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Defences against brood parasites from a social immunity perspective

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Summary

The widespread evolution of parasitic interactions has driven all multicellular organisms to evolve defence systems adapted to reduce their costs, whether the parasites they encounter are the "*classic parasites*" that feed on the individual, or "*brood parasites*" that usurp parental care. Many parallels have been drawn between defences deployed against both types of parasite, but typically, whilst defences against classic parasites have been selected to protect survival, those against brood parasites have been selected to protect the parent's inclusive fitness, suggesting that the selection pressures they impose are fundamentally different. However, there is another class of defences against classic parasites that have specifically been selected to protect an individual's inclusive fitness, known as "*social immunity*". Social immune responses include the anti-parasite defences typically provided for others in kin-structured groups, such as the antifungal secretions produced by termite workers to protect the brood. Defences against brood parasites, therefore, are more closely aligned with social immune responses. Much like social immunity, host defences against brood parasitism are employed by a donor (a parent) for the benefit of one or more recipients (typically kin), and as with social defences against classic parasites, defences have therefore evolved to protect the donor's inclusive fitness, not the survival or ultimately the fitness of individual recipients This can lead to severe conflicts between the different parties, whose interests are not always aligned. Here, we consider defences against brood parasitism in the light of social immunity, at different stages of parasite encounter, addressing where conflicts occur and how they might be resolved. We finish with considering how this approach could help us to address longstanding questions in our understanding of brood parasitism.

1. Introduction

Parasitic interactions, where one organism utilizes the resources of another to the detriment of the host, are so ubiquitous that all individuals can be expected to face a threat from a parasite at some point in their lives. The effects that parasites exert on hosts can range from minor reductions in fitness to rapid death. Therefore, parasites represent a widespread source of natural selection that

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operates across all stages of development, from egg traits [1] to secondary sexual traits [2]. The intensity of selection arising from parasitism has resulted in all multicellular organisms evolving a variety of defence mechanisms that counterbalance the fitness costs of parasitism, whether the parasites they encounter are the "*classic parasites*" that feed on the individual [3], or "*brood parasites*" that usurp parental care [4]. Both forms of parasitism provide the bedrock for theoretical and empirical work on addressing when parasites attack and how hosts respond with adaptive defences that vary extensively [3, 4]. Nevertheless, researchers studying brood parasites rarely also study classic parasites, and vice versa. In this synthesis paper, we take the novel step of placing defences against brood parasitism under the umbrella of "social immunity", a concept from classic parasitology, whereby defences have been selected in a donor because they benefit a recipient, who is the host (or potential host) of the parasite [5]. Our synthesis is not aimed to review the brood parasite literature in detail, as this has been done elsewhere [4, 6]. We rather select examples of defences from the classic and brood parasite literature to illustrate our points. We first reflect on the costs of classic versus brood parasitism, and then compare the social defences displayed against classic versus brood parasites at different stages of encounter. We conclude by considering how setting the evolution of host defences against brood parasitism in the "social immunity" framework may give us new insights into the brood parasitism phenomenon, and vice versa.

2. What are the costs of parasitism?

a) Classic parasitism

Classic parasites usurp an individual's resources that are destined for the use of that individual, and typically reduce the survival of their hosts as a consequence ('virulence')[7]. Although the effects of virulence on host mortality can be direct (especially from microparasites like bacterial or viral infections), many parasites rarely induce mortality directly. This is particularly true for macroparasites (e.g. gastrointestinal worms), but also true for many microparasites, such as the cold virus. Instead, these parasites reduce foraging efficiency or competitiveness for territories, for example, and as a consequence reduce mortality indirectly. Therefore, the extent of damage caused by a parasitic interaction can depend strongly on the host's condition, such that parasites that cause little damage in a high quality host may be heavily detrimental to a host suffering from malnourishment, for example [8]. Although virulence is generally defined in terms of host mortality e.g. [7], parasite-induced mortality does not necessarily reduce host fitness. Mortality can occur post-reproduction, for example, when the organism has already achieved its lifetime reproductive success (LRS). However, mortality of individuals at a pre-reproductive developmental stage will completely wipe out LRS, and mortality at any point during the reproductive life stage is predicted to drive fitness decays below those of non-parasitised individuals. Low virulence parasites can also directly or indirectly impact LRS through mechanisms other than mortality. Many trematodes that infect snails, for example, are 'castrating parasites' that cause a diversion of the resources that would be allocated by the host into reproduction, to growth or survival, increasing the chances that the parasite will be transmitted to the next host [9]. Like the indirect effects on mortality from low-virulence parasites, there can also be indirect effects on LRS via reduced competitiveness for mates [2]. It is therefore the relative difference in fitness among parasitised and non-parasitised hosts that determines the strong selection pressure to resist (reduce the numbers of) parasites or tolerate them (reduce their negative impact).

