Avian Malaria Vectors in Host-Seeking Behaviour

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Vector-borne infectious diseases (e.g., malaria, dengue fever, and yellow fever) result from a parasite transmitted to humans and other animals by blood-feeding arthropods. They are major contributors to the global disease burden, as they account for nearly a fifth of all infectious diseases worldwide. The interaction between vectors and their hosts plays a key role driving vector-borne disease transmission.

Keywords: haemosporidian ; mosquitoes ; parasite manipulation hypothesis ; preen oil ; vector attractants

1. Avian Haemosporidians and Their Vectors

Vector-borne diseases (e.g., malaria, yellow fever, dengue fever) are major contributors to the global disease burden. Malaria is probably the most deathly and prevalent parasitic disease in the history of mankind. Indeed, it is estimated that about 150–300 million people have died from the effects of malaria during the past 100 years [1]. In 2020, there were an estimated 241 million cases of malaria worldwide, and 40% of the world's population still lives in areas where malaria is transmitted [2].

However, the systematicity and diversity of malaria parasites is much larger and not restricted to human parasites. These protozoan intracellular pathogens belong to order Haemosporidia, with numerous species from 15 genera infecting reptiles, birds, and mammals all around the world [3]. Avian haemosporidians are the largest group among all the haemosporidians infecting vertebrates by number of described species [4][5]. So far, more than 4600 parasite lineages from the genera *Plasmodium*, *Haemoproteus*, *Leucocytozoon*, and *Fallisia* have been described in more than 1900 avian species (MALAVI database version 2.5.2, December 2021 [6]). Moreover, new lineages are reported every year revealing the remaining unexplored genetic diversity of these parasites, mainly in the tropics [7][8][9][10]. These blood parasites may provoke detrimental effects on their avian host by reducing their survival [11][12][13], minimizing their reproductive success [14][15] and provoking tissue damage [16], hence reducing bird populations and eventually being responsible for population extinctions following the introduction of exotic haemosporidian parasites beyond their natural range [17]. They are globally distributed, infecting individuals representing most bird clades in all the continents except Antarctica [18], thus constituting an excellent model for the study of vector-host–parasite interactions [4].

The term "malaria parasites" has been a debated issue among parasitologists, ecologists, and evolutionary researchers [19][20]. The controversy lies from the incomplete knowledge of the phylogenetic relationships and pathogenicity of non-human malaria parasites [21]. Although some similarities can be observed in the life cycles of *Plasmodium*, *Haemoproteus*, and *Leucocytozoon*, they still have some differences in vectors, life cycles, and epidemiology [22]. Therefore, traditional taxonomists and parasitologists only accept *Plasmodium* species as being the true malaria parasites [4]. However, based on molecular genetic studies describing the phylogeny of the group, other authors also include other genera (i.e., *Haemoproteus*, *Leucocytozoon*) among the term "malaria parasites" [19]. Haemosporidians are obligate heteroxenous parasites, with some parts of their life cycle developing within their blood-feeding arthropod vectors (sexual reproduction), whereas some stages occur within their vertebrate hosts (asexual reproduction). After the inoculation of haemosporidian sporozoites from an infective vector, the parasites may either complete their life cycle in a susceptible host or abort their development in a non-susceptible host unable to develop infective stages (gametocytes) to reach a new host [5].

The infection starts with the bite of a female dipteran insect transmitting infective stages (sporozoites) from its saliva into the blood stream of the avian host while taking a blood meal. Afterwards, the sporozoites initiate the development of exoerythrocytic meronts in the endothelial cells of many organs and tissues. Meronts undergo asexual divisions in these cells and form merozoites for a minimum of two generations before the parasite produce merozoites capable to infect erythrocytes. This part of the life cycle before the development of merozoites that are able of invading blood cells is called the prepatent period (10–14 days). This extraerythrocytic stage is essential to enhance the initial infectious source. The breakage of host endothelial cells releases merozoites into the blood stream, which may result in (i) additional infection of reticuloendothelial cells; or (ii) invasion of red blood cells giving rise to gametocytes (macrogametocytes and

microgametocytes), which are infective to vectors. Gametocytes remain inside erythrocytes until ingestion by a dipteran insect in which the sexual process and sporogony take place. The inoculation of infective sporozoites will initiate new infections in vertebrate hosts [4][5][22].

