plasma hormone concentration, the endocrine regulation of egg rejection will likely involve different components of endocrine networks such hormone receptor densities or carrier proteins (Breuner, Delehanty and Boonstra 2012; Rosvall 2013). Studying the physiological mechanisms mediating egg-rejection behaviour will bring new insights into the causes of individual variation in antiparasitic host defences and shed light on the proximate factors underlying flexible host responses to brood parasitism.

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Figure 1. Potential physiological mechanisms mediating egg-rejection. Egg rejection is a multi-stage process that requires effective perceptual and cognitive functions (cognitive performance), as well as the accurate integration of external cues of parasitism and the assessment of potential costs associated with the response (decision). Besides the genetic background, cognitive performance is determined by individual physiological state (cognitive flexibility; e.g., hormone levels, immunological state and oxidative status) and developmental conditions (developmental cognitive plasticity). Main hormones involved in the regulation of parental care, such as prolactin, CORT and testosterone, could also mediate changes in incubation and associative maternal behaviors in the nest, thereby impacting the probabilities of egg rejection.