b) Brood parasitism

The inclusive fitness of many animal species benefits from provision of resources to their offspring after they have detached from the parental body ('narrow-sense parental care'[10]), which increases the indirect fitness element of an individual's inclusive fitness (see [11] for a detailed discussion on assigning fitness to parents versus offspring). Parental care thereby provides a further source of energy that a parasite can exploit. These episodes of resource availability drive the origin of brood

parasitism. Parents may invest resources in the egg, and/or provide a more secure environment through direct protection from predators or parasites [12]. Parents may also protect their young against adverse environmental conditions by provisioning food, either by stocking the "larder", for example, nest provisioning in mason bees (*Osmia* spp.) [13], or by directly feeding offspring [10, 11, 14]. Parental care is extremely costly and wherever large amounts of costly resources are delivered by parents to their offspring, there is an opportunity for cheats to try to usurp those resources. The eponymous example of this parasitism of reproductive investment is performed by the Common cuckoo *(Cuculus canorus*) who targets passerine hosts with post-hatching parental care. She removes a host egg upon laying her own in the nest, which hatches more rapidly than its adopted nest-mates and promptly forcibly ejects all of the host's own offspring from the nest, ensuring that all subsequent parental investment is directed exclusively towards the parasite [15]. Interspecific brood parasitism has evolved 7 times independently in the birds alone [16] and brood parasitism in its diverse forms is widespread across animal taxa that display parental care [17].

In contrast to classic parasitism, brood parasitism is a direct attack on the indirect fitness of the parent. However, like classic parasitism, the costs of brood parasitism vary depending on the strategy of the parasite. For brood parasites, the magnitude of the costs are also a function of the level of parental investment in post-hatching care provided by the host. For example, in many cases of avian brood parasitism by cuckoos (*Cuculus* sp.) the combination of high levels of parental investment and an extremely virulent attack strategy by the parasite results in high parasite-induced inclusive fitness costs for hosts [16]. However, some avian parasites are less virulent, either because they do not eject the host's eggs or chicks, or because pre-hatching investment can be shared amongst the brood and the parasite has lower requirements for post-hatching care [16]. There is a comparable range of fitness costs associated with brood parasites in non-avian systems. For example inquiline social parasites of social hymenopteran colonies can completely replace the colony queen prior to the production of reproductives, thus reducing her LRS to zero [18], which is more extreme than even the most virulent avian brood parasites [16]. Other parasites are less virulent, typically reducing the overall success of the brood, but not destroying it completely, for example the cuckoo fungus in termites, *Fibularhizoctonia* sp [19], the inquiline thrips, *Akainothrips francisi* (and see Table 1 in [20] for further examples within the thysanoptera), cuckoo wasps, e.g. *Sapyga pumella* [13], slavemaking ants, *Temnothorax americanus* ([21] and references therein), and *Maculinea* caterpillars ([22] and references therein).

3. Social immunity - a framework to understand defences against classic vs brood parasites

a) What is social immunity?

Parasite defences, in the classic understanding of host-parasite interactions, are directed by the host individual against the parasite to protect the host's survival and therefore their direct fitness. In other words, they are "personal". However, there are a class of defences that increase the fitness of the individual producing that defence and one or more conspecific recipients - that is, social defences. This is known as "Social immunity" in the broad sense [5, 23], and is the definition we use throughout this paper, but see [24, 25] for a narrow-sense definition, which is restricted to anti-parasite defences occurring in eusocial species. Social immune responses include the anti-parasite defences typically provided for others in kin-structured groups, such as socially-breeding vertebrates, sub-social and social invertebrates, and even potentially plants and microbes [5, 23]. Instances of social anti-parasite defences abound, most notably in the highly social hymenoptera, whose colonial living and close relatedness make them especially valuable [24, 25]. Fever, for example, is employed by an individual for personal protection against parasites [26-28]. However, there is a social equivalent in honeybees, whereby workers collectively raise the temperature of the brood when they are infected with *Ascosphaera apis*, a heat sensitive fungus that causes chalkbrood disease [29]. This defence is generated by uninfected workers for the defence of the offspring and so constitutes a social

defence. Collective behaviours an extreme example of social immune responses and are typically only seen in eusocial species. There are, however, many examples of social immune responses that do not require collective action and are present both in eusocial species and in groups with lower levels of social organisation, such as nuclear families. For example, there are many cases of the provisioning of immune molecules from one individual to another, at a cost to the producer for the benefit of the receiver. This typically occurs between parents and offspring, for example, the maternal transfer of antibodies/other immune components in milk or eggs [30, 31], or between siblings, for example the transfer of antifungal secretions between termite workers [32]. A key aspect of social immunity is that selection operates through traits that maximise the indirect fitness of the donor by protecting their kin from infection. In contrast, personal immunity has typically been selected to protect an individual's direct fitness via survival [5]. A consequence of this is that social immunity sets up potential conflicts between donors and receivers, much in the same way that the provisioning of resources to a brood sets up parent-offspring conflict [33]. For example, with social immune responses, a donor's indirect fitness might best be maximised by killing one infected offspring to protect the remaining brood. However, this wipes out the direct fitness of the sacrificed individual (though they may still gain some indirect fitness via the survival of their kin).