The patent period of infection (interval during which parasites can be found in the blood stream) begins when parasites enter circulating erythrocytes, and encompasses different phases: (a) the acute stage, the initial phase when intensity of parasitaemia increases; (b) crisis, when parasitaemia reaches a maximum; and (c) the chronic phase, where the parasitaemia decreases and stabilizes at low levels. In haemosporidian infection, however, the chronic phase may be followed by a latent stage of infection, where parasites are absent in the blood stream but persist in internal organs. These tissue stages may initiate asexual replications leading to relapses and temporary increases of parasitaemia [4][5][22]. It has been shown that avian malaria *Plasmodium relictum* reacts to mosquito bites by increasing its overall parasitaemia in the blood during the chronic stage of the infection, which may result in enhanced probability of infection to mosquitoes and thus increased transmission rates [23].

To date, only species of blood-sucking dipteran insects (Diptera) have been described as vectors for haemosporidian parasites [24]. Culicidae mosquitoes from five genera (*Anopheles*, *Culex*, *Aedes*, *Culiseta*, *Coquillettidia*) are capable of transmitting avian *Plasmodium* parasites [24]. Other mosquito genera such as *Mansonia* and *Lutzia* have been found to carry *Plasmodium* lineages [25][26], but their competence in successfully transmitting malaria parasites still needs experimental confirmation (e.g., visual and molecular identification of sporozoites in salivary glands of these mosquitoes). Within the genus *Haemoproteus*, biting midges (mostly of the genus *Culicoides*, Ceratopogonidae) transmit parasites of the subgenus *Parahaemoproteus*, whereas parasites from subgenus *Haemoproteus* are vectored by louse flies (Hippoboscidae) [24]. For the genus *Leucocytozoon*, it is generally accepted that parasite species from subgenus *Leucocytozoon* are transmitted by black flies (Simuliidae), while ceratopogonid flies are responsible for the transmission of the only species of *Akiba* subgenus infecting birds (*A. caulleryi*) [27]. The only species of the genus *Fallisia* infecting birds is supposed to be transmitted by culicine mosquitoes [28], but this requires verification.

2. Cues Followed by Haemosporidian Vectors to Locate Their Hosts

Vector control is a crucial strategy for global malaria control in preventing infection and reducing disease transmission [29]. Although the contact between hosts and vectors may play a key role driving vector-borne disease transmission, vector density has been largely studied to analyse transmission risk, while host–vector contact dynamics, including host-seeking behaviour, have received less attention [30]. Historically, avian models have provided important insights to explain variations in disease risk, thus enhancing the knowledge on ecological and evolutionary processes ruling host–parasite interactions [31]. Identifying factors governing host selection by blood-feeding insects is essential to understand the transmission dynamics of vector-borne diseases [32]. Arthropod vectors may use a number of physical and chemical stimuli emitted by vertebrate hosts to detect their blood meal sources, including acoustic, visual, olfactory, moisture, and thermal cues (**Figure 1**) [33][34][35] (**Table 1**).

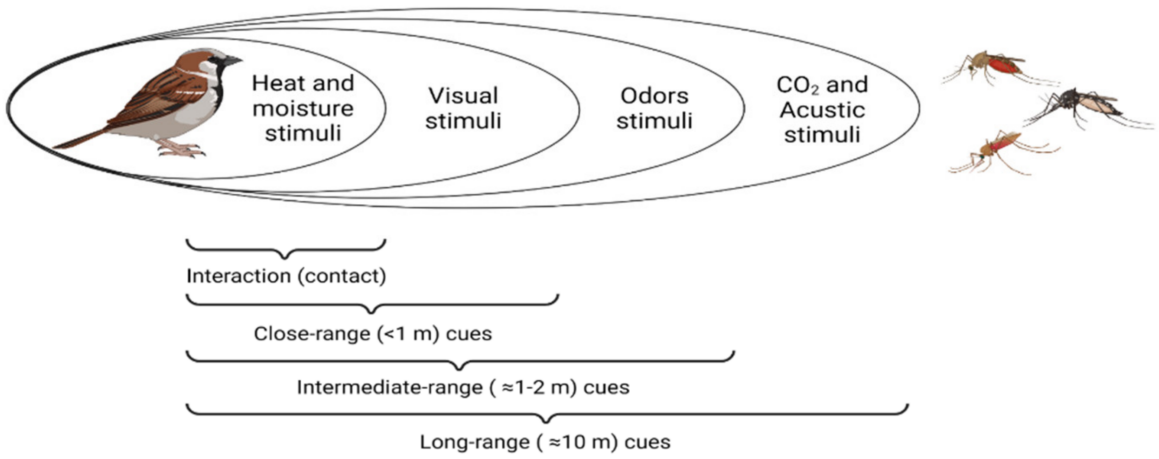

Figure 1. The sensory cues used by mosquitoes to detect their prey are distance-dependent. Mosquitoes follow a combination of cues to detect their potential hosts according to their proximity. Mosquitoes at larger distances can detect odours and CO_2 exhaled from host's breath, whereas vectors use body temperature and visual stimuli to locate their hosts at closer ranges. Adapted from [34][35].

Table 1. Summary of studies reporting increased (+), decreased (−), or neutral (0) attraction of avian haemosporidian vectors towards different stimuli.

Stimulus		Host	Vector	Effect	Explanation	Reference
Visual	Colour	49 North American bird species	*Culex pipiens*	+	Mosquitoes fed preferably on birds with lighter-coloured plumage.	[36]
	Motion	*Cyanistes caeruleus*	Biting midges	+	Abundance of biting midges was positively associated with parental provisioning effort (increased motion activity).	[37]
	Size	49 North American bird species	*Culex pipiens*	+	Mosquitoes fed preferably on birds with longer tarsi.	[36]

Stimulus		Host	Vector	Effect	Explanation	Reference
Heat and moisture	Temperature	*Ficedula hypoleuca*	Biting midges	+	Abundance of biting midges increased with temperature inside the bird nests.	[38]
	Temperature	*Parus major*	*Culex pipiens*	−	Birds with a lower body temperature were preferentially chosen by mosquitoes.	[39]
	Metabolic rate	*Passer domesticus*	*Culex pipiens*	−	House sparrows with lower metabolic rate suffered more mosquito bites.	[40]
	Moisture and temperature	*Cyanistes caerules*	Biting midges and black flies	0	No higher abundance of biting midges and black flies in nests with higher temperature and lower humidity.	[41]
Acoustic	Bird calls	*Passer, Fringila, Emberiza*	*Culex territans*	+	60% of female mosquitoes oriented toward the bird songs in phonotaxis experiments.	[42]
	Auditory stimulus	*Upupa epops*	Mosquitoes, blackflies and biting midges	0	Auditory cues of nestling hoopoes did not affect the abundance of vectors.	[43]

Stimulus		Host	Vector	Effect	Explanation	Reference
Olfactory	Carbon dioxide (CO_2)	*Cyanistes caeruleus*	Biting midges	+	Higher biting midge abundance in nests boxes with CO_2 levels higher than in the forest air.	[44]