The most developed social immune responses are, unsurprisingly, found in the most developed social systems, namely the eusocial insects [24, 25]. In those societies there has been a separation of brood into workers and reproductives, such that the colony functions much like that of an individual, where workers (functionally equivalent to somatic cells) can be sacrificed to protect the reproductives (germline). Here we see many examples of workers being killed, isolated, excluded or even excluding themselves from the colony when infected, to protect their kin (see examples in [24, 25]). In many cases workers are sterile, and where they can reproduce to a certain extent (e,g, the laying of unfertilised male eggs in hymenoptera), policing by the queen or other workers reduces the success of this strategy [34]. As such, there is little conflict between their own fitness and that of the colony, because their fitness is primarily indirect [25]. However, eusocial colonies are at the extreme end of social organisation and this relative lack of conflict over the response to parasites is not typical [35]. Social immune responses occur at multiple social levels, including nuclear families [5, 23], in which conflicts are rife [33]. Social living therefore provides both the ideal environment for parasites to thrive and a network of interactions between individuals whose response to those parasites is shaped by their own selfish interests. The concept of social immunity may therefore allow new insights into host-parasite interactions, whereby the infected individual is not necessarily at the centre of the defensive response, and the defences employed on their behalf are not necessarily in their best interests. Thus, can the concept of social immunity be applied to brood parasitism?

b) Is defence against brood parasitism a form of social immunity?

Much like social immunity, host defences against brood parasitism are employed by a donor (a parent) for the benefit of one or more recipients (typically kin), and as with social defences against classic parasites, selection acts on the donor, not the recipient. Defences have therefore evolved to protect the donor's indirect fitness, not the survival or ultimately the direct fitness of individual recipients. If the response that best maximises the donor's inclusive fitness does not necessarily maximise the inclusive fitness of all recipients, this will create conflicts within the social group. If we take the example of a reed warbler threatened with parasitism by the common cuckoo, the best response to finding a suspicious egg in the nest could be rejection, and the best threshold for rejection could be quite low. This is because the mistaken rejection of a host's own egg is significantly less costly to the host than the potential loss of an entire brood, should a cuckoo successfully parasitise the nest [36]. From the recipient brood's perspective, the remaining siblings will benefit from an increased share of their parents' resources, but the consequences are catastrophic for the mistakenly rejected reed warbler egg. Defences against brood parasitism therefore fit the paradigm of social immunity, and are subject to the same conflicts between donor and recipients that are present for the response to classic parasites. Much like classic parasites, these conflicts will be reduced or resolved for eusocial colonies who experience brood parasites, due to the reproductive division of labour. So

how do the responses of hosts to brood parasites compare to examples of social defences against classic parasites?

c) How do social immune defences against classic and brood parasites compare across different stages of parasite encounter?

Defences can be employed at any stage, from before parasites have been detected through employing risk-averse behavioural strategies, to immediately upon detection in the environment, at the point where parasites directly threaten the body/nest and even post-invasion where the damage they cause can be controlled. Here we compare the types of social defences displayed by donors against classic or brood parasites at each of these stages.

(i) Parasite avoidance

The most basic form of social defence against parasitism is to employ mechanisms that reduce the likelihood of encountering parasites in the first place. Avoidance behaviours can be employed against classic parasites in a social immune context, for example by avoiding laying eggs or raising young in contaminated locations. Carrion-breeding dung beetles have been shown to roll the carrion balls that they use to provision their young a distance from the carcass, either horizontally, or by digging to depths of up to 1m, at which the concentration of microbes, particularly those that cause infection, are greatly reduced [37]. The removal of corpses from a communal nest is a social defence found in ants [38], bees and termites [39], thus reducing the risk of infection to other colony mates. As brood parasites typically actively seek out their hosts, parasite avoidance is less straightforward. However, it would be possible to avoid risky locations, such as those that are known to host a brood parasite, or that has been host to brood parasites in the past [40-42].

A large number of potential hosts can facilitate parasitism, either by reducing search costs for an actively searching parasite, or by facilitating transmission of parasites passively contracted from the environment [43]. Social living is therefore subject to increased risks of parasitism. In beewolves, cuckoo activity is positively density-dependent, suggesting that cuckoos do indeed target sites with higher nest density [41]. Similarly, in this issue, Medina & Langmore [44] perform a comparative analysis across 242 species of host and non-host species of birds. This analysis revealed that species with smaller breeding areas (and thus, with higher breeding densities) were more likely to be hosts of brood parasites. Another mechanism of parasite avoidance could therefore be to avoid nesting near other conspecifics, but this outcome would be driven by the balance between the costs of the increased risk of parasitism against the benefits of social living, which can also include increased defences against parasitism (see next section).