Stimulus		Host	Vector	Effect	Explanation	Reference
Uropygial gland secretions	Uropygial secretion	*Gavia immer*	*Simulium euryadminiculum*	+	Black flies were attracted to the odour of the common loon's uropygial gland.	[45]
	Uropygial secretion	*Gavia immer*	*Simulium euryadminiculum*	+	Higher attraction of black flies to a combination of ether extract of the uropygial glands and CO_2 than to CO_2 alone.	[46]
	Ether extract	*Gavia immer*	*Simulium euryadminiculum*	+	Black flies were attracted to ether components of the uropygial gland.	[47]
	Cotton swabs coated with uropygial secretions	*Corvus brachyrhynchus*	*Culex pipiens, Culex restuans*	+	CDC traps baited with uropygial secretions captured more mosquitos than control traps.	[48]
	Diol volatile compounds from Natasauropygial gland secretion		*Culex quinquefasciatus Culex tarsalis, Culex nigripalpus, Aedes aegypti*	0	Meso-2,3-butanediol, 2,3-butanediol, and 2,3-docosanediol were not attractive to mosquitoes.	[49]
	Uropygial secretions	*Columba livia Cyanistes caeruleus*	Biting midges and black flies	0	No differences in the number of vectors captured in CDC traps or nests with this stimulus.	[50]

Stimulus		Host	Vector	Effect	Explanation	Reference
	Uropygial secretions	*Passer domesticus*	*Culex pipiens*, *Aedes caspius*	0	Mosquitoes were attracted equally to the ports containing uropygial secretion and to the control in olfactometer assays.	[51]
	Uropygial secretions	*Upupa epops*	Biting midges	–	Traps baited with uropygial secretion in pine forest significantly captured less biting midges than control traps.	[43]

Stimulus		Host	Vector	Effect	Explanation	Reference
Haemosporidian infection	Bird infected with malaria	*Serinus canaria*	*Culex pipiens*	+	Chronically infected birds attracted more vectors than either uninfected or acutely infected birds.	[52]
	Bird infected with malaria	*Passer domesticus*	*Culex pipiens*	+	Higher feeding preference of mosquitoes on infected sparrows.	[53]
	Bird infected with malaria	*Passer domesticus*	*Culex pipiens*	+	Mosquitoes were more attracted to the odour of malaria-infected sparrows.	[54]
	Bird infected with malaria	*Cyanistes caeruleus*	*Biting midges*	−	Higher abundance of biting midges in the nest attended by medicated birds with reduced parasitaemia.	[37]
	Bird infected with malaria	*Parus major*	*Culex pipiens*	−	Plasmodium-infected birds attracted significantly fewer mosquitoes than the uninfected ones.	[55]
	Bird infected with malaria	*Corvus monedula* *Passer domesticus*	*Culex pipiens, Aedes caspius*	0	Similar biting rates of mosquitoes on malaria infected and uninfected birds.	[56]

3. The Role of Uropygial Gland Secretion in Bird–Haemosporidian Vector Interactions

The uropygial gland (also called oil or preen gland) is an epidermal holocrine gland located at the dorsal base of the tail and present in all embryonic stage bird taxa, but degenerates in some adult birds such as Amazon parrots, ostriches, and some species of pigeons and doves [57][58]. It anatomically comprises the stratified epithelium, which contains secretory tubules filled with oil droplets that are in two similar size lobules, which drain into a single small papilla [59]. The uropygial secretion is a thick, transparent, complex oil (preening oil) that is spread on feathers and skin during preening [60]. The gland is covered by a tuft of down feathers, which may help in transmitting oil from the gland to the beak while preening [61] and facilitate perception of individual odour by conspecifics [62].

The uropygial gland secretion is a complex and variable mixture of chemical compounds. Lipids are the main components of preen oil, with a proportion of 59% of unsaturated fatty acids (mainly oleic acid), where saturated long chain fatty acids are in a percentage of approximately 34% [63][64]. Compounds of the preen oil are classified according to the size of the carbon chain as volatile (short-chain) or non-volatile (long-chain) [65]. The composition of uropygial gland secretion varies between and within species [66][67][68]. In addition to lipids, other substances, such as carotenoids, could be also present in the uropygial secretion of some species such as flamingos [69].