(ii) Defending the body/nest

Once parasites have successfully located their host, they need to enter or attach to the body, or enter the nest, in the case of brood parasites, so that the host's resources can be exploited. Hosts have an array of behavioural, physical or chemical defences that can be employed to resist parasite ingression. Behaviours include allogrooming with antimicrobial chemicals in termites [45] and ants [46], and excluding infected individuals from the nest, which is fatal for the individuals but protects the colony [24, 25] and references therein). Behavioural defences can also prevent brood parasites from accessing nests. These so-called 'frontline defences' are now the focus of active research, especially against avian brood parasites [47], after it was discovered that alarm calls and physical mobbing of cuckoos and cowbirds can reduce parasitism [48, 49] even to the point that attacks can kill the parasite [49, 50]. For some hosts, these attacks can become collaborative, where multiple individuals join to drive the parasite from the nest. Cooperatively breeding fairy wrens, for example, mob as a group [47], and otherwise non-cooperative Oriental reed warblers will join in attacking cuckoos at neighbours' nests [51]. Solitary bees also aggressively defend their provisioned nests against brood parasites, and indeed against parasites attempting to attack nearby nests [52] generating

similar group defences as in the avian examples. More broadly, observing the mobbing behaviour of neighbours ('social information') can also act to upregulate defences at the nest, even if it does not increase the number of active defenders beyond the nest-owners (e.g. [53]). After reed warblers, for example, witness neighbours attacking cuckoos they increase mobbing attacks back at their own nests [54]. This use of social information is thought to shift the reed warbler's recognition threshold of cuckoos versus the hawks that cuckoos mimic [55], thus allowing hosts to fine-tune defence of their nest. Vigilance behaviours and aggression towards brood parasites have also been shown in social insects e.g. aggression towards slavemaking *Temnothorax* ants by defending hosts [21], although the effects of social information on modulating expression of these defences has not been explored in detail.

Classic parasites can also be repelled through physical defences. For example, many social insects live in defensible nests, which provide physical protection from a range of threats, including detection by mobile parasites [56]. They also allow for effective vigilance, with entrance guards able to screen nestmates for parasitic infection. Both ants and birds respectively have been shown to protect their nests by collecting antiparasitic resins [57] or plants [58]. Several animal species also use self-produced antimicrobials in the fabric of a nesting structure e.g. tungara frogs [59] and several nest-building fish [60-62], termites [63], and burying beetles [64] and some even cooperate with bacteria to deter parasites e.g. bark beetles [65] and burying beetles [66-68], all of which reduce the likelihood of parasite ingression. Are there similar physical barriers to defend against brood parasites before they become established in the nest? There is evidence that the physical architecture of the nest may have evolved to reduce the likelihood of brood parasites accessing the brood, for example, the woven access tubes in the nests of *Ploceus* weaverbirds [69] and the capping of brood cells, or addition of empty brood cells in solitary bees [13, 70]. However, as these physical defences are likely to work against predators too, it is possible that they evolved for that purpose and thus, act secondarily against brood parasites. The use of a defensible nest by social insects can also provide protection against brood parasites, who need to gain access and avoid detection by guards [56]. In contrast, there is no evidence for the use of collected or self-produced antiparasitic substances to protect the nest against brood parasites. This may be inhibited by the taxonomic similarity between brood parasites and their hosts, particularly avian parasites, such that substances repellent to, or detrimental to the health of brood parasites are likely to have a similar effect on their hosts.

(iii) Reducing parasite success post invasion

If the parasite breaches the first line of defences, it still needs to become established in the host's body, or in the nest, to be successful. This hinges on two key elements: the donor's ability to *recognise* the parasite as non-self, and then to *respond* to it by either resisting (i.e., reducing parasite fitness), or tolerating its presence (i.e., reducing the parasite's negative impact on the host).

Recognition:

The mechanisms by which hosts can recognise classic parasites in their own bodies is now well understood and involves the detection of PAMPs (Pathogen Associated Molecular Patterns) via pathogen recognition receptors PRRs in both vertebrates and invertebrate hosts (see [3, 71] and references therein). For a social immune response, however, recognition is complicated by the fact that the donor of the defence has to recognise infection concealed inside the recipient's body. As such, PAMPs may not be detectable to the donor. Instead, the donor has to rely on visual or chemical signatures of infection being displayed by the infected individual e.g. ants [72-74] and termites [75], or even active transfer of information on infection status by the recipient e.g. warning dances in termites [76, 77]. Recognition of brood parasites in the nest should be more straightforward as the parasites are not concealed inside another individual's body. However, direct molecular recognition is unlikely due to a lack of interaction between parasite and host at the cellular level. Much like social immunity, recognition instead relies primarily on visual (birds and insects) or olfactory (insects) cues [4].

This visual apparency has selected for visual mimicry or camouflage of brood parasites to avoid detection [19, 78-80]. Hosts of avian brood parasites, for example, discriminate against parasite eggs when there is a mis-match in their colour and patterning with the host's own eggs. Research into how host "signatures" on the egg help identify non-self has exploded in recent years as appropriate analysis tools incorporating avian vision have been developed e.g. [81-86], or as the taxonomic range of brood parasite hosts has expanded [87]. We now know, for example, that signature elements are likely to interact in terms of the information they provide to hosts and allow more fine-scaled recognition [81, 88]. We also have evidence that the context of signatures matters, as both the location and developmental stage of the parasite can determine how readily it is recognised. For example, Yang *et al.* [89] show that poorly mimetic eggs placed outside of the nest cup will be retrieved, but later rejected once they are alongside the host's own eggs. Similarly, cuckoo catfish, *Synodontis multipunctatus*, eggs are readily collected by host cichlids, despite being visually non-mimetic [90, 91] but then recognised as non-self and rejected once in the host's mouth [87], potentially via chemical cues. Interestingly, eggs that are rejected can survive outside of their cichlid host and re-infect successfully as juveniles, at which stage recognition does not seem to occur [91]. Insect hosts of brood parasites, on the other hand, tend to have distinctive cuticular hydrocarbon profiles that can be used to discriminate kin or social partners [92-95], leading to chemical mimicry [96], or camouflage [97]. Kaur *et al*. [21] measured the aggressive responses displayed by hosts to slavemaking ants across a number of populations. Surprisingly, the best predictor of the host response, both behaviourally and in terms of gene expression in the brain, was the ecological success of the parasite. Parasites from some populations were just better at avoiding recognition, potentially due to their altered cuticular chemical profiles [21]. Brood parasite hosts therefore use external chemical or visual cues to recognise parasites as non-self, much as social immune donors use when recognising recipients infected with classic parasites.

Resistance:

For classic parasites, the post-infection social immune responses of the donor can cure the recipient e.g. social fever in honeybees (see section 3a) or directly kill the parasite e.g. ants [72] and termites [75]), thus protecting other kin sharing the social environment. In the most extreme cases, hosts can even abandon a nest that is heavily infected with parasites as has been shown in ants [38] and termites [39], thus killing the parasite by depriving it of hosts. Similarly, brood parasite hosts can also show resistance by ejecting the parasite from the nest [98] or directly killing it [21]. There is also evidence that some hosts will abandon infected nests after brood parasites have been detected. For example, brown-headed cowbird (*Molothrus ater*) hosts have been shown to desert parasitized nests [99] and Mason bees (*Osmia* spp) have been shown to abandon the tunnels they have been provisioning once one of the brood cells becomes parasitized [21]. Similarly *Myrmica* ants have been shown to abandon nests where a virulent species of *Maculinea* caterpillar has become established [100].

Resistance against both classic and brood parasites can be costly, however, either energetically or by inflicting damage to self. Spottiswoode and Busch [101] compare the vertebrate MHC parasite recognition system with the egg recognition systems of birds and conclude that both are selected to find the best balance between the inevitable and costly type I and type II errors. Type I errors occur when hosts wrongly attack self; body cells in the case of classic parasites and rejection of the hosts own eggs/offspring in the case of brood parasites. Type II errors occur when the host fails to recognise a parasite as non-self. Any mechanism that can ameliorate these costs will therefore be selected for. One such mechanism is self-medication, whereby an individual changes their diet upon infection to reduce the success of the parasite [102]. There are examples of self-medication in social parasite defence. For example, Monarch butterflies infected with a protozoan parasite that can be transmitted vertically to their offspring, choose to lay eggs on milkweeds containing high levels of cardenolides, the consumption of which can reduce parasite load and virulence [103]. The potential to use self- or brood-medication against brood parasites is less clear. However, if we consider self-medication simply as a shift in diet that favours the host in the host-parasite interaction, then it is a

possibility. As brood parasites can only survive in the host nest if the resources they are usurping are suitable, then there could be selection for a shift in host diet, either temporarily or permanently away from the diet preferred by the parasite. For example, the common European cuckoo only parasitizes insectivorous passerines [104] but is unable to parasitise the Asian flycatcher, which provisions its chicks with hard to digest beetles and grasshoppers, because it cannot thrive on that diet [105]. Selection could, therefore, act on heavily parasitized hosts to adjust their diet away from that preferred by the cuckoo to one on which the cuckoo cannot survive.