These compounds of preen secretions show singular properties, which has been associated with the different functionalities of uropygial secretions (see reviews in [60][64][65][70]). For example, lipids may constitute a waterproofing layer improving water repellence of feathers [60][71][72]. In addition, uropygial gland secretion may hold feather microstructure, which is necessary for keeping the plumage waterproof [65][73]. Moreover, volatile components may be implied in olfactory communication [74][75][76]. Furthermore, uropygial secretion may show antibacterial and antifungal properties and thus act as defensive barrier of skin and plumage. This antimicrobial function may be conferred by microbicidal activity of some uropygial gland chemical compounds [77][78][79][80][81][82][83] or by facilitating the growth of symbiotic feather bacteria that can defeat microbial antagonists [77][79][84][85][86]. Other proposed functions for uropygial secretion include drag reduction by facilitation of air flow during flight [87], excretion of pollutants [64][88], intensification of feather coloration for colour-mediated intraspecific communication [69][89], and lessening of the effects of oil contamination [90].

4. Do Bird Malaria Parasites Change the Host Attractiveness to Vectors?

The *host manipulation* hypothesis (also named the *parasite manipulation* hypothesis) states that parasites can modify the behaviour, appearance, and physiology of their hosts to increase their transmission success and, thereby, their fitness [91][92]. Hence, parasites able to manipulate their vector and/or vertebrate hosts to enhance their transmission should be favoured by natural selection [93].

4.1. Manipulation of Vector to Increase Parasite Transmission

According to *host manipulation* hypothesis, vector-borne parasites may induce changes in phenotypic traits of their vectors to increase their transmission rates to the non-arthropod host [94][95]. In haemosporidian-vector systems, behavioural and physiological alterations in the arthropod vector induced by malaria parasites have been frequently reported. These changes include a more persistent host-seeking behaviour and feeding persistence, longer duration of mosquito bites and increased mosquito biting rate (see review in [94] and [96]). For example, it has been shown that *Plasmodium* spp. impaired the salivary function in sporozoite-infected mosquitoes by decreasing the activity of the apyrase salivary protein (enzyme with anticoagulatory properties) [97]. These malaria-induced changes can minimize the vector's ability to engorge and hence induce infected mosquitoes to feed several times on vertebrate hosts to obtain the same amount of blood. This hypothesis was experimentally tested in birds by Rossignol et al. [98], showing an increased daily biting rate of *Ae. aegypti* mosquitoes infected with *Plasmodium gallinaceum* sporozoites (the transmission stage of the malaria parasite) compared to non-infected mosquitoes. In addition, Cornet et al. [99] monitored the effect of infection with avian malaria *P. relictum* on the blood feeding behaviour of *Culex pipiens quinquefasciatus* mosquitoes, showing that sporozoite-infected vectors completed their blood meal later and ended up with smaller blood meals than uninfected mosquitoes.

Furthermore, parasites would optimize their transmission rates favouring vector encounters with suitable hosts. Hence, a parasite manipulation of vector feeding preferences towards infected hosts should be expected (see review in [24]). In support for this idea, Yan et al. [53] found a higher feeding preference of *Cx. pipiens* mosquitoes on house sparrows naturally infected with malaria than in birds with experimentally reduced infection. In addition, Díez-Fernández et al. [54] showed that nulliparous (e.g., uninfected mosquitoes without previous access to blood) *Cx. pipiens* females were more

attracted towards the whole-body odour (headspace) of *Plasmodium*-infected house sparrows than to uninfected birds in a dual-choice olfactometer. However, no enhanced attraction of vectors towards *Plasmodium* infected birds [56] or even a decreased attractiveness of infected hosts to vectors has also been found. In this line, Tomás et al. [37] experimentally reduced haemosporidian parasitaemia in female blue tits, showing a higher abundance of biting midges in nests attended by these medicated females than in control nests cared by females with higher blood parasitaemias. Similarly, it has been documented that malaria infected great tits (*P. major*) were less attractive to *Cx. pipiens* mosquitoes [55]. These results suggesting a preference of haemosporidian vectors towards uninfected birds or hosts less infected with blood parasites could be explained by the detrimental effect of haemosporidian infection on the survival of their insect vectors. For example, Valkiūnas and Iezhova [100] reported higher mortality rates in biting midges *Culicoides impunctatus* experimentally infected with *Haemoproteus* than in uninfected control vectors. Likewise, Gutierrez-López et al. [101] experimentally reduced *Plasmodium* parasitaemia in house sparrows with an anti-malaria treatment, showing that the mosquitoes that fed on medicated birds had a higher lifespan than those that fed on control sparrows.