Tolerance:

Another cost-reducing mechanism of immune defence is tolerance, whereby the negative fitness effects of a given parasite load on the host are reduced [106]. Tolerance mechanisms have long been studied in plants, but have only recently found their way into the animal host-parasite literature [107]. Tolerance against classic parasites is typically measured as a fitness reaction norm across host genotypes for a range of parasite loads, to estimate genetic variation in the trait, or as differential fitness effects across environments of a given parasite load to measure environmental influences on tolerance (see table 1 in [108]). Mechanisms differ across host-parasite combinations, but can include reducing the damage to self from a strong immune response [109], reducing the virulence of parasites, for example by mopping up the cell-damaging toxins produced by pathogenic bacteria [110] or by altering reproductive responses, for example fecundity compensation can be a tolerance mechanism as it maintain fitness better for a given parasite load than the original reproductive schedule, which is likely to be curtailed by parasite induced mortality or sterility [111]. The presence of tolerance mechanisms in social immunity are hypothesised, but have not been explicitly tested [25], though there is some evidence for fecundity compensation to replace workers lost to parasitism in termites [112].

Can tolerance work in host-brood parasite interactions? Evidence to date suggests that the last mechanism described above, altering reproductive responses, can be employed to reduce the costs of brood parasitism to the host e.g. [113]. For example, spreading broods over more clutches by reducing the number of offspring per brood could reduce the costs if the likelihood of a single host being parasitized remained the same, as a single parasitism event would impact fewer offspring. This strategy would work for both highly virulent (e.g. those that destroy entire broods) and less virulent parasites (i.e. those that are reared alongside the host's brood). In contrast, increasing clutch size is a tolerance mechanism that can only work against parasites that share parental care with the brood. There is evidence for both of these strategies from a number of studies of avian brood-parasite hosts (see table 1 in [114]), but studies on tolerance to brood parasites have typically been correlational and other interpretations of the host response than tolerance could be invoked [114]. Potential tolerance responses in brood parasite hosts are hard to disentangle from resistance without direct experimental manipulation. For example, a larger clutch size might reduce the impact of a single low virulence parasite in the nest, but the parasite might also suffer a reduced growth rate due to greater competition with its foster siblings, such that this approach could also be a resistance mechanism. It is clear that more work is required to understand the potential role of tolerance in host-brood parasite interactions (for a more in depth discussion of this topic see [114]).

4. Conflict in social defences

a) How does the potential for conflict in social defences compare across the stages of parasite encounter?

As with all apparently altruistic acts, unless the interests of donor and recipient are perfectly aligned, there is a potential for conflict, and social defences against parasites are no exception. The point at which the parasite is encountered and the defences employed has a strong bearing on the potential levels of conflict the defences could induce (Figure 1). Early stage defences, such as avoiding parasites in the environment by avoiding risky nesting locations, for example, are unlikely to induce

much conflict, because the interests of the donor and the recipient are aligned. Both benefit from avoiding parasitism in the first place, whether that is from classic or brood parasites. More direct defences employed prior to parasite encounter, such as the construction of defensible [56, 69] or concealed nests [13, 37, 69, 70], or the collection/production of anti-parasite substances [57-64] have the potential to induce some conflict, because the donor is paying high energetic and time costs for the construction/ protection of the nest/ kin, and may have to do this on multiple occasions for future broods. The donor must balance the costs of investing now against their residual reproductive value, and therefore recipients will value greater levels of investment in protection than donors will be selected to provide [10, 14, 33].

The point at which the potential for conflict is greatest is after the parasite has successfully established itself in the nest/ host (Figure 1). At this point, whether they are dealing with a classic or brood parasite, the donor has to determine whether to kill or cure. For classic parasites, this choice is likely driven by the stage in the process at which the infection is detected, the virulence of the parasite and the cost to the donor of providing a cure e.g. [72, 75]. If the host is terminally infected, then their best interests are served by being killed, as this may protect their indirect fitness by reducing the likelihood that they will infect their kin. However, if they can be cured, but the cost to the donor of treating the infection is too high, or the risk to other individuals in the social group is too great, the donor would be under selection to kill, in conflict with the interests of the recipient. For brood parasites, this dilemma is different. Killing could be targeted specifically at the parasite, e.g. by egg or chick rejection, and in this endeavour, the interests of the donor and the recipient are aligned. However, selection for mimicry in brood parasites means that rejection is prone to type 1 errors, whereby donors fail in their recognition and accidentally reject their own kin [101]. As discussed above, this leads to conflicts as the threshold for rejection could be very different for donors and recipients. Finally, for both classic and brood parasites, donors can respond by nest abandonment, thus killing their entire brood/colony [13, 99, 100]. This has the highest potential for conflict as only in cases of irretrievable, terminal infection of all individuals by a classic parasite, or the presence of a highly virulent brood parasite against which the donor has no defence, would this response also serve the interests of the recipients.

b) When are these conflicts resolved?

Here we suggest two potential mechanisms that could lead to the resolution of conflicts associated with social defences in non-eusocial systems. (i) Selection could favour defences with the least conflict. For example, by focussing efforts on defences that occur early in the sequence of host-brood parasite interactions, such as nest placement and vigilance (Figure 1). Another possibility is the evolution of reduced-cost care defences in response to parasitism, such as changing the food provided to offspring as a form of medication [103], or to increase their condition such that they can better tolerate low-virulence classic, or brood parasites. Other tolerance mechanisms, such as changing the reproductive schedule in response to brood parasites can also reduce the potential for conflict, as the donor would be selected to reproduce in the way that maximises both their own and offspring survival in the face of parasitism. (ii) Donor(s) and recipient(s) could coevolve "united" defences**.** The costs to the donor can be reduced by donors working together; collective defences against cuckoos in fairy wrens are more effective than individual mobbing, thereby increasing the effectiveness, and so reducing the cost of the defence [47]. Alternately, recipients could take on the role of donor by contributing to the social defence themselves. For example, in burying beetles, parents produce antimicrobial secretions that reduce the presence of classic parasites on their offspring's food [64], at a substantial cost to themselves [115]. However, larvae also produce these secretions collectively [116, 117], reducing the cost to the parent and so reducing potential conflict over this social immune response [118]. These collective defences, or reciprocal actions where individuals take on the role of both donor and recipient are frequent occurrences in eusocial insect colonies, where conflicts over defence are typically reduced due to the reproductive division of

labour [23-25]. However, whether these behaviours are a consequence of eusociality [25], or one of its drivers [23] has yet to be resolved.

A final potential outcome is where the conflicts are not resolved, but one of the parties 'wins' as can happen in cases of parent-offspring [33] or sexual conflict [119]. This is most likely to be the parent for both classic and brood parasites due to the imbalance of power in the social relationship [120]. At the egg stage in particular, offspring have no power to defend themselves against rejection or eviction from the parent, and juveniles are physically weaker and dependent on parents for protection and food, and so unlikely to be able to defend themselves, should selection favour the parent to sacrifice them due to infection.

5. Future directions

a) Why do defences vary?

Despite decades of research on host-brood parasite interactions, we still lack a satisfactory explanation for why defences vary within and across host species. In contrast to many of the examples above, some avian brood parasite hosts show comparatively weak defences (e.g. redstarts may abandon nests, but rarely remove common cuckoo eggs even though this is likely to be a less costly strategy [121]) or more puzzling still, they express no resistance against brood parasites (e.g. dunnocks do not reject even the most non-mimetic of common cuckoo eggs [122]). This may be evolutionary lag (there has been insufficient time for natural selection to act [123]), hosts without defences may represent systems at an evolutionary equilibrium [123], or the costs of mounting defences are too great relative to the fitness benefit of avoiding parasitism [124]. Alternative hypotheses based on spatial population and habitat structure have also been suggested, where defences vary because gene flow from non-parasitised populations reduces the likelihood that genetic mechanisms underpinning behavioural defences will reach fixation. Distinguishing between these potential explanations has thus far been challenging in birds [6] [125], and most work has focussed on only one stage of defence (egg rejection). Can the social immunity framework, as we have applied it here, provide some insight into this problem?

Theory predicts that the presence and strength of social immune defences produced on behalf of kin will vary because of the balance of costs (c) vs. benefits (b), modified by the relatedness (r) of the donor to the recipient (Hamilton's rule: $r*b>c$ [126]) (Also see [127] for a similar approach from the brood parasite's perspective). As discussed above, any mechanisms that could reduce the costs of social defence could therefore shift the balance towards defence against, rather than acceptance of, brood parasitism. One possibility is the evolution of personal defences against the parasite by the brood, as we see in burying beetle larvae, who cooperate with their parents in the production of antimicrobial secretions [116, 117]. In the case of brood parasitism, the parasite could be considered a classic parasite from the brood's perspective, as it directly affects the brood's survival (see section 2a), but evidence for direct defence against parasites by the brood is lacking from well-studied avian systems. Instead, in some cases there appears to be a transition to mutualism as the presence of a brood parasite in the nest can even enhance survival of host young against predators [128], though this effect might be population- or context-specific [129]. It may be that selection fails to act on the brood because they don't have the mechanisms to recognise parasites in the nest. However, it has been shown that offspring of species that suffer a higher incidence of parasitism by the brown-headed cowbird (*Molothrus ater*) tend to beg louder [130], and grow more rapidly [131], potentially reducing the costs of parasitism, much like the parental-driven tolerance responses covered above (see section 3. iii). Furthermore, there is increasing evidence that offspring can cooperate to exploit parental resources, and so in theory the brood could potentially evolve effective defences of their own. Perhaps this helps to explain why many avian brood parasites attempt to evict or kill host nestmates within hours of hatching, and often before host eggs themselves have hatched [15] [16].