4.2. Manipulation of Vertebrate Host Attractiveness to Vectors

The feeding preference of haemosporidian vectors to infected hosts and/or hosts infected with transmissible stages of malaria leads to a more successful parasite transmission, which is in accordance with the parasite manipulation hypothesis. Although host attractiveness could be modified by the parasite, the definitive effect is the alteration of mosquito behaviour, which subsequently increases parasite transmission to the vector. Because host-seeking behaviour is mainly driven by a set of different stimuli [102], the question arisen from here is whether parasites may alter the host attractiveness to vectors by changing the appeal of cues followed by blood-sucking insects to detect their hosts.

Some studies have proposed that some clinical symptoms of malaria infection, such as fever and the increased production of sweat due to fever episodes, could guide *Anopheles* mosquitoes in host-seeking towards *Plasmodium*-infected humans [103][104]. Host-seeking behaviour in haemosporidian vectors is mainly prompted by olfactory perception of volatile organic compounds (VOCs) emitted by hosts [105]. Changes in VOCs profile during infection likely constitute the most important factor determining vector attraction. Therefore, *Plasmodium* parasites could increase the infected host attraction to mosquitoes by manipulating host-VOC profiles [106]. In agreement with this hypothesis, it has been documented that children suffering from high malaria parasitaemia produce larger amount of mosquito attractant VOCs (heptanal, octanal, nonanal, (E)-2-octenal, (E)-2-decenal, and 2-octanone) on their skins than patients having either low malaria parasite density or being parasite-free [107]. In addition, Schaber et al. [108] showed that children with malaria have a distinct shift in overall breath composition (higher breath levels of 2 mosquito-attractant terpenes, α-pinene, and 3-carene). In birds, Grieves et al. [109] compared the chemical profiles of uropygial secretion from song sparrows (*Melospiza melodia*) before and 13 days after malaria inoculation (corresponding to peak infection intensity), showing that wax ester profiles of uropygial secretion varied in sparrows that became acutely infected, but not in sham-inoculated control individuals. Contrasting results were found by Díez-Fernández et al. [110] when evaluating whether the chemical composition of uropygial secretions is associated with malaria infection in house sparrows. By using gas chromatography-mass spectrometry analyses, they found no significant differences in the composition of the volatile lipophilic components in the uropygial secretions of infected and uninfected house sparrows.

Skin and feather bacteria are responsible for the transformation of sweat components to VOCs [111]. Because the presence of blood parasites may modify the odour of an individual by altering the profile of symbiotic microbial community [112], the infection with malaria parasites may result in increased attractiveness of hosts. In this sense, an increased attractiveness of malaria-infected hosts to mosquitoes has been shown in humans [107][111][113][114], rodents [115][116], and birds ([52][54]; see review in [117]). However, to date there are no empirical studies linking malaria infection with changes in feather, skin, or preen gland microbiota and vector attraction. In birds, Videvall et al. [118] recently found that house sparrows infected with malaria harboured significantly higher abundances of bacteria from the genera *Arthrobacter* and *Micrococcus* in their uropygial gland, whereas uninfected sparrows had higher abundances of *Rhodococcus*, *Phenylobacterium*, and *Enhydrobacter*. These outcomes suggest a specific association between some symbiotic bacteria of the uropygial gland microbiota and *Plasmodium* parasites in birds, highlighting new questions on the role of the uropygial gland in host–parasite interaction.

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