In this review we have stressed that brood parasitism exerts detrimental effects on host fitness via a reduction in their indirect fitness across their lifetime. In terms of defences, selection acts on the donor to protect their lifetime indirect fitness rather than prioritising the current reproductive attempt. Studies attempting to empirically quantify the costs of defences, or the costs of parasitism across the host's lifespan remain few, however, and rely instead on assessing costs only in terms of the current brood (but see [132]). This is largely because inclusive fitness across the life course is difficult to measure in the field for many of the favoured brood parasite study systems, where hosts migrate or show high natal dispersal, and most of these are not amenable to experiments in the lab. Recent studies with captive Cichlid fish and their catfish cuckoos (e.g. [87, 90, 91, 133]) may provide a new avenue for replicating the advances in understanding resistance and tolerance against classic parasites, and the fitness benefits and costs of social immunity in particular, that have come from using invertebrate systems easily manipulated in the lab (e.g. [115, 134]).

b) Plasticity in immune defences – do social environments promote 'Density Dependent Prophylaxis'?

Many insect species that undergo boom and bust population cycles, for example, locusts [135] and armyworm caterpillars [136, 137], have been shown to use population density as a cue to increase investment in their immune systems, known as Density Dependent Prophylaxis (DDP) [138, 139]. This anticipates the increased risk of infection when living in close quarters with conspecifics and ensures that costly immune investment is targeted to high risk conditions. This response has been shown to occur across invertebrate taxa in response to classic parasites [139-142], and some studies provide evidence supporting its evolution in some vertebrate taxa e.g. rodents [143-145] and birds [146], but it has not explicitly been considered for brood parasites.

In this issue, Medina & Langmore [44] found that fairy wrens suffered greater levels of brood parasitism as their density increased, though at very high densities, this risk again reduced, such that hosts at intermediate densities suffered the most when parasitism levels were high [44]. High densities should increase the risk of parasitism as discussed above (see section 3c) i) Parasite avoidance), but fairy wrens in larger colonies mob cuckoo models more than those in low density colonies [147]. This suggests an upregulation of this defence in conditions where parasite risk is increased, which could arguably be considered a form of DDP. However, rather than the new phenotype being induced directly by density cues, as occurs in lepidoptera [136, 137] and orthoptera [148], it is thought to be driven by social learning, whereby individuals in larger colonies have more opportunities to learn the correct defensive response to potential parasites [149, 150]. Social cues could also act more broadly in a prophylactic manner if they enhance vigilance against brood parasites. For example, if male reed warblers witness a cuckoo at their nest during the female's egg laying period, then they guard the nest more closely [36]. Females, on the other hand, do not increase their nest attendance. Presumably this is because the opportunity costs of increased vigilance against cuckoos are too high when females need to forage to recoup the loss of resources incurred from producing eggs [36]. Social information reduces uncertainty about parasitism risk [151], because it reduces the relative cost of mistakes against the benefits of accurate defences when collecting personal information is costly [55, 150]. Perhaps females with increased social information about the risk of parasitism would also increase their nest attendance, but it is unknown if witnessing the aggressive behaviour of neighbours towards brood parasites influences vigilance *per se*, or indeed if nest attendance varies with host density.

c) Towards a macroecology of host-brood parasite dynamics?

A final reflection on the integrative expansions of the field of evolution of host-brood parasite dynamics points toward the need for comparative analyses at larger spatial and phylogenetic scales – that is, towards a macroecology of host-brood parasite interactions framed within the contest of spatially varying selection. While most fields in ecology and evolution have rapidly expanded in

scale given the advantages of large-scale studies as a means to understand the role of spatially varying selection on the predictability of adaptations across species [152, 153], comparative studies at such scales remain a 'pending debt' in the field of host-brood parasite interactions (but see [44, 154]). A broad range of sources of selection that vary along geographic gradients are core candidates to shape predictable spatial patterns of adaptive variation in host-brood parasite dynamics. Factors such as variation in resource availability [155], the effects of seasonality as a source of varying intensity of fecundity selection on clutch size [153, 156], variation in predator intensity [155, 157], and the intrinsic variation of species richness across space [158], offer a robust theoretical motivation to explore the adaptive expression of large-scale patterns of host-parasitic variation, which can then be linked to the phylogenetic patterns of emergence (and reversals) of these interactions, to ultimately draw a broader perspective on the factors and contexts that make the evolution of social immunity a viable strategy to counterbalance the costs of parasitism. The rapid accumulation of phylogenetic and environmental data reinforces the timely opportunity to expand the field under the context of macroecology.

Additional Information

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Authors' Contributions

All authors contributed equally to the manuscript.

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Figure captions

Figure 1 - The potential for conflict between donors and recipients is estimated across the stages of parasite encounter for defences against classic parasites (yellow), brood parasites (red) or both (orange). The potential for conflict typically increases as the threat from the parasite increases. Post-establishment, the donor can choose to care or kill, with the latter option providing the greatest potential conflicts between donor and recipients.